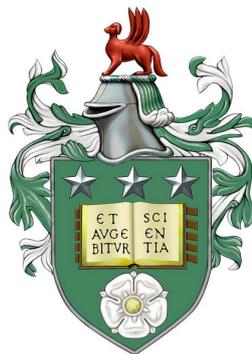


GEOSTATISTICAL MODELLING OF HEALTH INEQUALITIES ASSOCIATED WITH EXPOSURE TO ROAD-TRANSPORT EMISSIONS

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Doctor of Philosophy



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The candidate confirms that the work submitted is his own, except where work which has formed part of jointly-authored publications has been included. The contribution of the candidate and the other authors to this work has been explicitly indicated below. The candidate confirms that appropriate credit has been given within the thesis where reference has been made to the work of others.

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The jointly-authored publications include:

- Jephcote C & Chen H. (2012). Environmental injustices of children's exposure to air pollution from road-transport within the model British multicultural city of Leicester: 2000-09. *Science of the Total Environment*, Vol.414, pp.140-151
- Jephcote C & Chen H. (2013). Geospatial analysis of naturally occurring boundaries in road-transport emissions and children's respiratory health across a demographically diverse cityscape. *Social Science & Medicine*, Vol.82, pp.87-99
- Jephcote C, Chen H & Ropkins K. (201X). The effect of socio-environmental mechanisms on deteriorating respiratory health across urban communities during childhood. *Applied Geography* [Submitted for review in June 2013]

The listed joint-publications form part of my PhD work and contain elements from Chapters 4, 5, 6 and 7. I hereby declare I made the major intellectual contributions to the papers; the joint-authors were my PhD supervisors who offered advise at various stages of the research project.

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ABSTRACT

Road-transport accounts for a substantial proportion of the air quality objective pollutants experienced within the post-industrial cityscape. Traditionally, investigations have quantified the temporal health effects of such pollutants, yet the confined nature of European intra-urban environments often determine spatial variations in traffic pollutant levels, which tend to be associated with a plethora of social disparities. Recently, elements of spatial heterogeneity have attracted the attention of governmental advisory committees, whom acknowledge a limited understanding of spatially inclusive practices in spite of their potentially valuable applications (COMEAP 2006). Through considering spatial variations in children's respiratory health, across the model British multicultural City of Leicester (Vidal-Hall 2003), this project aimed to address the inadequacies of temporal models in capturing Pearce et al's (2010) wider 'triple jeopardy'.

The project's findings indicated significant global relationships to exist between children's hospitalisations, social-economic-status, ethnic minorities, and PM₁₀ road-transport emissions within Leicester. 'Local Indicators of Spatial Association' and 'Geographically Weighted Regression' identified important localised variations within the dataset, specifically relating to a 'double-burden' of residentially experienced road-transport emissions and deprivation effecting inner-city children's respiratory health. Further examination of the spatial field's, revealed critical distance-responses to exist between respiratory health fronts and select socio-environmental phenomenon, thus recognising the importance of exposure gradients found in the every-day environment.

It was suggested that exposure to detrimental socio-environmental factors initiated upper respiratory episodes, with prolonged contact impeding recovery leaving the child vulnerable to infection, exacerbating previous complaints and potentially causing conditions of greater severity. These findings provide a preliminary link between extreme cases of 'Catarrhal Child Syndrome' and socio-environmental influences, a conclusion previously eluding medical practitioners. Interestingly, affluent intra-urban communities tended to contribute the highest levels of emission from private transport, whilst residentially experiencing few environmental burdens. Thus, indicating that environmental injustices prevail across the model British multicultural city of Leicester. To readdress such environmental imbalances, the project suggested and explored a selection of general and community tailored transport schemes. In conclusion, geostatistical approaches are viewed to be an effective set of tools for health and urban planners, in the management of localised issues, which have previously been 'filtered' out by temporal practices.

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ABBREVIATIONS

ORGANISATIONS:

COMEAP	COMMITTEE ON THE MEDICAL EFFECTS OF AIR POLLUTANTS
DEFRA	DEPARTMENT FOR ENVIRONMENT, FOOD AND RURAL AFFAIRS
DFT	DEPARTMENT FOR TRANSPORT
DVLA	DRIVER AND VEHICLE LICENSING AGENCY
EC	EUROPEAN COMMISSION
EU	EUROPEAN UNION
HEI	HEALTH EFFECTS INSTITUTE
LCC	LEICESTER CITY COUNCIL
NAEI	NATIONAL ATMOSPHERIC EMISSION INVENTORY
NHS	NATIONAL HEALTH SERVICE
OECD	ORGANISATION FOR ECONOMIC CO-OPERATION AND DEVELOPMENT
ONS	OFFICE FOR NATIONAL STATISTICS
PCT	PRIMARY CARE TRUST
US EPA	US ENVIRONMENTAL PROTECTION AGENCY
WHO	WORLD HEALTH ORGANIZATION

MODELLING STRATEGIES:

EDA	EXPLORATORY DATA ANALYSIS
ESDA	EXPLORATORY SPATIAL DATA ANALYSIS
GAM	GENERALIZED ADDITIVE MODEL
GIS	GEOGRAPHIC INFORMATION SYSTEM
GISA	GLOBAL INDICATORS OF SPATIAL AUTOCORRELATION
GLM	GENERALIZED LINEAR MODEL
GWR	GEOGRAPHICALLY WEIGHTED REGRESSION
LISA	LOCAL INDICATORS OF SPATIAL AUTOCORRELATION
MM	MULTILEVEL MODELLING
OLS	ORDINARY LEAST SQUARES REGRESSION
OrK	ORDINARY KRIGING

POLLUTANTS:

BS	BLACK SMOKE
CO	CARBON MONOXIDE
CO ₂	CARBON DIOXIDE
NO _x	NITROGEN OXIDES
NO ₂	NITROGEN DIOXIDE
O ₃	OZONE
PM ₁₀	PARTICLES OF AERODYNAMIC DIAMETER TEN MICROMETRES OR LESS
PM _{2.5}	PARTICLES OF AERODYNAMIC DIAMETER 2.5 MICROMETRES OR LESS
SO ₂	SULPHUR DIOXIDE
TPM ₁₀	PM ₁₀ EXCLUSIVELY GENERATED BY ROAD-TRANSPORT
TSP	TOTAL SUSPENDED PARTICULATE MATTER
VOC	VOLATILE ORGANIC COMPOUND

OTHER TERMINOLOGIES:

AOR	ADJUSTED ODDS RATIO
APHEA2	AIR POLLUTION AND HEALTH: A EUROPEAN APPROACH 2
AQMA	AIR QUALITY MANAGEMENT AREA
BE	BOUNDARY ELEMENT
BLV	BOUNDARY LIKELIHOOD VALUE
CAS WARD	CENSUS AREA STATISTICS WARD
CBE	CANDIDATE BOUNDARY ELEMENT
CCS	CATARRHAL CHILD SYNDROME
EJ	ENVIRONMENTAL JUSTICE
FEV	FORCED EXPIRATORY VOLUME
FVC	FORCED VITAL CAPACITY
GP	GENERAL PRACTITIONER
H-H	CLUSTERING OF HIGH VALUES
H-L	HIGH OUTLYING VALUE
ICD-10	INTERNATIONAL CLASSIFICATION OF DISEASES (ICD): 10TH REVISION
L-L	CLUSTERING OF LOW VALUES
L-H	LOW OUTLYING VALUE
LCC	LONDON CONGESTION CHARGING SCHEME
LEZ	LOW EMISSION ZONE
LLSOA	LOWER LEVEL SUPER OUTPUT AREA
LRTI	LOWER RESPIRATORY TRACT INFECTIONS
NMMAPS	NATIONAL MORBIDITY AND MORTALITY AIR POLLUTION STUDY
NN	NEAREST NEIGHBOUR
OR	ODDS RATIO
PEACE	POLLUTION EFFECTS ON ASTHMATIC CHILDREN IN EUROPE
PEF	PEAK EXPIRATORY FLOW
PPP	POLLUTER PAYS PRINCIPLES
RR	RELATIVE RISK
RSV	RESPIRATORY SYNCYTIAL VIRUS
TRI	TOXIC RELEASE INVENTORY
UA	UNITARY AUTHORITY
URTI	UPPER RESPIRATORY TRACT INFECTIONS

UNITS:

μg	MICROGRAM
$\mu\text{g}/\text{m}^3$	MICROGRAMS PER CUBIC METRE
$\mu\text{g}/\text{ml}$	MICROGRAMS PER MILLILITRE
km	KILOMETRES
km/PEP	KILOMETRES TRAVELLED PER EMPLOYED PERSON
L	LITRES
L/sec	LITRES PER SECOND
m	METRES
mg/m^3	MILLIGRAM PER CUBIC METRE
mg/ml	MILLIGRAM PER CUBIC MILLILITRE
mm/min	MILLIMETRES PER MINUTE
ppm	PARTS PER MILLION
t/yr.	TONNES PER YEAR
YLG ₁₀	YEARS-OF-LIFE-GAINED PER 100,000 PERSONS OVER A 10-YEAR PERIOD

INTRODUCTION

“As crude a weapon as the cave man's club, the chemical barrage has been hurled against the fabric of life a fabric on the one hand delicate and destructible, on the other miraculously tough and resilient, and capable of striking back in unexpected ways.” (Carson 1962/2002, p297)

“The automobile has not merely taken over the street, it has dissolved the living tissue of the city ... Gas-filled, noisy and hazardous, our streets have become the most inhumane landscape in the world.” (Fitch 1960, p7)

“Why should we tolerate a diet of weak poisons, a home in insipid surroundings, a circle of acquaintances who are not quite our enemies, the noise of motors with just enough relief to prevent insanity? Who would want to live in a world which is just not quite fatal?” (Carson 1962/2002, p12)

“By respecting nature's limits and investing in nature's wealth, we can protect and enhance the environment's ability to sustain human wellbeing. But how humans interact with nature is intimately tied to how we interact with each other. Those who are relatively powerful and wealthy typically gain disproportionate benefits from the economic activities that degrade the environment, while those who are relatively powerless and poor typically bear disproportionate costs. All else equal, wider political and economic inequalities tend to result in higher levels of environmental harm. For this reason, efforts to safeguard the natural environment must go hand-in-hand with efforts to achieve more equitable distributions of power and wealth in human societies” (Boyce 2007, p267)

1.1. MOTIVATION

Clean air is considered to be a basic requirement of human health and well-being, however air pollution continues to pose a significant threat to health worldwide, resulting in >2 million premature deaths each year (WHO 2006). Epidemiological studies have demonstrated that many health problems (e.g. respiratory and cardiovascular) can be caused or worsened by exposure to air pollution on a day-to-day basis, with the level of severity varying from mortality in extreme cases to less serious morbidity concerns such as the increased use of inhalers by asthmatics (HEI 2009, COMEAP 2009).

The extent of these acute health effects have predominantly been documented through time-series analyses of sensitive populaces often including the young and elderly, which commonly reveal a significant level of increase in hospital admissions to occur even across exposure levels below US EPA and WHO Guidelines (Brunekreef et al 1995). For instance, children's respiratory hospitalisations from Australia and New Zealand's seven largest cities have reportedly risen by 2.3-2.5% per $10\mu\text{g}/\text{m}^3$ increase in 24-hour PM_{10} levels (Barnett 2005). Investigations specifically documenting the shifting temporal effects of transport derived air pollutants within an urban setting, also report a series of immediate and detrimental impacts towards health. For example, Fusco et al (2001) found admissions for acute respiratory infections in the general population (lag 0 days, +4.0%) and asthma among children (lag 1 day, +10.7%) to occur close to episodes of higher pollution.

However, whilst ambient levels of PM_{10} across Southern California were identified to increase the risk of bronchitic symptoms occurring in children with a history of asthma (Odds Ratio [OR] 1.4 per $19\mu\text{g}/\text{m}^3$), no associations were found amongst children without preexisting respiratory conditions, at these pollutant levels (McConnell et al 1999). This highlights the difficulties enforcement agencies face in setting standards that protect public health, as critical thresholds for certain population groups are conceivably non-existent. Still, there remains a requirement for a continued development of the literature surrounding sensitive populaces (extreme risk groups), to allow for the incorporation of adequate safety margins into state funded multilocational valuations of the wider populace.

Interestingly, an investigation of General Practitioner (GP) consultations for upper respiratory conditions in relation to extreme shifts in pollutant concentrations across London (10-90th percentile change), identified the presence of pollutant specific age structuring. For the multi-pollutant model, consultations of children, adult and elderly patients were observed to respectively alter by 3.8%, 2.8% and 4.6% per $30\mu\text{g}/\text{m}^3$ rise in PM_{10} , whereas a 1.5%, 2.7% and 4.3% change was attributed to a $25\mu\text{g}/\text{m}^3$ increase in NO_2 (Hajat et al 2002). Whilst the various cocktail of pollutants unanimously affected children, the effects of gaseous pollutants

were of a reduced nature; whereas the elderly populace persistently exhibited raised levels of environmental sensitivity. However the viability of examining adult let alone elderly persons should be questioned, when considering that a general deterioration of health occurs with age, and that preceding (and often unaccountable) environmental events or lifestyle choices are the potential underlying cause for such ailments.

This problematic phenomenon of mortality/morbidity displacement known as 'Harvesting', defines scenarios where a substantial proportion of cases occur only in those of frail health, to whom it would have happened in a few days anyway. Whilst crudely controlled for in multilocational enquires, insufficient historic databases presently exist to minimise any local false positives accredited to 'harvesting' in a significant manner. For local inquires, one would therefore recommend that epidemiological focus be placed on the young, whom (a) offer the most viable response out of all sensitive groups as historical socio-environmental events are rare; and (b) present the required conditions in which respiratory responses initially materialise.

Whilst city specific studies have yielded important information to assist with the quantification of acute respiratory responses, select authors focusing on the sensitivity of these results have shown how different temporal techniques allow for various interpretations of the responses magnitude (Smith et al 2000). Thus, the case for tighter regulations cannot be based solely on studies of this nature, and as a consequence of such statistical uncertainties, the cornerstone of all regulatory charters primarily stem from a select few large-scale research efforts.

Two prospective cohort studies, known as the 'Six Cities Study' (Dockery et al 1993) and the 'American Cancer Society (ACS) Study' (Pope et al 1995), represent the first of these epidemiology studies to pool together information from multiple urban sites, in their quantification of annual mortality responses attributed to fine particulates. In their study of 8,111 adults, Dockery et al (1993) observed a 26% difference in adjusted mortality rates between those most and least polluted urban municipalities, whereas Pope et al (1995) encountered a separation of 18% between the two extremes in their study of 295,223 adults. Initially, medical records from both enquiries were kept private to maintain participant confidentiality, yet this decision proved controversial, with select parties insisting that any data generated using federal funding should be made public. In 1997, the US Environmental Protection Agency (EPA) used these findings to uphold PM₁₀ air quality standards, further igniting levels of public scrutiny, so much so that both parties concerned requested the Health Effects Institute (HEI) make an independent reanalysis. Upon validating the quality of the original cohort datasets and successfully replicating the original outputs of the 'Cox Models',

the reanalysis Team conducted a series of investigations to assess their analytical robustness. For the 'Six Cities Study', hazard models which now extended the number of covariates, incorporated temporal changes in secondary health influences (i.e. smoking and BMI) and accounted for population mobility, respectively provided relative risks (RR) for PM_{2.5} of 1.28, 1.32 and 1.23 (HEI 2000); similar minor discrepancies were also observed from the ACS studies original outputs.

Interestingly, the nationwide distribution of the 50 municipalities from the ACS dataset also legitimised the application of spatial analytical methods, to validate from another statistical tangent and to shed light on the viability of such emerging methodologies. In these models, pollutant covariates were replaced by an indicator function for each city (avoiding assumptions of independence), with a second stage then incorporating ecological and spatial considerations. The base model achieved validation of the original outputs, whilst the inclusion of spatial independence at a regional level resulted in a reduced RR of 1.16 (HEI 2000). Interestingly, Pope et al (1995) had also investigated the effects of pollutants on mortality with a second environmental tracer (sulphate), across a wider geographic sample (552,138 adults in 154 US cities), allowing for a more detailed spatial reanalysis. Sulphate base models also validated those original outputs (RR=1.17), with the inclusion of spatial independence at a regional, and filtered provincial level respectively providing RR values of 1.19 and 1.09 (HEI 2000). Spatial analytic methods therefore identified associations between mortality and pollutant tracers to remain, but at a noticeably diminished level. Clearly, such evidence points towards a need for future epidemiological studies to either filter or explore the spatial relationships inherently present within datasets of a geographic nature, a process seldom achieved.

To date, perhaps the most influential pooling of urban hospitalisation estimates, in terms of short-term fluctuations in air pollutant concentrations, has been achieved within the framework of the National Morbidity and Mortality Air Pollution Study (NMMAPS). However, soon after its initial publication the projects thresholds were shrouded in controversy, as sensitivity analyses detected imprecise standard errors and an upward bias of effect estimates; produced by an insufficient default convergence criteria for Generalized Additive Models (GAMs) in S-Plus Ver3.4 (Dominici et al 2002). To combat these issues identical GAM functions with stricter convergence criteria were reapplied to the dataset, in addition to fully parametric solutions, consisting of Generalized Linear Models (GLMs) with quantile fitted natural cubic splines; which favourably provide unbiased estimates and suitable converge at the cost of a more rigid account of fluctuating climatic influences. Originally, the estimated effect of particulates on mortality from non-external causes, across 90 US cities, was associated with a

0.41% increase per $10\mu\text{g}/\text{m}^3$ of PM_{10} ; yet in the reanalysis under more stringent GAMs the estimate fell to 0.27%, and with GLMs, the effect reduced even further to 0.21% (Dominici et al 2003). Prior to these findings, GAMs were widely used in air pollution epidemiology often with the same lackadaisical selection of model characteristics, to the extent that a reevaluation of parallel European projects became necessary.

In the appraisal of 'The Air Pollution and Health: A European Approach 2' (APHEA2) projects inspection of particulate matter on hospitalisations across 29 European cities, effect estimates under different criteria appeared generally stable, reconfirming the projects central findings and validating its robust framework. Here, children's asthma induced hospitalisations were respectively associated with a 1.2% and 1.5% increase per $10\mu\text{g}/\text{m}^3$ of PM_{10} under the original and more stringent GAM schemes; meanwhile hospitalisations of the entire respiratory set made by the elderly were respectively associated with proliferations of 0.9% and 1.0% (Atkinson et al 2003). Still, this only acts to highlight our recent overdependence on studies with a temporal framework, which is of particular concern when evaluating an environmental phenomenon whose spatial parameters are of equal important in its distribution. To quell such discontent, it would appear necessary for researchers to conduct small-scale studies assessing the viability of existing spatial datasets and their associated techniques, prior to contemplating a large-scale project.

A recent 2004 Eurobarometer survey revealed air pollution to be the greatest environmental concern for 45% of European citizens, which is of little surprise considering its accountability for 370,000 premature deaths and an overall economic cost on health of 276-790 billion euros each annum across the EU (EC 2005). Whilst the post-industrial city has observed a rapid decline in airborne chemical concentrations with the out-of-town relocation of heavy industry, improvements in urban air quality soon appeared offset through a rise in unrestricted mobility, provided by the internal-combustion engine. At present, particulate matter (PM) is the most serious environmental health risk in the EU, with an estimated 21 % of the urban population exposed to concentrations higher than the EU limit ($40\mu\text{g}/\text{m}^3$ 24-hour mean) designed to safeguard health (EC 2012). Furthermore, the World Health Organisation recently ranked urban air pollution as the 13th highest contributor to global deaths (WHO, 2002), thus documenting the immediate importance of mitigating the detrimental impacts of environmental exposures on human welfare.

Extensive epidemiological research (of a temporal nature) drawn from across the UK, USA and Continental Europe has suggested an additional 0.75% of premature deaths and 0.80% of respiratory hospitalisations are caused per $10\mu\text{g}/\text{m}^3$ increment in (PM_{10})particulate matter $\leq 10\mu\text{m}$ in aerodynamic diameter (COMEAP 1998). Across the UK, anthropogenic

particulate matter air pollution is thought to annually contribute to approximately 29,000 deaths (COMEAP 2010). Meanwhile, the latest official government figures from within the UK, reveal personal exposure to air pollutants as accountable for up to 50,000 premature deaths per year, in addition to reducing the life expectancy of the general population by an average of 7-8 months (Environmental Audit Committee, 2010). Combining these figures the importance of PM₁₀ is revealed, directly influencing 58% of health outcomes, in addition to acting as a tracer pollutant for the vast cocktail of other detrimental pollutants emitted from combustion sources.

In recent decades various forms of road-transportation have been identified as the most significant emitter of pollutants, particularly in the urban environment, where transportation contributes to over half of the total emissions of NO₂ and PM₁₀ (DEFRA 2001, Harrison et al 2001). What's more, a UK Government panel of experts have shown that when particulate levels exceed health standards (50µg/m³), road traffic's contribution is typically in the range of 75-84% (QUARG 1996). Across the 25 EU Member States, the adverse health risk from particulates close to major roads is substantially above that of other urban sources, with 6.4% of EU citizens exposed to pollution >1% above the PM₁₀ limit directly caused by road-transport, and 13.7% of the EU population living ≤300m from a major road (EC 2006). Logically one would therefore argue for road transportation as a considerable burden of influence on respiratory health problems within the urban environment. Thus, it should be of necessity for both health and transport planners to further their understanding of such issues with the utmost importance, especially considering the forecasts of a continuously growing traffic fleet volume occurring across a global scale.

1.2. PROPOSED AREA OF RESEARCH

1.2.1. HEALTH EFFECTS OF TRAFFIC POLLUTANTS

Currently considerable efforts are being made by manufacturers to reduce vehicle emissions at the source, and by scientists to develop new technology to exploit newer and cleaner fuels including electric and hydrogen fuel cells. Existing levels of vehicular emissions are regulated through Euro 3, 4 and 5 vehicle's emissions standards, which have brought about drastic reductions in traffic emissions levels (OECD 2003). However, emissions are predicted to increase due to increased traffic growth (DfT 1997). A hydrogen economy holds the promise of reducing such emissions to a minimum, but this is not likely to have a considerable market share until 2035 (Jacobson et al, 2005). Reducing traffic emissions through traffic management

is therefore one of the key issues to be addressed in developing a sustainable, environment friendly urban transport system (DfT, 2004).

Such issues are starting to be addressed through legislation in the form of the Traffic Management Act (TMA 2004), which imposes a duty on local authorities to manage their traffic networks more efficiently and reduce traffic emissions. However the implementation of such strategies require large-scale data management with real time demands and computationally intensive modelling tools for evaluating traffic and air pollution levels. Furthermore, such tools necessitate a data platform for the decision makers to identify solutions resulting in both traffic network efficiency and air quality management. Subsequently, both researchers and planners are starting to adopt and implement the use of geographic information system (GIS) tools, in order to view and interpret the vast amount of data through a more accessible visual format, which may be used in conjunction with a detailed statistical analysis.

Increasingly, it has come to the attention of researchers and policy makers alike that the distribution of exposure to air pollution is not equitable, but this inequity has until recently received little formal epidemiologic attention (Naess et al 2007). Traditionally, time-based based epidemiological studies of air pollution have treated socioeconomic positioning as a confounding influence, removed by any available indicators in an attempt to achieve burden estimates independent of the social environment. Yet the confined nature of European intra-urban environments often determines those spatial variations in traffic pollutant levels, which tend to be closely associated with a plethora of social disparities. For instance, across England substantial demographic disparities are reported in relation to PM₁₀ exposure, with 20.3% of the most deprived decile residing within locations experiencing the highest 10% of PM₁₀ concentrations, compared to only 2.0% of the country's most affluent decile experiencing such burdens (DEFRA 2006). The importance of such socio-environmental interactions are detailed within Environmental Justice (EJ) research, which consistently report a 'double-burden' of deprivation and air pollutant exposure as a key explanatory factor in defining health disparities (Crouse et al 2009, Kingham et al 2007, Naess et al 2007, Namdeo & Stringer 2008, Wheeler & Ben-Shlomo 2005). Within the UK, this relationship between deprivation and exposure would appear most prolific across the 0-15 year age group, with population-weighted PM₁₀ exposures per child of 29.1µg/m³ and 22.8µg/m³ experience by England's most deprived and affluent demographics respectively (DEFRA 2006).

Although the environmental justice movement concerning air pollution has received increased attention in recent years, it should be noted that a majority of the early literature has tended to focus around the inequalities associated with industrial pollutants, particularly within a North American context (Morello-Frosch et al 2001, Hipp & Lakon 2010). Traditionally,

EJ research has also faced a plethora of challenges in causally associating environmental pollutants with adverse health outcomes, yielded through the absence of standardised assessment techniques and a tendency of measuring exposure via proximity to source rather than through actual pollutant distributions. Furthermore, the quantitative exploration of EJ matters through conventional multivariate regression is prone to obscure local variations in models of environmental equity. This is of particular concern, when considering that EJ is an explicitly spatial problem, concerning geographic elements rarely distributed in a uniform manner.

Thus far, a single study has applied appropriate spatial models that globally examine the adverse health risks from automobiles within a metropolitan area (Chakraborty 2009). Gilbert & Chakraborty (2011), also present the only EJ study to assess the local influence of social and environmental elements on areal health using a technique known as Geographically Weighted Regression (GWR). However in both circumstances areal health, in the form of cancer risk, was derived from Toxic Release Inventory coefficients combined with modelled emission rates; a method which presumes the detrimental effect toxins have within the study region. To avoid biased health outcomes future research should aim to use ground-truthed health datasets, comprising of conditions preferably recorded by medical professionals. Furthermore through mapping health, social and environmental intensities over time, it would be possible for local authorities to evaluate the wider geographic equity of implemented local transport and air quality action plans, which could then be adjusted accordingly.

1.2.2 POLLUTER PAYS ENVIRONMENTAL JUSTICE RESEARCH

Mitchell & Dorling's (2003) paper titled 'An environmental justice analysis of British air quality', presents the results of the first national study of air quality in Britain to consider the implications of pollutant distribution across over ten thousand local communities in terms of potential environmental injustice. Of particular interest is the way in which the paper attempts to tackle topical issues regarding the 'Polluter Pays Principles' (PPP), by investigating the role of personal vehicle emissions in the air-quality-poverty relationship via the use of 'static models' (actual population movements were not unaccounted for).

The subsequent data analysis consisted of simple plots of NO_x emissions created by (Static Model) and experienced within community Wards (National Atmospheric Emissions Inventory 1998 levels) in relation to poverty (Breadline Britain Index), grouped by deciles. Significant conclusions drawn from this project included:

- I. Those most affluent wards appear to be the least polluted.
- II. Poor and affluent areas both have the same polluting potential, with outputs from a higher volume of vehicles in affluent areas existing at a rate similar to those emissions emitted by the smaller fleet of aged vehicles typically found within deprived areas. Subsequently one could possibly conclude that a 'Polluter Pays' scheme already exists, with the poor contributing the most emissions per car.
- III. Strong inequality does occur with respect to NO_x in Britain, with deprived groups experiencing elevated burdens at their place of residence. This would imply that certain communities are not paying for their fair share of environmental contributions.

These findings would suggest that strong socio-environmental inequalities prevail throughout modern Britain, igniting the previously highlighted need for ethical groundwork prior to the implementation of future, traffic management schemes. Whilst Mitchell & Dorling (2003) establish this tangent of EJ research at a local rather than international level, further research is required. For instance, localised PPP issues have yet to be explored within the context of health outcomes, or across smaller intra-urban communities with highly variable demographics. In particular, intra-urban areas are thought to represent some of the most extreme disparities in socio-environmental attributes, and as such it is conceivable that their responses were previously smoothed out by this broader spatial analysis. In exploring whether these conclusions uphold across a complex urban environment, this project intends to develop upon Mitchell & Dorling's (2003) initial framework via a number of methodological improvements consisting of:

- I. A more realistic measurement of community contributions to the pollution problem are to be achieved through 'Dynamic' vehicle emission models, considering real-world population movements; thus allowing for a truer account of census area emissions.
- II. A direct comparison of emissions created with experienced National Atmospheric Emission Inventory (NAEI) road-transport emissions, rather than overall air pollution concentrations; so as to avoid the contributions from other emission and non-local transport sources.

- III. An examination of the 'Polluter Pays Principles' in greater detail through:
- Considering the relationships behind Pearce et al's (2010) 'triple jeopardy' of those wider social, health and environmental inequalities.
 - The incorporation of spatial techniques to hold communities to account, and to discover whether those most affected contribute to their own environmental downfall.
 - A wider examination of environmental attitudes, including the uptake of public and 'green' transportation modes, to see whether an environmental balance can be achieved.

Upon conducting such procedures, this body of research as a whole, can confidently say that it has geographically located and measured health effects and interactions of those vulnerable intra-urban populations, whilst distinctively holding select communities to account in an environmental context. It is intended that this project will display and validate the application of emerging spatial techniques within epidemiological fields, which in recent times have seen an over-reliance on temporal techniques.

1.3. RESEARCH AIMS

The thesis primarily intends to assess the burdens associated with transport derived air pollutants, across diverging sub-city communities within the context of Environmental Justice (EJ). Subsequently the aims of this research are as follows:

- I. To consider spatial variations in respiratory health, establishing their affiliations with airborne pollutants emitted from mobile sources, across the City of Leicester's high-resolution Lower Level Super Output Area (LLSOA) census blocks.
- II. To investigate how spatial variations in social-ethnic status relate to and interact with airborne pollutants emitted from mobile sources, across the model British multicultural City of Leicester. Thus, understanding the failure of temporal models in capturing Pearce et al's (2010) 'triple jeopardy', within a setting where environmental injustices are considered minimalistic.

- III. To dynamically assess the mobile polluting potentials of sub-city population groups, in order to ascertain whether those contributing towards the environmental degradation of the city, experience proportional environmental, social and or health burdens.

1.4. RESEARCH OBJECTIVES

As such, the objectives and targets of this research project are:

1. To determine whether respiratory hospitalisations geographically correlate to areas experiencing elevated annual air pollution levels, through a cluster analysis of mapped NAEI road transport emissions and NHS hospital records.
2. To determine whether ethnic minorities and/or deprived communities reside within locations experiencing reduced levels of air quality, through a cluster analysis of mapped NAEI road transport emissions with the various UK Census datasets.
3. To construct spatially suitable models for respiratory related hospital admissions, in order to ascertain the global relationship and significance of certain socio-environmental forces (i.e. Multilevel Modelling).
4. To construct spatially suitable models for respiratory related hospital admissions, in order to ascertain the local relationships and significance of certain socio-environmental forces (i.e. Geographically Weighted Regression Modelling)
5. To investigate whether spatial relationships exist between relatively minor and severe respiratory conditions, and if so, then to what extent do socio-environmental mechanisms play in the decline of respiratory health.
6. To detect, describe and analyse the existence of geographic boundaries between air pollutants, hospital admissions, deprivation and ethnicity in order to investigate whether multiple burdens exist with respect to environmental exposures.
7. To dynamically acquire emission rates for personal modes of transport across each of Leicester's Lower Level Super Output Areas (LLSOA's), through the use of workforce

population movement Origin-Destination matrices combined with DVLA citations of vehicle ownership.

8. To assess whether significant transport related pollutant levels are caused by local communities or through the movement of external social groups residing within other sectors of the city.

9. To assess whether social groups creating the greatest levels of transport emissions experience equally high environmental and or health burdens. Thus in effect examining whether either a traditional environmental or health based Polluter Pays scheme is already in operation. If this is not the case, a number of policy and personal schemes are to be considered with the intention to redress those imbalances.

1.5. THESIS OUTLINE

In total, this thesis consists of eight chapters. Following on from this overview of the research project, Chapter 2 provides an in-depth exploration of relevant background information, while Chapter 3 describes the datasets and methods selected to conduct a spatial analysis. The results of the spatial analyses are presented within Chapters 4, 5, 6 and 7, with the overall project conclusions and Leicester's local plan of action discussed in Chapter 8.

Chapter 2 opens with a brief historical overview of air pollution within the urban environment, depicting the decline in industrial operations and establishment of personal modes of mechanical transportation, whose emissions currently pose the greatest threats towards human health. Focus is placed on particulates formed as a result of incomplete combustion, exploring how the respirable fraction may bypass respiratory defence mechanisms, to directly agitate the hosts airways and lungs. Emerging literature, into the indirect influence prolonged exposure places on host resistance to infections, will also be considered as a function of prolonging and/or exacerbating preceding conditions. This will be followed by a review of the recent temporal epidemiological evidence linking particulates to public health amongst children, highlighting the limited level of research into acute respiratory infections; despite such ailments acting as the most common form of respiratory illness during childhood. The final section of the literature review will examine the environmental injustices of air pollution, through an appraisal of international and UK based EJ research, with specific focus placed on the use (or lack) of spatially appropriate procedures.

Chapter 3 initially provides the reader with a geodemographic account of the study area, the City of Leicester, focusing on why its multicultural assets are appropriate for an in-depth EJ enquiry. This chapter also provides information regarding the collection and preparation of the geocoded datasets used within this enquiry. After which, a general overview of the studies designs and selection of appropriate quantitative spatial procedures will be discussed, citing the use and recommendation of such applications from the literature where necessary. Pattern recognition strategies are presented as a means of capturing the magnitude and mutual location of spatial distributions, whose common spatial transitions and gradients may be further deliberated through the use of overlap statistics. Global regression procedures summarising interactions across the study area as a whole, include a traditional ordinary least squares (OLS) model with no spatial features (base), in addition to multilevel procedures which entail the removal of broad spatial structures. Meanwhile, localised regression procedures capturing and incorporating multiple spatial elements within model estimates are to be realised through Geographically Weighted Regression (GWR) modelling.

Chapter 4 concentrates on the first stage of this research project, examining the beneficial impacts, and or, burdens placed on a child's overall respiratory health by influential socio-environmental factors; realised in terms of hospitalisations across the entire respiratory set (ICD-10: J00-99). Here, Global and Local Indicators of Spatial Autocorrelation (GISA, LISA) statistically describe and illustrate the spatial nature socio-environmental influences and annual average hospitalisation rates for children residing within Leicester UA from 2000-09. Spatially appropriate modelling procedures, accounting for underlying geographical structures within the datasets, are then applied to define the extent to which socio-environmental variables of interest individually influenced respiratory health during childhood at global and local scales.

Chapter 5 expands on these initial findings, by exploring whether spatial relationships exist between specific relatively minor and severe respiratory conditions, and if so, what is the extent to which socio-environmental mechanisms play in the decline of a child's respiratory health. Specific focus is to be placed upon respiratory infections of the upper (ICD-10: J00-06) and lower (ICD-10: J20-22) respiratory tract, which are recognised as the primary cause of children's respiratory related complaints (58.52%) and portray a progressive decline in respiratory health. Here, spatially appropriate modelling procedures define the extent to which socio-environmental variables of interest individually influence relatively minor and severe respiratory complaints during childhood via shared pathways.

Chapter 6 moves onto the second stage of the research project, which entails the examination of spatial fields, in-order to establish whether critical distance-response

connections exist between respiratory and socio-environmental phenomenon. Rather than conducting a traditional inspection of health issues across artificially created buffers, the analyses is to be achieved with boundary statistics, describing naturally occurring shifts of magnitude in socio-environmental and health outcomes across the wider urban area. To date, distance-threshold techniques have solely explored the response environmental attributes, without considering the combined influence of additional social burdens.

Chapter 7 documents the third and final stage of the research project, which aims to advance our understanding of social, health and environmental injustices across the post-industrial cityscape, through developing upon Mitchell & Dorling's (2003) localised Polluter-Pays Principles. Here, levels of environmental accountability were measured in relation to community mobility and the uptake of various transportation modes, to assess the extent to which one pays for ones actions. In locating those most vulnerable, and holding specific communities to account, one intends to assist the decision making process of future transport policy makers; be that through a targeted incentive of environmentally 'friendly' transportation modes, or through suggesting a set of wider schemes to redress Leicester's environmental imbalances.

Finally, Chapter 8 presents the conclusions of the research project. Upon conducting the procedures set out in the previous chapters, this body of research as a whole can confidently announce that it has geographically located and measured (in a numerical and proximal form) those vulnerable intra-urban populations, whilst distinctively holding select communities to account in an environmental context. A partial solution is also offered to the prescribed problems of a medium sized post-industrial cityscape, where the adoption of drastic transportation schemes would likely impede a city's financial standing.

Whilst demonstrating the credibility of current spatial techniques within the fields of epidemiology and environmental justice, many limitations still remain. Certainly, temporal techniques fail to capture fleeting social-environmental interactions and are unable to locate those most at risk, yet they are able to display the sensitive nature in which environmental and climatic forces operate. As such, the project highlights a need for greater integration between the two approaches, which may only be found through the continued gathering of and construction of new databases. Despite addressing local environmental issues and opening up new avenues within the debate of social justice, this project should be primarily viewed as a benchmark for impending socioecological research of a spatial nature, until validated across multiple equally complex cityscapes. As such, several possible avenues for supplementary and additional research are also discussed here.

LITERATURE REVIEW

OVERVIEW

Through conducting a multidisciplinary review this chapter intends to provide the reader with an adequate understanding of the urban inequalities in respiratory health, attributed to environmental exposures specific to road-transportation. The chapter opens with a brief historical overview of air pollution within the urban environment, depicting the decline in industrial operations and its offset by personal modes of mechanical transportation. Subsequent focus is placed on particulates formed from incomplete combustion, exploring how the respirable fraction may bypass respiratory defence mechanisms, to directly agitate the host's airways and indirectly cause immunosuppressive responses, theoretically prolonging and/or exacerbating preceding conditions.

An overview of the recent temporal epidemiological evidence, linking particulates to public health concerns amongst children, highlights the limited level of research into acute respiratory infections; despite such ailments acting as the most common form of respiratory illness during childhood. Finally the chapter concludes with an examination of the environmental injustices of air pollution, with specific focus placed on the use (or lack) of spatially appropriate procedures. This is of particular concern, considering that environmental justice is an explicitly spatial problem, involving geographic elements rarely distributed in a uniform manner. Through conducting this review, one intends to define the gaps present within the literature, further clarifying the focus provided by the projects aims and objectives.

2.1. URBAN AIR POLLUTION

2.1.1. A RECENT HISTORY OF URBAN AIR POLLUTION

A variety of chemicals are emitted into the air from both natural and anthropogenic sources on a daily basis, with the latter potentially working to unbalance the fine-tuned atmospheric cycles operating on planet earth. Nevertheless, the formation of anthropogenic air pollution has only rapidly increased in our relatively recent history since the forthcoming of industrialisation. A period in which western civilisations powered by the extensive use of coal, observed rapid levels of prosperity and social change, accompanied by equally intense deteriorations in environmental and societal health. Yet, these industrial processes were soon accompanied by mounting concerns of the gentry, initially addressed within the UK through the 'Railways Clauses Consolidation Act' (1845), legislating for locomotive engines to consume their own smoke; similar rudimentary constraints were soon applied to factory furnaces in 'The Town Improvement Clauses Act' (1847).

By the 1920's, technological strides including the electrification of many urban railway terminals and factories, witnessed a considerable transfer of urban pollutants to a few point sources; yet public attitudes and an inadequate understanding of airborne by-products, impeded further legislative change (Boubel et al 1994). A heightened state of public concern was to arrive only in response to early industrial episodes, like the Muese Valley Incident of 1930 and Great London Smog Disaster of 1952, triggered by a combination of winter weather and poor practices. In the United Kingdom, the London smog of December 1952 proved a turning point in the history of air pollution and attempts at its control. A vast quantity of research into this episode attributes a rise in daily average concentrations of sulphur dioxide ($\leq 3500 \mu\text{g}/\text{m}^3$) and smoke levels ($4000 \mu\text{g}/\text{m}^3$), to have caused 4,000-12,000 excess deaths in the period immediately following the event (Appendix A1); undoubtedly highlighting the associations between a deterioration in public health with increased pollutant concentrations. Whilst all age groups were affected, infants and the elderly were found to be most at risk, with the main causes of death occurring in response to respiratory and cardiac disease.

Because of such incidents, major efforts have been made to reduce air pollution within the European Region in recent decades through legislation, primarily focusing on tackling the problem of industrial pollutants. Most significantly, 'The Clean Air Act' (1956), introduced 'smoke control areas' in some towns and cities across the UK, in direct response to the London smog of 1952. This legislation included the relocation of power stations away from densely populated areas, in addition to raising the height of industrial stacks, to mitigate future risks of fumigation under select climatic conditions. Residential properties were also encouraged to obtain their sources of heat from cleaner forms of coal, and alternative energy sources (i.e. gas

and electricity supplies). In recent years, European air quality directives adopted by the European Commission (EC), have also placed legal obligation on member states to achieve set limit values for individual pollutants by specified dates. As part of this scheme, member states, including the UK, are required to undertake air quality assessments, reporting their findings to the EC on an annual basis. The first European directives date back to 1980, in the setting of ambient air quality limits for sulphur dioxide and suspended particles (EC 1980).

Through strict legislation, industrial emissions declined significantly across urban areas, with the most pronounced effect observed for sulphur dioxide (SO₂), which reduced by approximately 50% in the period 1980-1995 (WHO 2000). Within the UK, levels of smoke, measured by the blackness of filters through which air passed, were also found to decline from 175µg/m³ in 1958 to 75 µg/m³ in 1968 (Royal Commission 1971). However unfortunately, the reduction of smoke and SO₂ levels within the city brought about by the Clean Air Act (1956) and its surrounding legislation, was soon offset by an increase in CO, NO_x, PM₁₀ (constructed from PAH, PAN, Pb, Br, Cl) and O₃ concentrations, as a direct result of the ever-increasing volume of motor-vehicles (Alloway & Ayres 1997). This phenomenon is best observed through annual motor-vehicle sales within the US, recorded at 4,192 in 1900; 4,265,830 in 1925; and 8,003,058 in 1950, a figure from which annual sale figures have seldom deviated (Boubel et al 1994). Whilst Europe was experiencing a decline in the intensity and frequency of industrial driven 'winter smog' events, stateside cities typically of a subtropical temperament, began to experience photochemical episodes largely attributed to the rise of the automobile, circa 1950. Photochemical smog is a unique type of air pollution, in which secondary pollutants are formed via sunlight-driven oxidation reactions, converting NO_x into O₃, with reactive hydrocarbons and NO_x collectively resulting in the formation of peroxyacetyl nitrate (PAN). Both O₃ and PAN are strong oxidants, of considerable phytotoxic risk, with the potential to aggravate the mammalian respiratory tract.

In particular, the effects of photochemical episodes were heavily documented throughout California's South Coast Air Basin (SoCAB), with persons often complaining of headaches and irritations of the eyes, throat and chest (minor restricted activity day [MRAD]). For instance, ambient exposure to 'Los Angeles Smog' (0.165ppm O₃, 227µg/m³ total suspended particulates [TSP]) was observed to decrease the FVC (Forced Vital Capacity) of 60 non-asthmatic exercising adults by 3.45% (Avol et al 1983). In-fact, major health incidents (RAD) appeared almost exclusive to persons with existing conditions, with smog days accounting for a 14% variation in the number of asthma attacks (Schoettlin & Landau 1961). Nevertheless, a 1989 regional benefit assessment of California's SoCAB, recorded up to 17 days per year in which O₃ levels exceeded critical standards (Max 1hr >0.12ppm), with PM₁₀ exposures associated with such events (Max 24hr >150µg/m³) increasing the risk of death to

1/10,000 (Hall et al 1992). The annual economic value of avoiding the health impacts of O₃ was estimated at \$2.7 billion (MRAD, RAD), with the impact of PM₁₀ priced at \$6.4 billion (RAD, mortality); which in total equated to an annual cost of \$750 per person (Hall et al 1992).

Whilst markedly beneath pollutant levels of classic pollutant episodes, such research identifies the continued threat pollutants pose to one's wellbeing via monetary contexts, which naively demonstrate the newfound importance of the individual in post-industrial societies. Still exploring health in a financial term highlights the prominent role our personal actions would now play in the destruction of one's local environment, the intensity of which remains largely fashioned by natural climatic forces. In particular, the respirable fraction of particulate matter (PM₁₀) appeared key to this new type of air pollution, primarily derived from the combustion of oil based vehicular fuels. Not only were increments of PM₁₀ strongly linked to a constellation of systemic health effects, unlike other pollutants these associations appeared robust to weather variations, with discernible effects present even when other pollutants such as SO₂, O₃ or acid aerosols are virtually absent (Bates 1999). What's more, extensive epidemiological research drawn from across the UK, USA and Continental Europe, identify a prevalence of health concerns even at ambient concentrations, with current guidelines suggesting a 0.80% rise in respiratory hospitalisations per 10µg/m³ of PM₁₀ (COMEAP 1998). In contrast, those impacts of the second most crucial element of vehicular emissions, ground-level O₃, appear focused towards vegetation; reportedly causing over 30% losses in certain commercial species across California's SoCAB (Grantz & Shrestha 2005). Thus, it is of little surprise that the pollutants of most concern today, within the EU, are ground-level O₃ and particulate matter (EC 2005).

To combat developing environmental nuances attributed to the motor-vehicle, the initial 1980 European Commission Council directives on ambient air quality were soon extended, to incorporate nitrogen dioxide (EC 1985a). Critically around this time, the EU also introduced environmental specifications applicable to fuels, prohibit the marketing of leaded petrol within all member states by 2000 (EC 1985b); thus eliminating a particularly toxic fraction of PM₁₀. Later, in 1999 air quality limit values were revised and tightened, with further regulation occurring more recently in 2008 (EC 1999, EC 2008). In particular, the implementation of Euro Standards on motor vehicles during the 1990's has played a key role in the reduction of directive pollutants. For example, a petrol-car of Euro IV Standard (2006) emits levels of CO, NO_x and Hydrocarbons at rates approximately 96%, 97% and 98% respectively lower than a Pre-Euro Standard (>1990) vehicle (OECD 2003). However, even with these constructive reductions in air pollutants driven through ever-evolving legislation, trends in concentrations of urban air pollutants related to mobile sources (including particulates and nitrogen dioxides) remain less clear, as vehicle fleets increase. An issue highlighted by only 2

out of the 27 EU Member States achieving PM₁₀ and NO₂ limits across urban areas, in time for their respective deadlines of 2005 and 2010 (EC 2008). Still, findings from recent epidemiology studies have foreseen these pollutants to constitute a risk to human health even at low concentrations, with some pollutants potentially yielding virtually non-existent safety thresholds. Thus, there remains a need for continued environmental scrutiny, particularly when adequate attention is yet to be paid directly towards road-transport, the primary source of pollution within the post-industrial cityscape.

2.1.2. AN OVERVIEW OF THE MOTOR-VEHICLES DIRECT CONTRIBUTION

A vast majority of the transport sector with a few omissions are responsible for the release of a variety of air pollutants, derived from processes involving the combustion of liquid fossil fuels. Consequently, a majority of transport sources emit similar pollutants, making it extremely difficult to distinguish contributions from specific forms of transportation, especially in regions where integrated transport systems are within close proximity. However, it is feasible to allocate emissions proportionately in relation to concentrations and composition, which varies according to fuel composition and combustion conditions. Alternatively, one may approach such issues via emission inventories, which yield a more accurate description of the individual contributions, whilst neglecting meteorological parameters and thus the full account of its spatial distribution.

Motor vehicle pollutant sources include emissions from the exhaust pipe, blow-by from the engine crankcase, fuel evaporative emissions from the fuel tank and carburettor, as well as particulate emissions from the wear-and-tear of tyres and breaks (OECD 1988). Major pollutants emitted from fossil-fuelled vehicles consist of carbon monoxide (CO), nitrous oxides (NO_x) and volatile hydrocarbons emitted as vapour in the exhaust, or particulate matter (PM₁₀) derived primarily from incompletely burnt fuel. The most significant transport emissions to the atmosphere by mass are carbon dioxide and water vapour from the complete combustion of fuel (Colvile et al 2001). However, the toxicological effects of carbon dioxide (CO₂) on human health, including drowsiness and in extreme circumstances unconsciousness, only start to occur at phenomenally high concentrations (>10,000 ppm); hence the concerns of CO₂ are purely environmental. Furthermore, it is often very difficult to attribute CO₂ directly to a source of creation, let alone a specified form of transportation, due to its distribution existing in vast quantities from a number of potential sources (Figure 2.1A). Therefore, CO₂ should not be deemed suitable as a tracer pollutant for the impacts of the various pollutants produced by motor-vehicles.

A feature that distinguishes other mobile combustion sources from almost all stationary sources, however, is that combustion is incomplete (Colville et al 2001), thus resulting in the production of volatile hydrocarbons and carbonaceous particles which can be chemically linked to specific forms of transport (Figure 2.1B). Consequently, particulate matter is often used as a marker to examine the effects of particular forms of transport on human health.

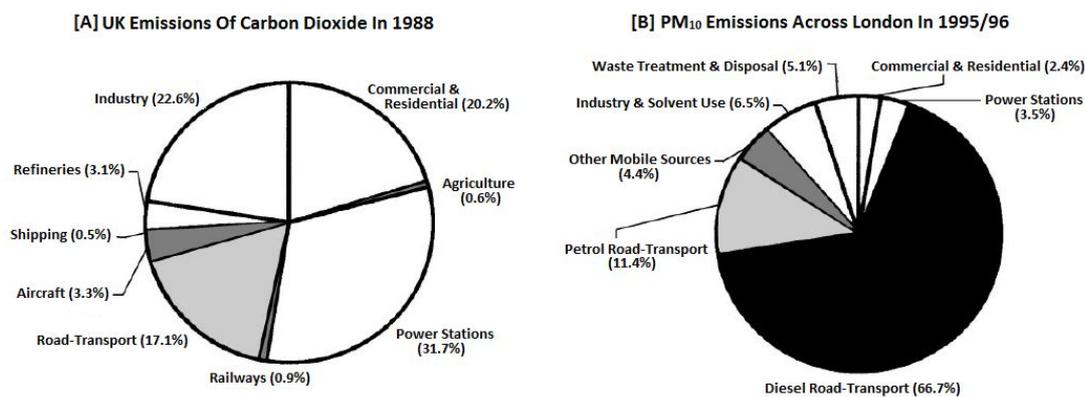


FIGURE 2.1: Carbon related UK emission inventories (Adapted from: Colville et al 2001)

In addition to hydrocarbons, most fuels contain impurities such as sulphur, which either oxidises into SO_2 on combustion or forms particulate accumulations in the engine, in the form of sulphate. However, the major source of sulphur emissions derive from impure fossil fuels used at point sources, with natural gas, petrol and diesel fuels containing relatively low sulphur contents (Figure 2.2A). Consequently, sulphur emissions from transportation pose a relatively limited threat to human health.

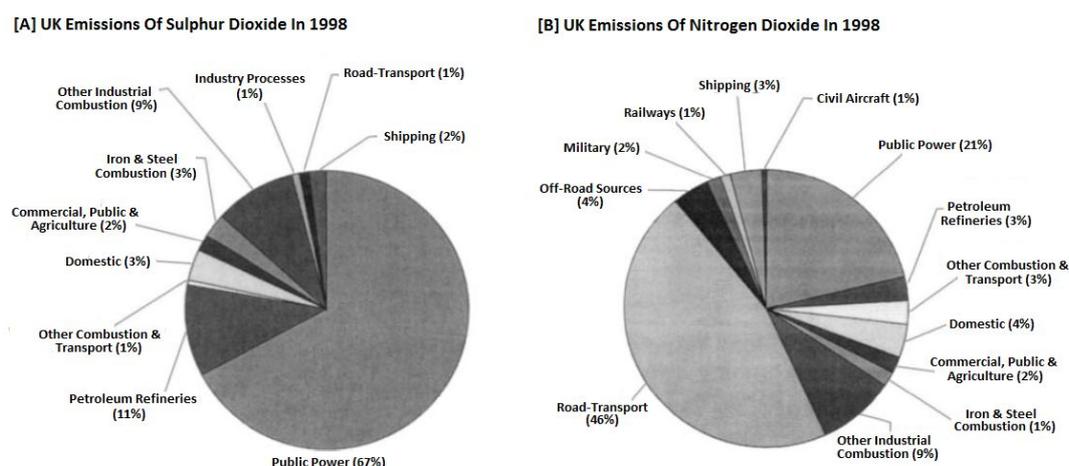


FIGURE 2.2: SO_2 and NO_2 UK emission inventories (Adapted from: Harrison et al 2001)

Other pollutants originate from the high combustion temperatures occurring in the engine, which is responsible for the oxidation of atmospheric nitrogen into nitric oxide (NO) and

Nitrogen dioxide (NO₂). Road transport is a major contributor of various nitrogen oxides, accounting for 46% of the total UK's NO₂ emissions (Figure 2.2B). As with particulates, the motor-vehicle contributions towards the overall levels of airborne nitrogen dominate the urban landscape, accounting for up to 85.0% of NO_x in London (Blair et al 2004). Consequently, nitrogen oxides are integral pollutants in vehicle emission investigations due to their high emission correlation rates at both street level and regional monitoring stations.

In 1988, road-transport was calculated to typically account for 100% of CO levels, for at least 60% NO_x and hydrocarbon levels, for about 10% of SO₂ and 50% of particulate levels, in the centre of an average built up area; with the street environment encouraging either the containment or dispersion of such pollutants (OECD 1988). Interestingly, an assessment of transport emission trends from 1990-2007 undertaken by the US Environmental Protection Agency (EPA), documented levels of CO, non-methane hydrocarbons (NMHC), NO_x and PM₁₀ to have all fallen by 54%, 48%, 27% and 34% respectively during this timeframe (HEI 2009). A similar response is expected to have occurred in a European setting over this timespan, as an outcome of the Euro Emission Standards, which have seen significant pollutant reductions in both petrol and diesel vehicles (Table 2.1). However, this decrease in emission rates from motor vehicles is likely to have been offset by changing trends in world fleets, as an upshot of increased rates of social wellbeing resulting in increasing rates of population growth, urbanisation, economics and urban sprawl. Subsequently, since 1990, approximately 27 million additional motor vehicles have been added to the world highways each year (HEI 2009), a factor likely to counterbalance any improvements in vehicle emission technologies.

Type of vehicle	Pre-Euro I	Euro I	Euro II	Euro III	Euro IV	Percentage change
	1990	1993	1997	2001	2006	
Petrol car						
Carbon monoxide	100	15	10	7	4	-96
Hydrocarbons	100	9	4	3	2	-98
Oxides of nitrogen	100	19	9	6	3	-97
Particulates	5	2	2	2	2	-55
Diesel car						
Carbon monoxide	7	4	3	2	2	-71
Hydrocarbons	10	4	3	2	1	-90
Oxides of nitrogen	43	29	21	13	7	-84
Particulates	100	55	31	20	10	-90
Semi-trailer truck						
Carbon monoxide	44	22	18	9	7	-84
Hydrocarbons	183	87	78	47	33	-82
Oxides of nitrogen	1 704	893	650	461	325	-81
Particulates	700	482	185	124	24	-97
Bus						
Carbon monoxide	63	28	22	11	8	-87
Hydrocarbons	83	90	84	50	35	-58
Oxides of nitrogen	795	859	614	436	307	-61
Particulates	458	304	187	125	24	-95

By fuel and emission standard on an urban test cycle, per vehicle-kilometre
Index Pre-Euro I gasoline car = 100, except for particulates

TABLE 2.1: The staged development of Euro Emission Standards, as a control measure from road-transport's environmental contributions across Europe (OECD 2003)

Interestingly, this trend for increased motor-vehicle usage and its subsequent increase in pollutant levels, does not appear to be restricted solely to developing countries. A fact illustrated by the United Nations Framework Convention on Climate Change (UNFCCC) in 2006, who noted that across developed countries, GHG emissions from the transport sector had grown by 16% from 1990-2006 (HEI 2009). Furthermore, national mobility studies predicted that the 2002 total distance driven by UK residents of 540 billion km, will have risen to 653 billion km per year by 2010 (Colls 2002). As previously discussed, such factors are likely to impede current emission abatement technologies.

In either scenario, one can conclude that the motor-vehicle remains the central underlying cause for urban air pollution within a European setting. A case perfectly illustrated by the City of Oxford's 'Air Quality Management Area' (AQMA). Within this AQMA, Oxford City Council recognised the motor-vehicle to significantly affect particulate levels in addition to accounting for on average 60-65% of NO_x emissions throughout the city, increasing to 80% of NO_x levels at city centre locations (Oxford City Council 2006). In addition, 2004/05 urban roadside monitoring at Marylebone Road in London, revealed passing traffic to be directly responsible for 85% of urban NO_x and 40.2% of urban PM₁₀ emissions for this particular street; with other local sources accounting for only 1.9% of PM₁₀ emissions (Blair 2004). In viewing Figure 2.1, one may observe that a substantial amount of urban background particulates also originate from road-transportation (78.1%), of which diesel vehicles have the greatest impact. Whilst both pollutant concentrations appear substantially determined by localised forces, the heightened spatial sensitivity of gaseous pollutants is problematic for use as a marker of road-transport, considering the resolution restrictions imposed by accompanying datasets that experience geographic anonymisation (i.e. hospital records). Meanwhile, PM₁₀ offers a more forgiving spatial description of local processes, in addition to describing the emission component thought to cause most harm. Based on this information, it would be most appropriate to use particulates as the surrogate measure of road-transportations overall influence within the following geostatistical analyses.

It is predicted that for at least the next decade or two, conventional diesel and petrol engines will remain the dominant technology of the automotive sector, throughout Europe. What's more, trends indicate that diesel-powered vehicles will continue to increase their share of the market, as they continue to attract the thrifty motorist by boasting a superior fuel economy and lower (carbon focused) taxation charges. Although, commanding a premium price from retailers, diesel cars generally offer a more efficient use of energy than their petrol counterparts do, with such vehicles now reaching a stage where negligible differences exist in ride quality. For instance, a medium sized diesel vehicle (Vauxhall Astra) initially costs the consumer an extra £2,370, yet under real-world urban driving conditions, annually requires

£500 less in fuel with a payback period of 4.7 years (Consumers' Association 2013). In the period 1990-2020, diesel's share of the market is expected to increase from 40% to 59% at the expense of petrol's share, with alternative fuels predicting only a modest penetration (0.3%) into the market by 2020 (Figure 2.3).

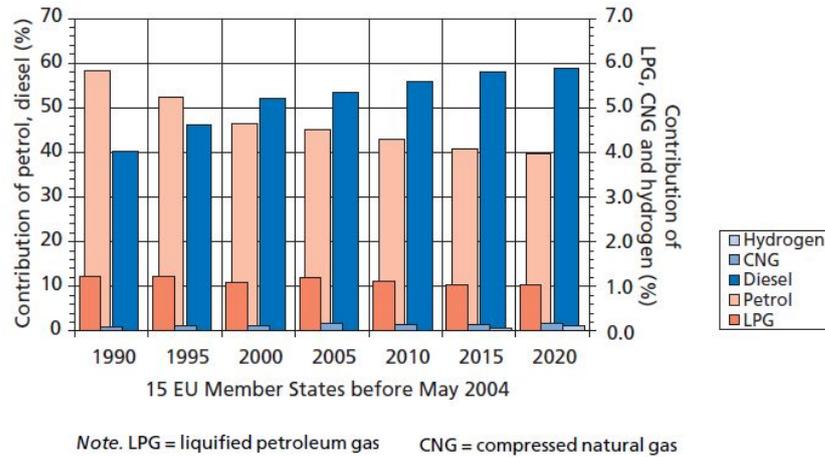


FIGURE 2.3: Fuel use in the road-transport sector, recorded by the 15 founding EU Member States, projected from 1990 to 2020 (Adapted from: WHO 2005)

In the past, petrol engines were recognised through emitting high levels of NO_x, a pollutant compared to particulates as the lesser of two ills on human health (WHO 2005). As petrol engines maintain a stoichiometric ratio between fuel and air, incomplete combustion and thus the formation of particulate emissions are rare; a factor favouring the uptake of three-way catalyst aftertreatment devices which, eliminate virtually all toxic gases leaving the tailpipe (Table 2.1). Because of the different operating characteristics of diesel engines (i.e. oxygen rich environment, high soot content), it is impracticable to use the same catalyst technology to clean up diesel exhaust gases, which continue to contribute modest levels of NO_x after exhaust recirculation measures (Table 2.1). Whilst exhaust gases rarely reach temperatures required to combust soot (>500°C), some gains have been achieved through particulate filters which may also be retrofitted to older vehicle stock. Based on these outputs and their amplified level of uptake, diesel emissions will play a proportionately greater role in urban air pollution in the coming years, a key feature of which are their particulate contributions.

There are three peaks (or modes) of distribution for airborne particles, with primary exhaust particles typically occupying the transient nuclei mode, formed through the condensation of hot vapours (including hydrocarbons, nitrates, sulphates and VOC's). Fine carbonaceous particles (PM_{2.5}) also generated from the combustion process, provide a stable site for accumulation of these reactive species, potentially creating a product of a highly toxic nature. As previously stated, Figure 2.4 confirms the petrol car to be a negligible source of particulate emissions from the exhaust, with unregulated vehicles providing the most

significant outputs, although few of these vehicles exist within a European setting. Vehicle class would also appear to play an important role, with larger diesel passenger vehicles currently emitting similar levels of condensed volatile materials to an unregulated vehicle, whereas this fraction appears drastically reduced amongst smaller diesel vehicles. Nevertheless, the continued importance of particulate emissions from diesel vehicles remains clear.

For all motor-vehicles, the respirable fraction of coarse particles ($PM_{2.5-10}$) chiefly originating from the brake pads and clutch, often contains metals (such as iron, copper and lead) and organic materials known to cause harm at a cellular level. These coarse particles also offer a favourable site for interactions amongst transient nuclei, typically of diesel origins. In fact, non-exhaust particle sources will become increasingly important as diesel exhaust emissions decrease, through improved technology entering the market. For instance, a Euro I (1992) diesel vehicles particulate contributions from the exhaust, brakes, tyres and clutch were respectively recorded at 91%, 3%, 3% and 2%; whereas for a Euro IV (2005) vehicle these values exist at 60%, 15%, 14% and 9% (WHO 2005). Thus reinstating the importance of evaluating the entire respiratory fraction of particulate matter emitted from anthropogenic sources, despite recent academic focus towards the finer end of the spectrum.

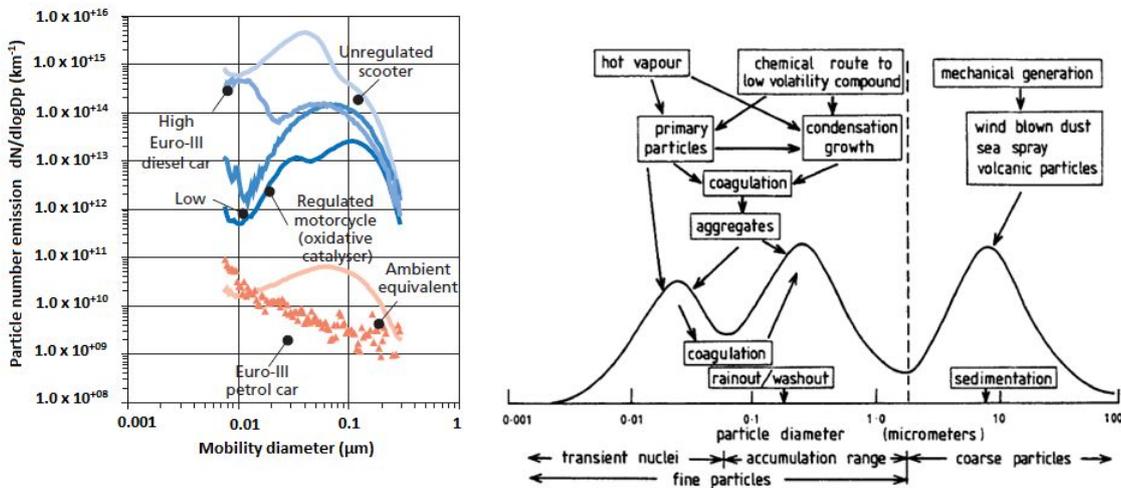


FIGURE 2.4: Typical size distributions of exhaust particulates by vehicle type, in context to generalised formation mechanisms (Adapted from: Ntziachristos et al 2003, Harrison et al 2001)

2.1.3. HEALTH BURDEN OF URBAN AIR POLLUTANTS

Numerous investigations since the 1970's have suggested that air pollution may cause severe long-term as well as short-term effects on human health. Whilst marked improvements in air quality standards have been experienced within the developed world over recent decades, many cities are now experiencing an influx in the levels of motor-vehicles, which aim to offset

targets for key pollutants such as nitrogen oxides (NO_x), ozone (O₃) and particulate matter (PM). All of which, on an individual and collective basis, pose a substantial threat towards the human respiratory tract.

2.1.3.1. HEALTH IMPACTS OF PARTICULATE MATTER (PM)

Particulate matter consists of a variety of solid or liquid particles found in the atmosphere, which vary in chemical compositions related to their creation process. Coarse particles (>2.5µm) are often derived from natural processes, including silicates and carbonates from windblown dusts, and thus posing little harm to human health. Still certain exceptions to this rule remain in the urban environment, where incomplete combustion or the mechanical wear of manmade materials occurs. In contrast, fine particles (PM_{2.5}) are derived chiefly from local anthropogenic combustion processes, due to their relatively short residence times (Pope 2000). Across most urban areas, PM_{2.5} comprises of primary-source particles and secondary combustion particles consisting of nitrates often derived from local street sources. As such, fine particulate matter may itself be toxic or carry carcinogenic substances absorbed to its surface.

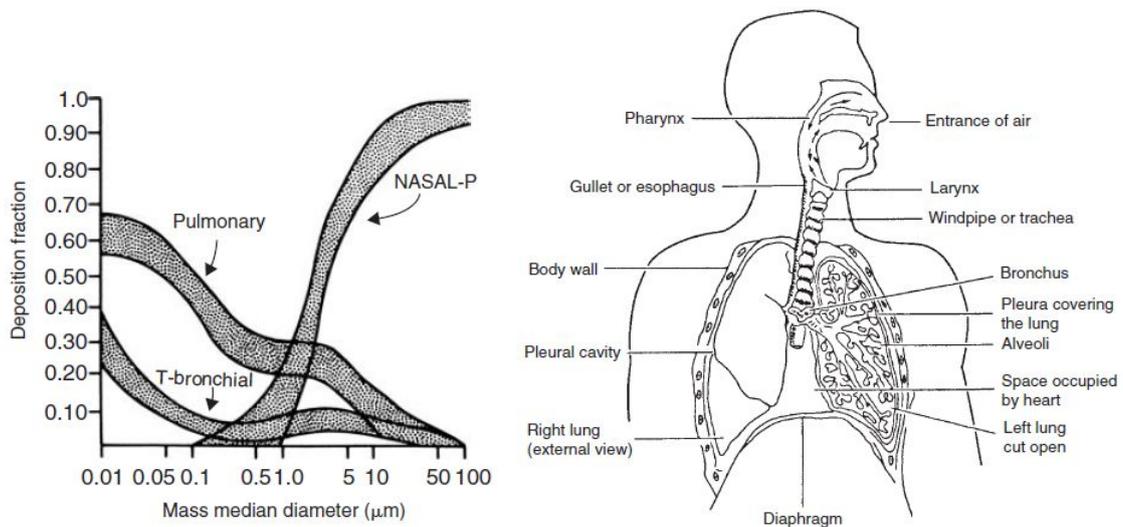


FIGURE 2.5: Particulate deposition in relation to the respiratory system. The nasopharyngeal region consists of the nose and throat; the tracheobronchial region consists of the windpipe and large airways; and the pulmonary region consists of the small bronchi and the alveolar sacs. (Adapted from: Boubel et al 1994)

Whilst all particles may aggravate the linings of the respiratory tract, diesel particulates are of particular concern, due to their carcinogenic properties caused by high levels of trace metals, and small size (<2.5µm) which allows passage through the narrowest lung passages (Figure 2.5). However, PM_{2.5} measuring equipment is often limited to only a few urban stations, therefore direct correlations between motor vehicles and human health has to be indirectly associated through measurements of PM₁₀. Still COMEAP exposure-response coefficients

across London have revealed citywide background pollution levels as responsible for 1.66% of all mortalities; a figure rising to 2.5% in locations within close proximity to busy road networks (Namdeo & Bell 2005). Furthermore, DEFRA's expert panel on air quality standards found a 4% rise in daily asthmatic symptoms to occur per $10\mu\text{g}/\text{m}^3$ of PM_{10} originating from motor-transport (EPAQS 1995).

2.1.3.2. HEALTH IMPACTS OF NITROGEN OXIDES (NO_x)

Nitrogen oxides are a class of compounds that have a variety of direct and indirect effects on human health, of which NO_2 poses the greatest concern. Acute exposure to NO_2 decreases gaseous exchanges in the blood and increases respiratory symptoms producing lower lung function values, with further irritation resulting in the swelling of tissues or irreversible lung diseases such as emphysema (OECD 1988). Even short-term exposure within sensitive groups such as children can result in a range of respiratory problems including coughs and sore throats. It has been found that levels as low as $120\mu\text{g}/\text{m}^3$ (0.1ppm) per hour can cause adverse effects on asthmatics (OECD 1988).

This evidence is further supported by COMEAP (1998), who reported NO_2 to result in acute health issues at an exposure-response coefficient of 0.5% per $10\mu\text{g}/\text{m}^3$. In following this exposure-response coefficient, NO_2 would be accountable for a 2.5% increase in the current base rate of London's Respiratory Hospital Admissions (RHA) solely because of background pollution levels, with perhaps a 4.18% increase in RHA near busy roadside locations (Namdeo & Bell 2005). Interestingly, NO_x can be closely correlated to roadside particulates (Appendix A2), further murkyng the separation of the pollutant's individual health effects across urban environments. Upon facing this challenge, it is proposed that a single pollutant should be used as a tracer, representative of the collective impact of road-transportation pollutants. Favourably, in focusing on the pollutant thought most detrimental and representative of the wider mix, one obtains a conservative estimate of source specific impacts; in that the broad trend will be captured, but inevitably some contaminants from a complex source will remain omitted.

2.1.3.3. HEALTH IMPACTS OF OZONE (O_3)

Ozone is a secondary pollutant created through a series of photochemical reactions involving precursor pollutants, such as the oxides of nitrogen (NO_x) and volatile organic compounds (VOCs). Such reactions are cyclic in nature particularly within the urban environment, with pollutants contained in street canyons undergoing a series of conversions between oxides of nitrogen and ozone (Appendix A3).

As ozone concentrations increase above the guideline value, health effects at the population level become increasingly numerous and severe. Such effects can occur in places where concentrations are currently high due to human activities or are elevated during episodes of very hot weather. Prolonged periods of stagnation and intense sunlight coupled by vast transport fleets and heavy industry have been held accountable for the numerous severe photochemical smog incidents across American coastal cities particularly during the 1970/80's. A retrospective study of the 1988 and 1989 New Jersey photochemical smog episodes recorded elevated ozone concentrations of 140-150 $\mu\text{g}/\text{m}^3$ to be accountable for approximately 13.2-15.4% (95% CI) of asthma hospitalisation incidences (Cody et al 1992). Still, across Europe photochemical smog events are yet to be experienced at such magnitudes.

Ozone unlike its precursors NO/NO₂ that only have residence times of a few days, may affect the environment at a regional scale with residence times over hundreds of days (Harrison et al 2001). Subsequently ozone is viewed as transboundary pollutant, whose precursors are often emitted within an urban setting, radiating into the surrounding region if favourable conditions prevail. Consequently, ozone remains a difficult pollutant to regulate and control without complete International cooperation. The current Air Quality Strategy for England, Scotland, Wales and Northern Ireland sets the current standard for ozone exposure levels at 100 $\mu\text{g}/\text{m}^3$, measured as a daily maximum of a running 8-hour mean (DEFRA 2007). A figure, defined through extensive controlled chamber tests into the transient changes in lung function and lung inflammation, within healthy young adults undertaking intermittent exercise.

At present tests have revealed exposure to ozone levels of 0.06 ppm (115-120 $\mu\text{g}/\text{m}^3$) to result in a 2.85% reduction in a participants FEV₁ [forced expiratory volume in 1 second] (Brown 2008). Furthermore, there is some evidence that long-term exposure to ozone may result in chronic health effects, however the evidence is not sufficient to recommend an annual guideline (WHO 2006). Whilst road-transport is heavily involved within the formation of ozone, this pollutant forms in distant locales and is primarily an agricultural concern, only occasionally burdening those individuals with existing conditions. As such, ozone will not be the focus of this enquiry, analysing the impact of the motor-vehicle on respiratory health within an urban setting.

2.2. HUMAN RESPIRATORY SYSTEM

2.2.1. BIOLOGICAL MECHANISMS OF LUNG INJURY CAUSED BY PM₁₀

In the process of exchanging 10,000 - 20,000 litres of air each day, the human respiratory system is exposed to a multitude of potentially harmful foreign substances and microorganisms (Seaton et al 1989, Weinberger et al 2008). Initially the upper respiratory tract (nose and pharynx) acts to warm, humidify and filter the air before it reaches the delicate lungs for gas exchange to occur. Here, the nasal passages act as the first line of defence, in which hairs and mucus provide a physical barrier, which particles adhere to. The branching passage of airways from the bronchi down to the respiratory tree continues to encourage the deposition of particulates, where they may be removed via the mucociliary escalator. Within the lower respiratory tract, mechanical clearance processes are replaced by a sole dependency on humoral and cellular responses, to quarantine (inflammatory response) and or destroy foreign substances, prior to their removal back up the respiratory tract. During either stage, the toxic nature of particulates may prove harmful, yet even the presence of an insoluble particle has the potentially to stimulate and unbalance a host's immune response. Here, the excessive release of antimicrobials has the potential to inflict severe damage upon healthy cells within the nearby vicinity, if left unchecked (Cumming & Semple 1980, Seaton et al 1989, Weinberger et al 2008, Ward et al 2010). Upon understanding how the respiratory immune system functions, this section aim to explore in detail the mechanisms by which PM₁₀ achieves its adverse conclusions across specific target tissues.

The anatomical structure of the nasopharynx, by enlarge prevents the passage of materials towards the coarse end of the spectrum of respirable particulates; with only 8-21% of particles 10µm in diameter (typical of mechanical brake and clutch wear) entering the large airways, and 0-21% reaching the bronchial tree for clearance via mucociliary processes (Figure 2.5). Yet, along the airway walls and mucociliary escalator lie several potential targets for particles, including the smooth muscle cells and mesenchymal cells, which perform functions essential for continual tissue development. Furthermore, it may take up to 40 minutes for mucus from large bronchi to reach the pharynx (Ward et al 2010), an ample period for such interaction to occur.

For particles 2.5µm in diameter, anatomical structures have a diminished influence, with 32-58% of all these particles leaving the large airways; it is here that the mucociliary escalator takes over, to clear 22-32% of the total fraction from the tracheobronchial region (Figure 2.5). Still one should note that 0-36% of inhaled particles 2.5µm in diameter remain free within the terminal airways, despite the presence of mechanical clearance processes. Thus in the terminal airways, macrophages play an important role in the removal of particles by

phagocytosis ('cellular eating'), which eventually migrate to the start of the mucociliary escalator, where they leave the lung with their cargo of particles bound for the gut (Donaldson & MacNee 1998).

Beyond the ciliated airways, the net flow of air is zero, and as such, the deposition efficiency for extremely small particles rapidly increases as diffusion processes take over. For particles 0.01 μm in diameter, 57-68% of this inhaled fraction deposits within the bronchial tree (Figure 2.5). Whilst their size may beneficially assist in deposition processes, this is also of detriment, as a larger surface area increases the likelihood for adverse interactions to occur, with their small size potentially enabling passage into the pulmonary interstitium and lymph nodes. If particles cross the epithelium they are no longer likely to be cleared by normal processes, either remaining in the subepithelial regions close to key response cells, or migrating towards the draining lymph nodes; the site in which lymphocytes grow and mature. The effects of dust in the lymph node are not known, however adjuvant effects are to be anticipated (Donaldson & MacNee 1998). One should note that granulocytes (white blood cells) release proteases and reactive oxygen species during phagocytosis, which if occurring in the interstitium could impair host cells.

Cardiovascular deaths are thought to be another important adverse health effect of PM₁₀, classically caused by blood clots in coronary vessels (heart attack) and the brain microvascular (stroke) (COMEAP 2006). Whilst the effect of inhaled particles on the respiratory tract is understandable, the link between airway depositions and an increased likelihood of clotting is tentative at best. Donaldson & MacNee (1998) hypothesise that inflammatory responses of the lung, triggered by inhaled particulates, are likely to cause a local production of procoagulant factors, and or effect how mediators from the lung interact with the liver, to increase the overall synthesis of procoagulant factors. Although a relatively untested hypothesis, epidemiological evidence has shown an increased level of blood viscosity to exist during air pollution episodes (Peters et al 1997, Brook & Rajagopalan 2009).

The manner in which PM₁₀ appears to cause adverse health effects, unrestricted by any specific exposure threshold (WHO 2006), would suggest that PM₁₀ is a highly toxic material, yet the individual components on their own are often not particularly toxic at ambient air levels. It is on this understanding that transitional metals and very small particles are of the utmost importance in mediating those health burdens associated with PM₁₀ (Donaldson & MacNee 1998). In particular, ultrafine particles (<0.1 μm) are viewed to be highly toxic towards the lungs, even when those particles are formed from materials that are non-toxic as larger but still respirable particles (Grassian et al 2007, Hamilton et al 2009, Karlsson et al 2009). This would suggest that ultrafine particles have a toxicity that is a result of their small size, and hence large surface area, rather than their chemical composition.

In humans, ultrafines are known to have the potential to deposit in high numbers towards the terminal airways. When rodents have been exposed to high levels of ultrafines, 'classical particle overload' has been reported to impair alveolar macrophage mediated clearance from the distal regions of the lung, culminating in fibrosis and cancer of the lung (Oberdorster 1995). Within this original hypothesis, signs of impaired movement have been reported when phagocytosed particles constitute 6% of a macrophages internal volume, with 60% volume levels preventing the movement and thus clearance mechanisms provided by macrophages (Renwick et al 2001). An expert panel concluded, whilst it is uncertain whether high lung burdens of ultrafines can lead to lung injury in humans via mechanisms similar to those of the rat, in the absence of mechanistic data to the contrary, it must be assumed that the rat model can identify potential carcinogenic hazards to humans (ILSI Risk Science Institute 2000).

The biological effects of several different types of particle contained within PM₁₀, including inert components, have been shown to be mediated by their transitional metal content (Castranova et al 1997, Gilmour et al 1996). Oxidative stress is considered to arise first from the transition metals themselves, such as iron and copper, which have a well-documented ability to generate hydroxyl free radicals via Fenton chemistry (Donaldson & McNee 2001). A response supplemented by an influx of inflammatory cells that result from the primary interaction between lung cells and general particle deposition. Whilst inflammation acts to seal off infected areas and attract additional immune cells, if left unregulated, the proteases and reactive oxygen species (ROS) released by surplus granulocytes will impair host cells, causing oxidative DNA damage. The potential magnitude of this mediation is best observed within Castranova et al's (1997) laboratory study of F344 rats exposed to 20mg/m³ doses of 2µm chemically inert quartz particles. Here, the pathogenic properties of quartz within the lung drastically rose after contamination with trace levels of iron; to cause a 537% increase in leukocyte recruitment and 71% increase in nitric oxide production from macrophages (Castranova et al 1997).

Ultimately the deposition of particles that deliver an oxidative stress signal to the lungs result in the activation of NF-κB proteins, which initiate a coagulation cascade involving the proinflammatory mediator thrombin, potentially causing a state of haemostasis (Donaldson & McNee 1998, Maki et al 2010). This increased production of inflammatory mediators, along with the increased permeability of antigens, is potentially most problematic for persons with underlying inflammation of the airways (i.e. tracheobronchial and asthmatic conditions). Figure 2.6 summarises the hypothetical interactions between PM₁₀ and target cells in the generation of respiratory conditions discussed within this section.

To conclude, a recent appraisal of particulate compositions across four English roadside locations respectively identified PM_{2.5-10} and PM_{2.5} fractions to be dominated by carbonaceous combustion particles (21.1%, 62.9%), secondary nitrates and sulphate (14.5%, 22.3%), and coarse dusts (60.2%, 8.5%) (Harrison et al 2004). As anticipated, the PM_{2.5} fraction contained a substantial carbonaceous component, which is formed from incomplete combustion and defines the most important components relating to toxicity. Yet interestingly, the composition of coarse dusts within the PM_{2.5-10} fraction was also observed to be rich in iron (65.8%), hypothetically originating from brake wear and corrosion in addition to engine emissions. This abundance of transition metals within the coarser particulate fraction, although typically limited to shorter residential times within the main airways, reinstates the need for one to address the wider impact of road transport on health, not limited to exhaust emissions or a particularly sized fraction.

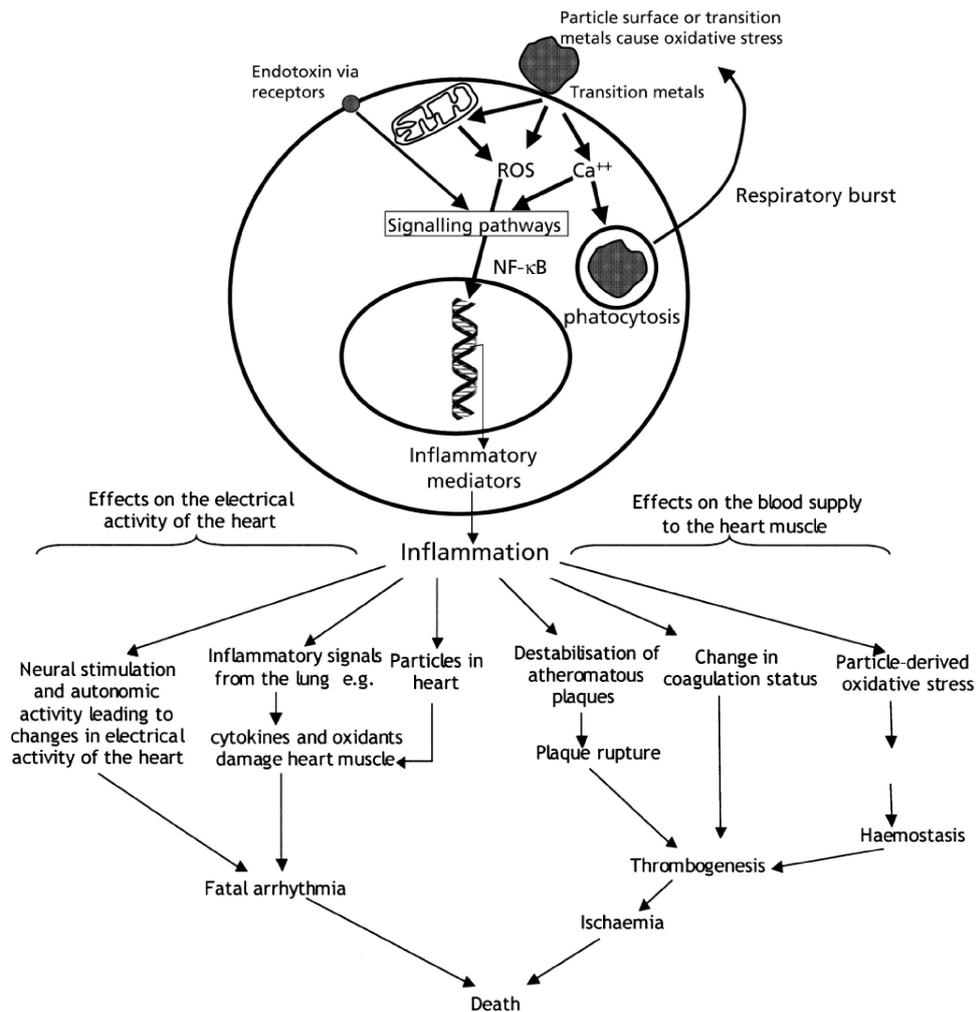


FIGURE 2.6: Hypothetical interactions between PM₁₀ and target cells in the generation of respiratory conditions (marked by inflammation), with relation to further issues of the human cardiovascular system (Modified from Donaldson & MacNee 2001)

2.2.2. PM₁₀ A MODULATOR OF PULMONARY HOST RESISTANCE

Particulate induced health effects are understood to predominantly involve the destruction of cellular DNA via oxidative stress, followed by a natural inflammatory response, which isolates then attacks both the foreign entity and surrounding area affected by its presence. Yet it has also been shown that exposure to pollutants can alter the host's defence mechanisms, increasing the likelihood for infections to occur following an exposure episode (Becker & Soukup 1999, Zelikoff et al 2003). This secondary mechanism may be of far greater importance, when considering that a 3-year Australian Cohort of 263 infants detected viruses in 69% of non-hospitalising acute respiratory illnesses (Kusel et al 2006). Meanwhile, a 2-year Finish Cohort of 293 children hospitalised with acute expiratory wheezing, detected causative viral agents in 88% of cases (Jartti et al 2004). The literature would therefore suggest that viral activity has an important role in initiating, prolonging or exacerbating respiratory conditions, particularly during childhood.

Animal toxicology studies have consistently identified the immunosuppressive influence of pollutants in host susceptibility to viral and bacterial infections. Mice challenged with the influenza virus, ensuing a 6 month exposure period to diesel engine emissions (2mg/m³), were shown to experience significantly higher levels of lung consolidation (61.5%) than their air-exposed counterparts (33.3%) (Hahon et al 1985). Rats infected with a strain of *S. pneumoniae*, then exposed to a 5-hour dose of concentrated ambient particulates (65-150µg/m³), have also displayed bacteria burdens 300% above air-exposed subjects (Zelikoff et al 2003). Furthermore, mice instilled with 40µg of black carbon 3-days after Respiratory syncytial virus (RSV) infection, demonstrated an exacerbation of RSV-induced airway hyper-responsiveness and excessive pulmonary inflammation responses (Lambert et al 2003).

Human in-vitro investigations have indicated alterations in proinflammatory cytokine production of cultured bronchial epithelial cells following diesel exhaust particulate concentrations of 0.08 - 0.33mg/mL (up to 39x control value), potentially upsetting the immune homeostasis of the lung (Steerenberg et al 1998). Becker & Soukup (1999) recorded a 50% decrease in the uptake of RSV in human alveolar macrophages (AM) in the presence of PM₁₀, with PM₁₀ exposure in the absence of infection significantly increasing the production of macrophage inflammatory proteins. This would imply that exposure to PM₁₀ alters AM-regulated inflammatory responses to viruses, enhancing the spread of infection. Furthermore, Jaspers et al (2005) identified solutions comprising of 25µg/cm² diesel exhausts to increase viral RNA levels in bronchial epithelial cells, 80% above samples that had only been infected with the influenza virus.

For the adenovirus group, Fujii et al (2002) has demonstrated ambient urban PM₁₀, collected from Ottawa, to directly induced lung inflammation via a response amplified by

latent viral infection. Here, human lung epithelial cell lines challenged with a 6-hour exposure of 100µg/ml PM₁₀, solely infected with the adenovirus, or a combination of the two, respectively recorded a 22%, 46% and 132% increase in the release of the proinflammatory mediator IL-8 over control samples (Fujii et al 2002). Similar results have been produced in human lung alveolar cells, infected with the adenovirus 24-hours prior to an 18-hour treatment of 100mg/ml ambient urban PM₁₀, collected in London. Here, cell lines only infected with the adenovirus or challenged by PM₁₀, or a combination of the two, respectively recorded a 170%, 200% and 580% increase in the release of IL-8 over control samples (Gilmour et al 2001). Thus reconfirming the individual detrimental importance of both factors, whose actions appear exacerbated when mutually presented. It is suggested that the presence of adenoviral primes the cell transcriptional machinery for oxidative stress signalling and therefore facilitates amplification of proinflammatory responses, leaving the individual susceptibility to exacerbation of the airways in response to particulate air pollution (Gilmour et al 2001).

Mushtaq et al (2011) have also demonstrated the colonisation of *Streptococcus pneumoniae*, a common cause of bacterial pneumonia, to be promoted by 4-hour exposures of ambient urban PM₁₀, collected in Leicester. Here, the adhesion of *s. pneumoniae* to lines of human airway and bronchial epithelial cells, as assessed by confocal microscopy fluorescence levels, respectively recorded 120% and 240% more viral infected cells to be present than at the control sample, when 30 µg/ml and 50µg/ml PM₁₀ doses are introduced.

Unsurprisingly, studies have also indicated that pollutants capable of oxidative stress, inclusive of NO₂ and O₃, can amplify the generation of proinflammatory mediators by infected cells. Spannhake et al (2002), have demonstrated that after a 3-hour exposure to NO₂ (2.0 ppm) or O₃ (0.2 ppm), human nasal and bronchial cells burdened by rhinovirus and an oxidant respectively produced 42-250% and 41-67% more IL-8, than those environmentally unburdened infected cells; note that lower and upper bounds relate to cell to viral concentrations of 1:3,000 and 1:300. Yet in using influenza as the viral contagion, studies have demonstrated inhalation exposure to NO₂ to provide no effect on (Lefkowitz et al 1986), increase (Ehrlich et al 1975) or decrease (Buckley & Loosle 1969) viral induced impairment of murine lungs. Whilst less is known concerning the possible interactions between NO₂ and viral infections, a plethora of toxicological studies have demonstrated the suppressive effect upon host responses to bacterial infection.

In particular, Bouley et al (1986) noted a preceding 4-day NO₂ (20ppm) exposure period as insignificantly altering the natural resistance of non-immunised mice challenged with *Klebsiella pneumoniae*. Meanwhile, in their immunised counterparts, the number of inoculated bacteria per mouse was reduced by 19.6%, to a level only 6.7% above that of the non-immunised mice (Bouley et al 1986). This would suggest that rather than reducing host

immunity beyond its baseline, NO₂ only acts to impede the operations of one's immune system, where one has been developed. Thus, if one was to extrapolate these findings to humans, children are candidates at particular risk, considering their reliance on a respiratory immune system still in its early stages of development.

In a series of studies, Jakab (1987) conclusively demonstrated NO₂ as a modulator of murine pulmonary antibacterial defences, establishing a threshold dose for adverse effects to occur across a range of bacterial strains. In unexposed animals 7% of *S. aureus* remained after 4-hours, antibacterial defences appeared suppressed at NO₂ levels of 4ppm and greatly reduced at 15ppm, with 11% and 48% of bacteria respectively lingering (Jakab 1987). For *P. pneumotropica* antibacterial defences were impaired at 10ppm, with bacteria residence levels shifting from 19% (control) to 26%, rising more gradually to 37% under a 30ppm exposure (Jakab 1987). Yet interestingly, exposure to 10ppm was found to enhance the intrapulmonary killing of *P. mirabilis*, as recorded by bacterial levels reducing from 24% (control) to 18%, with bactericidal activity only becoming impaired at 20ppm (Jakab 1987). Thus demonstrating how low levels of oxidants may act to induce beneficial responses against certain pathogenic strains.

From this wealth of toxicological research, air pollutants are expected to provide an immunosuppressive influence on human susceptibility to viral and bacterial infections. Of which particulates are decidedly linked to infections of a viral nature, with oxidising pollutants sharing closer ties with bacterial pathogens. As mentioned above and discussed in detail within the succeeding section of this chapter, viral infections are heavily associated with respiratory burdens, whereas bacterial infections are specialised to only a selection of cases. Thus, not only are particulates thought to provide a greater direct respiratory burden, they also appear most entwined with these secondary (pathogenic) mechanisms of detriment. Whilst epidemiological evidence in this field is lacking, one investigation of 2,604 Washington State infants hospitalised with RSV-Bronchiolitis provides some evidence for this immunosuppressive influence. Here, a 10µg/m³ increase in PM_{2.5} was associated with a 14% and 4% rise in bronchiolitis hospitalisations in RSV infected and non-infected infants respectively (Karr et al 2009).

In their medical overview of respiratory system disorders, Cumming & Semple (1980) discuss that although the human lower respiratory tract of healthy is virtually sterile, microorganisms may be cultured from the upper respiratory tract for transport into the deeper regions of the lung. Following this logic, one should therefore consider the possibility of socio-environmental stimulus' by enlarge acting to weaken those upper respiratory regions (where contact is greatest), priming these locations for pathogenic colonisation. Subsequent exposures are then either likely to facilitate the passage of these pathogens towards the lungs,

or have direct impact in the lower regions themselves caused by the hosts reduced immunosuppressive response. Upon viewing Figure 2.7, one may clearly observe that many viruses associated with mild URTI cases have a secondary distribution within the LRT, involving tracheobronchial symptoms. It is thus of interest for this research project to uncover the level of involvement immunosuppressive stimuli have in respiratory decline.

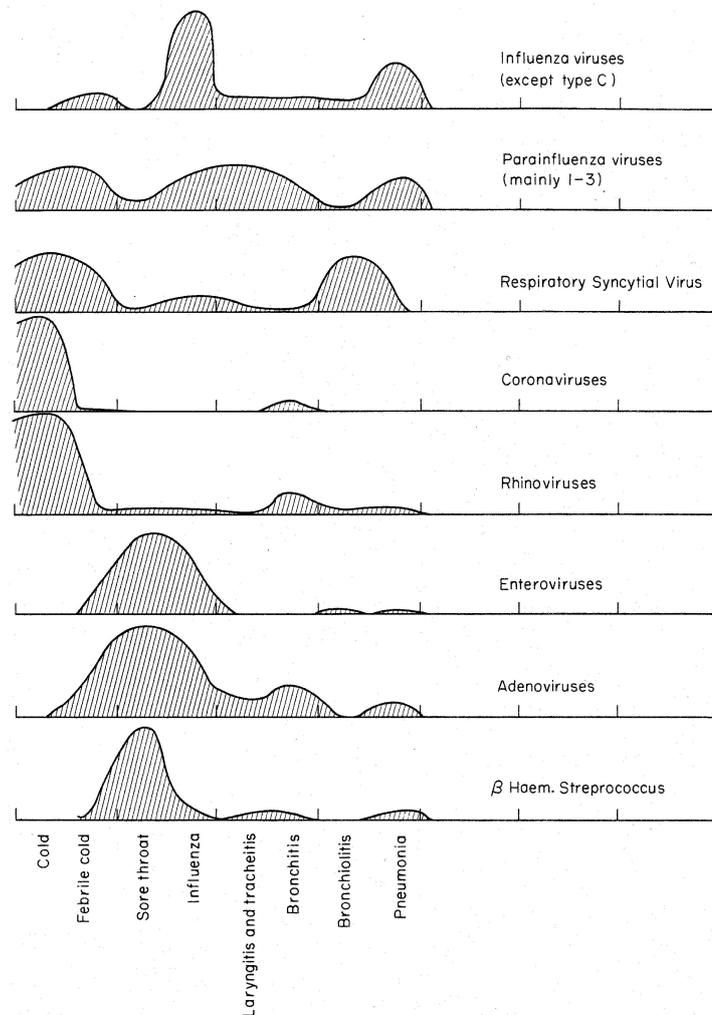


FIGURE 2.7: The influence of pathogenic agents on acute respiratory infections (qualitative estimate), occurring in the proximal to distal regions of the human respiratory tract (Cumming & Semple 1980)

2.2.3. PREVALENCE OF PEDIATRIC RESPIRATORY DISORDERS

Acute respiratory infections characterised by the inflammation of mucous membranes lining the upper and or lower respiratory passages, are a major international health concern, recorded as the third highest global cause of mortality at a rate of 4,259,000 per annum (7.2% total); with lower respiratory tract infections (LRT) responsible for 98.1% of these cases (WHO 2008). In addition, acute respiratory infections are deemed the leading cause of global disease burden, annually accounting for 94,511,000 disability-adjusted life years (6.2% total), with conditions of the LRT once again holding the majority stake (96.6%) of this figure (WHO 2008).

Such findings are of little surprise considering that the mammalian respiratory tract is the easiest portal of entry to outside pathogens, irritants and pollutants. Within a UK context, the average person likely to suffer from 5-7 episodes of acute respiratory infections per annum, with acute respiratory infections accounting for 30-35% of all new complaints presented to GP's, the majority of which appear viral related (Cumming & Semple 1980).

	Per 10,000 Persons
Upper Respiratory Tract Infection (URTI)	2,235
▪ Common cold & influenza	1,450
▪ 'Flu-like' illness	170
▪ Throat infections	600
▪ Glandular fever	15
▪ Ear infection	850
▪ Nasal infection	260
Acute Chest Infections	640
▪ Bronchitis	580
▪ Pneumonia	60
Chronic Chest Conditions	450
▪ Chronic bronchitis-emphysema	250
▪ Asthma	200
Other Condition (lung cancer, hay-fever, etc.)	530

TABLE 2.2: Third National Morbidity Survey annual patient consulting rates relating to conditions of the respiratory tract (Adapted from Fry & Sandler 1993)

Upon viewing a summary of respiratory consultations recorded within 'The Third National Morbidity Survey', representative of UK primary care consultations for >300,000 person-years at risk, one may understand the respiratory burden practitioners face in greater detail (Table 2.2). Out of this broad categorisation of respiratory complaints, one may observe that URTI's account for the majority (58.0%) of respiratory consultations, which typically involve symptoms associated with the common cold. Whilst it has been demonstrated that URTI's pose a limited lasting threat to health, they may instigate infections of the lower respiratory tract (LRT), thus demonstrating a systematic deconstruction of the respiratory system. Concerning the LRT's involvement in 28.2% of respiratory consults (Table 2.2), infections resulting in bronchitis are at fault 53.2% of the time. Interestingly, the more commonly explored condition in air pollutant investigations, asthma, only accounts for 18.3% of the LRT's burden. On another note of interest, respiratory disorders are the largest group of conditions causing absence from work or prolonged invalidity, accounting for 25% of all causes within the UK [Bronchitis 9%, Acute URTI 5%, Flu-like illness 5%, Asthma 3%, Other 3%] (Fry & Sandler 1993). Such information reinforces the importance of intermediating infections, not only of the lower, but of the upper tract as well.

2.2.3.1. CENTRAL RESPIRATORY CONDITIONS

Respiratory conditions of prominent importance would appear to focus around three specific classifications of infection, two of which involve the URTI, entwined with each other and infections of the LRT:

I. The common cold (acute coryza) & influenza

Whilst probably the largest group of common diseases in the world, URTI's are a confusing mass of uncertainty of nature, causes and management (Fry & Sandler 1993). The most common of which is the common cold (or acute coryza), defined as a self-limiting illness of short duration typified by catarrhal symptoms including nasal discharge, sneezing and sore throat, often characterised by viral participation. As such, their prevalence peaks in mid-winter from December-April, although symptoms are present throughout the year (Fry & Sandler 1993). Rhinoviruses are the primary causative agents of the common cold accounting for 30% of all cases, with other agents including coronaviruses, enteroviruses, parainfluenza viruses and RSV; however, fascinating 30% of all cases still transpire from unknown causes (Seaton et al 1989). While bacteria are not the primary causes of acute coryza, they may cause secondary infections by streptococcus pyogenes, haemophilus influenza and pneumococci (Fry & Sandler 1993).

To remove foreign material from the nasopharynx, damaged columnar epithelial cells containing viral antigenic material and invasive pathogens (and or particulate matter) are shed into the nasal discharge. The shedding of cells is usually completed within a few days, and although mucosal damage is minimal in the majority of cases, cellular recovery may take up to 2-weeks (Seaton et al 1989). This leaves the region primed for secondary infections, and or damage from environmental stimuli. Under certain conditions, an accumulation of shed cells also has the potential to pass into the lower regions of the respiratory system. As previously noted, adenovirus and coronaviruses are responsible for a variety of respiratory infections, and RSV is strongly associated with impairment of the tracheobronchial tree (Figure 2.7). The common cold and influenza share many clinical features, with differing only by its increased severity and abrupt onset (hours), compared to the gradual appearance of acute cold symptoms over a few days.

II. Infections of the throat & nasal passages

Pharyngitis, tonsillitis, sinusitis, and laryngitis encompass a collection of disorders whose prominent clinical features are sore throat with variable degrees of accompanying ill health, with their similarities, further separation into individual diagnostic labels is considered somewhat unhelpful (Fry & Sandler 1993, Seaton et al 1989). Pharyngotonsillitis is the most

common classification for acute throat infections, characterised by a localised swelling of the tonsils, which are often covered with exudate and have a pitted follicular look; complications are rare and generally associated with streptococcus bacterial infections, resulting in scarlet fever (Fry & Sandler 1993, Seaton et al 1989). Most cases naturally recover within a week, with recurrent attacks frequent in children and adolescents (caused by minor immunological changes between adolescence and adulthood), which cease after the age of 25 (Appendix A4). Viruses account for 70-85% of pharyngotonsillitis cases in children >3 years and for 90-95% of cases in adults, the bacteria streptococcus pyogenes is typically responsible for those other cases (Mazur 2010). The adenovirus is commonly found to be the primary viral agent here, accounting for up to 73% of all children's cases (Donati et al 1998). Likewise, cases of laryngitis are habitually accredited to viral infection, parainfluenza acts as the causative agent 75-100% of the time, with bacterial laryngitis known to follow or occur alongside the viral illness (Donati et al 1998, Nadel 2009).

In contrast to the above conditions, sinusitis is a bacterial infection that occurs commonly in the population, complicating about 1 in 200 upper respiratory tract infections, with bacterial isolates for *S. pyogenes*, *H. influenza* and *S. pneumoniae* respectively found in 94%, 78% and 69% of all cases (Seaton et al 1989, Jousimies-Somer et al 1989). It is supposed that a disruption of normal defensive mechanisms associated with the common cold and throat infections, result in the accumulation of a mucous exudate, which then becomes secondarily infected by bacterial pathogens, causing infections of the sinus and potentially chest. In addition, sinus and throat infections often directly spread to the ears, and may indirectly cause problems of the inner-ear through the congestion of nasal passages. Treatment is empirical, a logical choice being penicillin; where a severe infection is unresponsive to 10-day course, sinus contents may be aspirated by direct puncture (Seaton et al 1989).

III. Acute chest infections: Tracheobronchitis & pneumonia

This group labelled as the 'acute chest infections' refer to a series of common inflammatory conditions affecting part or the whole of the tracheobronchial tree. These conditions, which overlap and are ill-defined clinically, frequently follow infection with any of the common cold viruses; thus illustrating the gulf between the specific postulation of conditions in an artificial context and practical realities, as a process hindering the assessment and management of these patients (Fry & Sandler 1993, Seaton et al 1989). Tracheobronchitis may affect any age group but is reported more commonly in children and the elderly. It initiates as a dry cough followed by an excessive production of sputum, and as such is usually described by adults as 'a cold that has gone to my chest' (Seaton et al 1989, p276). Underlying causes may be identified

through the sputum, with a green-yellowish tinge indicating a bacterial infection, 'rusty' colouration pneumonia, with a thin mucoid representing viral bronchitis (Fry & Sandler 1993). Wheezing in adults is not usually a feature unless the patient has a chronic respiratory condition, but it is more common in children, potentially resulting in diagnostic confusion with chronic conditions. When a bacterial infection of the windpipe (Tracheitis) is present, the patient may complain of chest tightness, sometimes described as burning that may be heightened by inspiration or coughing (Seaton et al 1989).

Within Dr John Fry's Greater London general practice, the term 'pneumonitis' is a portmanteau attempt to combine pneumonia and bronchitis, suggesting something less severe than pneumonia and not quite bronchitis. Here, a causative diagnosis is only observed in 33% of pneumonitis episodes, with some bacteria (pneumococci, staphylococci) and viruses (influenza, parainfluenza, RSV) recognised to instigate such acute chest infections; however this is of limited use in a primary care context, where combinations of causal agents are usually indefinable and superimposed on individual and social factors (Fry & Sandler 1993).

2.2.3.2. THE CATARRHAL CHILD SYNDROME (CCS)

Children in their first 10 years of life are most susceptible to a wide spectrum of clinical respiratory conditions (Appendix A4). In general practices, illness involving the upper and lower respiratory tracts account for almost 50% of all attendances for children, with over 25% of cases referred to paediatric hospital services involving the specific combination, sinobronchitis (Nichols 1959). Yet in spite of their frequency, their causes remain uncertain and unproven. This has resulted in the rather generalised classification of these acute respiratory conditions, specific to this early developmental phase in life, known as 'Catarrhal Child Syndrome' (CCS).

"It is assumed that they are infections caused by viruses but the specific pathogens are rarely isolated; bacteria may be responsible for a minority of ear, throat and chest infections but then again they are isolated in a minority of cases. Whilst allergy has been put forward as a possibility there is no reliable evidence for this. Undoubtedly some children and some families appear to suffer more frequently and more seriously than others and therefore it is likely that there may be underlying social familial and genetic factors"

(Fry & Sandler 1993, p48)

To understand this syndrome, catarrh is fluid flowing from a mucous membrane, and thus the catarrhal child is characterised by an excessive response of the mucous membranes to disturbing factors; with each candidate typically responding by producing a single aspect of the

syndrome, reoccurring at a specific site (Nichols 1959). CCS may be divided into four main clinical groups of children's respiratory infection. The largest, with a clinical consulting rate of 58 cases per 100 children (Fry & Sandler 1993), involves the inflammation of the mucous membranes of the airways causing a frequent reoccurrence of coughs and colds. The second group involves the condition Otis media, which relates to earache, deafness and purulent discharge of the ears, occurring at a rate of 13 consults per 100 children (Fry & Sandler 1993). Communication between the auditory and respiratory systems is achieved by the eustachian tube connecting the middle ear to pharynx, yet this link may also assist the spread of infection to the middle ear; thus explaining why Otis media is included as a respiratory complaint.

The third group relates to infections of the throat, a set of complaints tending to occur almost exclusively only in older children, as recorded by a rate of 7 consults per 100 children (Fry & Sandler 1993). Unlike the three previous subsets, the final group characterises infections of the lower respiratory tract (or acute chest infections), which exist at the lowest rate of 6 consults per 100 children (Fry & Sandler 1993). This group includes episodes of acute wheezing, debatably labelled as asthma or acute bronchitis, which generally ceases in susceptible children >10 years of age (Fry & Sandler 1993, Seaton et al 1989). This group also accounts for generalised (acute bronchitis) and localised (pneumonia) signs of chest distress, involving an accumulation of extravascular fluid in the lungs (moist rale), which is potentially life-threatening.

“Considering the well-nigh inevitability of children suffering from variants of one syndrome and the fact that they appear to ‘outgrow it’ and gradually cease to suffer from it after the age of 7 to 8 it is likely to be a natural response of an immature immunological system to various external pathogens, pollutants and irritants that given time a natural immunity develops”

(Fry & Sandler 1993, p49)

Still this collective vulnerability has puzzled GP's as to why this syndrome has remained so difficult to define and is resistant to specific therapy, with the common link appearing to be the mothers of these catarrhal children whom often display anxiety disproportionate to the severity of the condition (Nichols 1959). Nevertheless, many children are thought to experience these features, the majority of which are rarely presented to the doctor.

In this project, it is considered that through analysing the spatial outbreak of URT and LRT infections, one may be able to shed light on the perceived involvement of various socio-environmental stimuli on CCS, which until now have only been theorised in an extensive body of anecdotal research (Fry 1966, Fry 1993). Through using hospital data (tip of the clinical iceberg), cases of hypersensitive awareness presented at the practitioner's level should be

filtered, hopefully allowing for a clearer account of CCS. By focusing on these extreme cases, not only may solutions be offered to mitigate the introduction of long-term conditions in a few select subjects, such knowledge also has the potential to assist with reducing accounts of mild CCS across the wider populace.

2.3. EPIDEMIOLOGICAL EVIDENCE

2.3.1. AIR POLLUTANTS & CHILDREN'S RESPIRATORY HEALTH

Childhood is a critical period for the development and maturation of the delicate spongy organs of the cardiorespiratory system, which are particularly susceptible to the absorption of external environmental agents experienced within the urban arena. Children also spend more time outdoors compared to adults, conducting activities that increase ventilation rates (Cooper et al 2010, Steele et al 2010); factors that respectively extend the contact period and time-proportional intake of ambient pollutants experienced by children. Furthermore, a child's lung surface area is also considerably larger in relation to their body mass, with children potentially breathing up to 50% more air per kilogram of body weight (Schwartz 2004). In-fact, a laboratory analysis of PM_{2.5} deposition rates normalised by lung surface, recorded levels in children aged 7-14 years to be 35% above of those of adults during resting breathing; attributed to a superior rate of ventilation in relation to lung size (Bennett & Zeman 1998). As such, air pollutant exposure during childhood is of particular concern, with prolonged contact periods stunting the development of vital cardiorespiratory organs, thought to induce ailments that prevail into adulthood (Grigg 1999, Stick 2000, Mathieu-Nolf 2002, Schwartz 2004)

A plethora of studies has found associations between selected air pollutants and adverse health effects in children. With respect to particulates, these adverse health effects have tended to focus upon physician validated signs of acute respiratory illness, or deficits in lung functionality. The most prominent investigation of these outcomes was realised by the 'Pollution Effects on Asthmatic Children in Europe' (PEACE) study, conducted in the winter of 1993/94 across 14 European research centres (Roemer et al 1998). In total, 2,010 children participated over 28 panels, with each centre providing an urban and suburban (with no major traffic or industrial sources) locale, to compare differences in pollutant effects caused by level and composition. Enrolled children aged 6-12 years with chronic respiratory symptoms in the last 12 months and/or doctor-diagnosed asthma ever in life, personally monitored their health status through diary records and measurements of peak expiratory flow (PEF) conducted on a bi-daily basis.

A standardised panel-study protocol was applied to each panel separately. Initially, individual PEF readings were transformed into daily population morning and evening mean values. Symptoms reported within the diaries were recoded at 0 (no symptom) and 1 (slight, moderate or severe symptom) to obtain measurements of daily prevalence. Linear regression models accounting for minimum temperature, weekday, time trends and autocorrelation were created to measure the association between daily pollutant exposures on population weighted health outcomes. A single pollutant model was preferred because of the high correlation ($R > 0.6$) between PM_{10} , BS, SO_2 and NO_2 . In the second stage of this investigation, singular effect estimates of air pollution on PEF or the daily prevalence of respiratory symptoms were calculated from panel-specific effect estimates using fixed effect meta-analysis techniques. In the presence of heterogeneity, random effect estimation was calculated using the non-iterative method with unequal weights; the simplest method of addressing both within-study and between-study variance.

The overall findings of the multicentre PEACE study revealed no clear associations to exist between levels of pollutant and PEF, or the prevalence of respiratory symptoms. Unexpectedly, the majority of combined effect estimates for air pollutants were associated with a beneficial PEF response, albeit at a non-significant level. A significant negative association was only found for PM_{10} (1 day lag) and evening PEF levels, inducing a change of -0.6 L/min (CI: -1.1, -0.1) per $100\mu g/m^3$ (Roemer et al 1998). Furthermore, significant odds ratios (OR) were not identified in relation to symptoms of either the upper or lower respiratory tracts, with a $100\mu g/m^3$ increase in PM_{10} found only to influence phlegm production (1.02; CI: 0.94-1.11) (Roemer et al 1998). The lack of effect in Roemer et al's (1998) multicentre study cannot be explained by insufficient statistical power, or by low levels of exposure, as burdens have been documented across lower pollutant concentrations. Interestingly, successive reports of the wider PEACE project identified a concurrent influenza epidemic to be of some influence, unavoidable by the panel's short and common timeframes, which had beneficially reduced the potential for heterogeneity (Roemer et al 2000).

In response to such uncertainties, Ward & Ayres (2004) conducted a meta-analysis of temporally based cohort (or panel) studies, measuring individual levels of lung function amongst children, 0-15 years of age. Using the 'Web of Science Interface', major bibliographic databases were searched from 1966 to June 2002 for appropriate materials, complemented by an inspection of paper references, consulting books and reports known to the authors. The search identified 13 suitable panel studies of children that used daily measures of PM_{10} as a marker of PEF. Four were summer studies; one was set across an entire year, whilst another eight were conducted in winter conditions, the largest study of which was part of the PEACE project documented by Roemer et al (1998). The majority of studies recruited panels of

children either diagnosed with asthma or with reported existing respiratory symptoms ('symptomatic subjects'), with all except two studies reporting daily average PM_{10} concentrations in excess of $50\mu\text{g}/\text{m}^3$. Older children were included in four studies; three extending the range to 13 years, and one to 15 years. A two stage analytical approach was adopted by two studies, whereby individuals were modelled then pooled to form an average PEF value, the remainder employing a population daily average PEF outcome in their analyses. Potential autocorrelation effects were included within all studies, with all models adjusting for temperature. A descriptive summary of these studies used for this enquiry is presented within Appendix A5.

Taken as individual study components, a wide spread of results are observed in relation to PM_{10} , with all except one recording lung performance in an adverse direction (Figure 2.8). Pooled results under a fixed effects model identified a -0.012 L/min change in PEF per $1\mu\text{g}/\text{m}^3$ increase in PM_{10} (CI: $-0.017, -0.008$), increasing to -0.033 L/min (CI: $-0.047, -0.019$) under a random effects scheme which placed less weight on the PEACE study (Ward & Ayres 2004). However, in using an asymmetrical funnel plot to explore the possibility of publication bias, Ward & Ayres (2004) observed strong associations between increasing effect size and decreasing size of the study estimate's standard error. Whilst smaller studies are less likely to be published presenting negative findings, it is also plausible for the precision of an effect estimate to be determined by variability in exposure. Still, the degree of heterogeneity evident between panel studies, indicated by Q-combinability tests (derivative of chi-square), questions the transferability of estimated effect sizes between locations or populations, limiting the direct use of such summary measures.

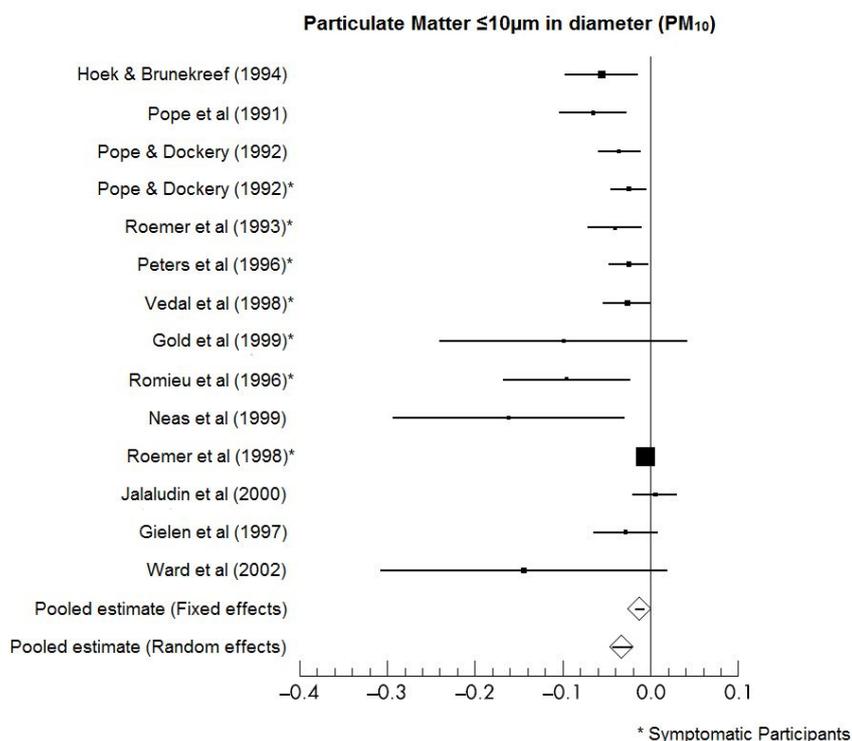


FIGURE 2.8: Meta-analysis of mean and 95% confidence interval estimates of the impact of PM_{10} on PEF, expressed as litre/minute change per $1\mu\text{g}/\text{m}^3$ rise (Adapted from Ward & Ayres 2004)

A vast quantity of pediatric research, examining the health effects of particulates, has also placed focus on understanding its associations with asthmatic symptoms on a day-to-day scale. Whilst PM_{10} has often been associated with increases in symptomatic frequency amongst children, Weinmayr et al (2010) also recognised inconsistencies within this tangent of research, yet to be addressed by comprehensive quantitative evaluation.

To quell these discrepancies Weinmayr et al (2010) conducted a systematic search of the literature contained within the MEDLINE database from 1990 through to 2008, for children aged 0-18 years. Indoor and laboratory studies were excluded, while panel studies of asthmatic or symptomatic children were included under restricting criteria of one publication per dataset. In total, 29 studies comprising of 43 populations, were identified to present suitable measurements for a meta-analysis of asthmatic symptoms. Of these populations, 24 represented urban settings, 32 were from Europe and 11 from elsewhere, mainly the United States. As before, publication bias was assessed in a graphical manner through a funnel plot of population estimates plotted against their standard error (Light & Pillemer 1984). In addition, statistical measures were applied to confirm evidence of funnel plot asymmetry, whereby the standardised effect size is regressed against the inverse of the standard error (Egger et al 1997).

	N°	Fixed Model OR [95% CI]	Random Model OR [95% CI]	I ² [P-value] Heterogeneity	Egger Bias P-value
All Studies	43	1.023 [1.013, 1.034]	1.028 [1.006, 1.051]	59% [<0.01]	0.779
Without PEACE Studies	17	1.035 [1.023, 1.047]	1.055 [1.032, 1.078]	56% [<0.01]	0
Trim-and-Fill Estimate	24	1.028 [1.016, 1.039]	1.035 [1.012, 1.058]	61% [<0.01]	

TABLE 2.3: Associations between PM₁₀ exposure and clinician diagnosed asthmatic symptoms, summarised by change in odds ratio (OR) per 10µg/m³ increase in pollutant under fixed and random effects models (Adapted from Weinmayr et al 2010)

When all studies were considered, Weinmayr et al (2010) found no evidence of publication bias; however, bias was noted to arise on the exclusion of the generally inconclusive PEACE studies. Whilst research contained within the wider PEACE project provides the only multicentre set of investigations conducted with a unified protocol (limiting the presence of bias), concern exists that an influenza epidemic confounded the entire study series. To address such issues a trim-and-fill procedure was applied (Duval & Tweedie 2000), which caused the random-effects estimate excluding PEACE studies to decrease from 5.5% to 3.5% asthmatic episodes per 10µg/m³ of PM₁₀ (Table 2.3). Whilst this meta-analysis found clear evidence of effects of PM₁₀ on the occurrence of asthmatic episodes, despite underestimations from the PEACE project, some concern was raised involving the proportion of variation in estimate due to heterogeneity caused by the lack of a standardised study design.

In contrast to the literature summarised in the meta-analyses above, the strength of association between particulates and respiratory-based hospital admissions in children appears ill defined. Recently, Anderson et al (2004) published the findings of a meta-analysis conducted by the St. George's Hospital Medical School Group, documenting the weight of PM₁₀ on hospitalisation cases across individual age groups. Time-series and panel studies were identified through a search of three bibliographic databases (Medline/Embase/Web of Science) up until 2003, with search strings tested against known literature. The St. Georges research group concluded that >3 studies were required to justify a meta-analysis, with studies included only on meeting the following criteria: The use of single-pollutant models (1-day lag) conducted in a European setting, only supplemented by North American studies where necessary. The investigation concluded that an insufficient number of respiratory hospital admission studies had been conducted on children age 0-14 years and adult's age 15-64 years, for a meaningful meta-analysis (Table 2.6). In contrast, a sufficient numbers of estimates (6 originate from the APHEA 2 project) were available only for the ≥65 year age group, whom recorded a 7% rise in respiratory hospitalisations per 10µg/m³ increase in PM₁₀ (Table 2.4).

Age Group	Study Design	Number of estimates	Relative Risk [95% CI]
0-14 years	Standard	3 (Insufficient)	1.010 [0.998, 1.021]
15-64 years	Standard	3 (Insufficient)	1.008 [1.001, 1.015]
≥65 years	Standard	8 (Satisfactory)	1.007 [1.002, 1.013]
	Trim-and-fill	10 (Satisfactory)	1.006 [1.000, 1.011]

TABLE 2.4: Original and revised summary relative risk estimates per 10µg/m³ increase in PM₁₀ and respiratory hospital admissions (Adapted from Anderson et al 2004)

Barnett et al (2005) offer some solace towards seeking a suitable association between outdoor air pollution and hospital admissions in children, through their multicentre case-crossover study of Australia and New Zealand. Here, daily hospital and central monitoring pollution data were collected for the period 1998-2001, across the 5 largest cities in Australia (Brisbane, Canberra, Melbourne, Perth, and Sydney) and 2 largest cities in New Zealand (Auckland, Christchurch), respectively accounting for 53% and 44% of each country's population. Unlike previous hospital inquiries, which traditionally quantify a single pollutant outcome for children of all ages (0-15 years), Barnett et al (2005) decided to examine three individual age bands so as to differentiate the lung functions and immune systems of infant and teenage children.

Following traditional cohort designs, a case-crossover analysis was conducted to look at the effects of factors thought to increase the risk of children's respiratory health in the short term. Here, exposure information is obtained for the individual whom acts as their own control, comparing the presence of risk factors immediately prior to onset with reference periods of good health (across a 28-day-window prior to onset). Covariates also applied to control for the day-of- week and a plethora of meteorological variables. To obtain an average dose-response outcome, city estimates were combined using a random effects meta-analysis. Out of the individual pollutants evaluated, statistically significant increases were found for PM_{2.5}, PM₁₀, NO₂ and SO₂, but not for CO or O₃. Typical of urban environments, Barnett et al (2005) observed strong correlations between certain pollutants, which is unsurprising considering their likelihood of originating from a common emission source (primarily motor vehicles). Given these correlations, matched pollutant models were run to identify whether pollutant impacts differed or were related to each other.

Barnett et al's (2005) multicentre study demonstrated statistically significant associations between outdoor air pollution and children's respiratory hospitalisations to exist across Australia and New Zealand, at levels generally below those found in European and North America cities. Within this multisite investigation, a 2.3% and 2.5% increase in respiratory hospitalisations per 10µg/m³ increment in PM₁₀ were respectively recorded for children aged 1-4 years and 5-14 years (Barnett et al 2005). Interestingly, significant association with PM₁₀ disappeared for children aged 5-14 after matching with NO₂, indicating

that the two pollutants could not be separated. In contrast, the health associations per interquartile shift in NO_2 remained after matching with PM_{10} , identifying this gaseous pollutant to have a far wider source of origins (Barnett et al 2005). Interestingly, respiratory admissions in children aged 1-4 years rose to 7.3% when the effects of PM_{10} and $\text{PM}_{2.5}$ were matched, showing that the different particulate combustion and wear components provide separate health effects; reconfirming how important it is to explore the entire spectrum of airborne particulates (Barnett et al 2005).

However perhaps it is far too simplistic and potentially misleading for investigations to summarise the effects of pollutants across the entire spectrum of respiratory conditions, which have resulted in cases of hospitalisation. It is far more realistic that episodic exposures are to be followed by equally acute responses, and that through including chronic conditions, the actual involvement of environmental agents are reported at a somewhat diminished value. As such, stricter legislation is less likely to materialise, despite the fact that respiratory infections account for over half of all respiratory hospitalisations. Whilst of a shorter duration, and thus reduced cost, one should still consider that reoccurring episodes during developmental stages might initiate the later onset of conditions more chronic in nature. In light of this, I conducted a narrower systematic search of the literature from 1955 through to 2013, which focused on the short-term effects of outdoor PM_{10} on hospitalisations attributed to infections of the respiratory tract. Using the 'Web of Science Interface', major bibliographic databases were searched using the broad strings of "respiratory infection", "PM10 OR PM (10)" and "children". A total of 1,030 results were returned, and the abstracts of potential articles of interest were checked for relevance.

Authors	Study Setting	Study Period	Outcome	Age Group [Pediatric Cases]	24-hr PM ₁₀ µg/m ³	Study Design	Odds Ratio (95% CI)
Amarillo & Carreras (2012)	Cordoba, Argentina	Jan 2005 - Dec 2008	LRTI	0-15y [46,902]	65.6 [26.7 - 122.0]	[B]	1.019 [1.015, 1.024]
Le et al (2012)	Saigon, Vietnam	Jan 2003 - Dec 2005	LRTI	0-5y [15,717]	73.2 [19.3 - 195.7]	[A]	1.003 [0.991, 1.015]
Wong et al (2010)	Hong Kong, China	Jan 1996 - Dec 2002	LRTI	0-14y [32,473]	51.6 [13.5 - 188.5]	[B]	1.007 [1.001, 1.014]
Moura et al (2009)	Rio de Janero, Brazil	Apr 2002 - Mar 2003	LRTI	0-12y [6,801]	34.7 [11.2 - 79.0]	[A]	1.013 [0.970, 1.057]
Hernandez-Cadena et al (2007)	Ciudad Juarez, Mexico	Jul 1997 - Dec 2001	LRTI	0-16y [11,448]	38.7 [6.8 - >150.0]	[A]	1.021 [0.963, 1.086]
Farhat et al (2005)	Sao Paulo, Brazil	Aug 1996 - Aug 1997	LRTI	0-13y [5,555]	62.6 [25.5 - 186.3]	[A]	1.017 [0.985, 1.050]
Romero-Placeres et al (2004)	Havana City, Mexico	Oct 1996 - Mar 1998	LRTI	0-14y [44,029]	59.2 [7.6 - 201.9]	[B]	1.008 [1.000, 1.018]
Hajat et al (1999)*	Greater London, UK	Jan 1992 - Dec 1994	LRTI	0-14y [N/A]	28.5 [15.8 - 46.5]	[B]	1.012 [1.000, 1.025]
Lin et al (2005)	Toronto, Canada	Jan 1998 – Dec 2001	URTI/LRTI	0-14y [6,782]	20.4 [4.0 - 73.0]	[C]	1.064 [0.992, 1.152]
Amarillo & Carreras (2012)	Cordoba, Argentina	Jan 2005 - Dec 2008	URTI	0-15y[34,667]	65.6 [26.7 - 122.0]	[B]	1.008 [1.002, 1.014]
Wong et al (2010)	Hong Kong, China	Jan 1996 - Dec 2002	URTI	0-14y [153,675]	51.6 [13.5 - 188.5]	[B]	1.003 [1.000, 1.007]
Hernandez-Cadena et al (2007)	Ciudad Juarez, Mexico	Jul 1997 - Dec 2001	URTI	0-16y [28,461]	38.7 [6.8 - >150.0]	[A]	1.021 [0.981, 1.063]
Romero-Placeres et al (2004)	Havana City, Mexico	Oct 1996 - Mar 1998	URTI	0-14y [99,441]	59.2 [7.6 - 201.9]	[B]	0.998 [0.990, 1.007]
Hajat et al (2002)*	Greater London, UK	Jan 1992 - Dec 1994	URTI	0-14y [N/A]	28.5 [15.8 - 46.5]	[B]	1.007 [0.999, 1.014]
Hernandez-Cadena et al (2000)	Ciudad Juarez, Mexico	Jul 1997 - Dec 1998	URTI	0-15y [12,721]	34.5 [6.8 - 167.5]	[A]	1.023 [1.004, 1.042]

Footnotes:

*Study of General Practitioners surgeries (primary health care) rather than hospital datasets (secondary care)
[A] Time-Series: GAM (Poisson Link); **[B]** Time-Series: GAM (Quasi-Poisson Link); **[C]** Bidirectional Case-Crossover Design

TABLE 2.5: Literature review of the associations between PM₁₀ exposure and children's hospitalisations relating to infections of the upper or lower respiratory tract (URTI, LRTI), as summarised by change in odds ratio per 10µg/m³ increase in pollutant

In total 11 studies of interest were revealed (Table 2.5), most of which had been produced in the years following the meta-analyses of Anderson et al (2004) and Barnett et al (2005). PM₁₀ hospitalisation coefficient estimates for the LRT and URT were respectively provided in 7 and 5 of the studies, all of which were based in a South American or Asian setting. Equal weighting of these study estimates provided within Table 2.5, respectively inform of a 1.3% and 1.1% rise in children's LRTI and URTI admissions per 10µg/m³ increment of PM₁₀. Whilst some respiratory infection studies had been conducted within a European setting, measurements only existed at a primary care level. For a western setting, one Canadian study was noted to collectively examine the particulate impact of on upper and lower respiratory infections, recording children's hospitalisations as rising by 6.4% per 10µg/m³ increment of PM₁₀ (Lin et al 2005)

From this body of research, one may conclude that at present there is a limited amount of available research quantifying how particulate pollutants influence cases of children's respiratory hospitalisations; despite evidence firmly supporting the likelihood of such unfavourable outcomes across this most susceptible age group.

2.3.2. ROAD-TRANSPORT & CHILDRENS RESPIRATORY HEALTH

A concern towards the involvement of air pollutants on public health has been widely publicised by a series of epidemiology studies since the 1970's, but few studies have successfully distinguished source specific impacts, with pollutants in the urban environment often originating from a plethora of sources. In particular, traffic-related sources constitute the predominant source of outdoor air pollution within the urban arena, emitting a concoction of air quality objective pollutants and carcinogenic hydrocarbons within close proximity to residential districts.

The most direct approach towards distinguishing each community's unique air pollution exposure has involved the utilisation of surrogate measures, such as residential proximity to major road links (Appendix A6). An initial body of research founded upon such practices appeared in the early 1990's, predominantly focusing on self/clinician-reported minor respiratory ailments by children and adolescents housed along streets with high levels of road-transport activity (Appendix A6). Selected on the principles that the young spend a greater proportion of time at or within close proximity to their place of residence, and that they are unlikely to have experienced any other significant lifetime exposure events. Likewise, some investigations have been conducted on elderly populaces, although prior exposure events make their causative conclusions less definitive (Garshick et al 2003, Lipfert et al 2008).

The earliest of these minor ailment studies chose to focus purely on the level of vehicles passing directly through the child's residential street, in relation to self-reported wheeze, with symptoms rising by 94-147% when comparing European streets with limited and constant levels of truck traffic (Duhme et al 1996, Keil et al 1996). Later studies have since explored the spatial extent of minor respiratory ailments associated with road-transport pollutants through the examination of artificially created response buffers, traditionally set across a range of subjective distances from specified major road links (Appendix A7). Likewise, a noteworthy selection of studies exists examining the reduced performance of lung responses with respect to source proximity. The most prominent followed lung development within Californian children across an 8-year period, observing respective declines in Forced Vital Capacity (FVC) of -63ml and -19ml for residents housed $\leq 500\text{m}$ and 1000-1500m away from freeways (Gauderman et al 2007). As before, investigations have also targeted specific components of the vehicle fleet recognised as primary polluters, with one such study identifying truck-traffic pollutants to diminish the FVC of Dutch Children residing 300m and 1000m from motorways by -3.6% and -2.0% respectively (Brunekreef et al 1997).

Yet, surprisingly few studies have formally quantified distance based exposure thresholds, with respects to more severe respiratory outcomes amongst children. In-fact only six investigations were identified to use hospitalisation cases across the period 1999-2011, all limited to the evaluation of asthma (Wilkinson et al 1999, Lin et al 2002, Maantay 2007, Newcomb & Li 2008, Chang et al 2009, Li et al 2011). In their investigation, Lin et al (2002) used a study population of 417 asthma hospitalisation cases within Erie County, USA during 1990-1993, verified spatially independent from non-respiratory hospital markers of poor-health. Through an initial control distance of 600m and a near distance of 200m, asthma hospitalisation odds ratios were reported at 1.24 [CI: 0.87, 1.77] (Lin et al 2002). Yet perhaps the findings of most interest were observed when a distance of 200m was held to compare roads of low and high traffic density, resulting in an odds ratio of 1.93 [CI: 1.13, 3.29] (Lin et al 2002). This highlights the shortcomings of certain studies in only investigating proximity as a marker of spatial influence, revealing a need to use a variable that characterises the multitude of geographic traits associated with transport (i.e. proximity, flow, vehicle classification)

Furthermore, Li et al (2011) recently reported health-exposure relations to remain across buffers zones previously considered to be distant in nature (Figure 2.9), showing the fundamental limitation of arbitrary buffers in presenting critical thresholds for health managers to focus on.

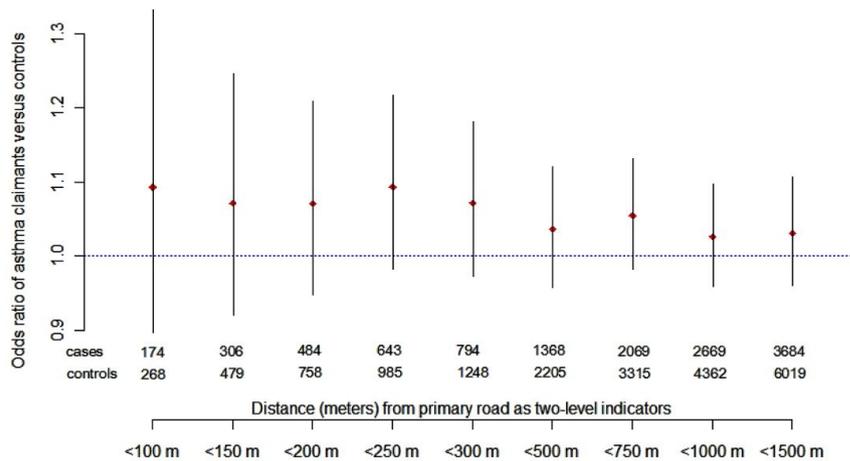


FIGURE 2.9: Estimated odds ratios reporting children’s asthma hospital visitations with respect to distance from a primary road, through a conditional logistic regression case-control analyses in Detroit, USA: 2004-2006 (Li et al 2011)

An alternative means to evaluate distance thresholds, involves the use of boundary statistics, which are widely used within the fields of genetics and ecology to detect and quantify overlap between naturally occurring geographic frontiers (Barbujani et al 1989, Hall 2008). However, until now, their potential for distinguishing critical transport thresholds has yet to be evaluated, despite offering an unbiased selection of response buffers and road inclusion, if suitable measures of transport are to be used. While surrogate measures, have been related to hefty health effects, it should still be considered that such techniques are highly prone to exposure misclassification. These issues may be mitigated through Geographic Information System (GIS) techniques, which offer a more sophisticated assessment of road traffic pollutants across vast populations, to provide the additional explanatory power required for a more traditional regression based analysis.

In one such study, Gehring et al (2002) utilised Land Use Regression (LUR) models to evaluate the effects of traffic related air pollution and respiratory health during the first 2yrs of life, within a sample of 1,757 infants located in the German city of Munich during the period 1995-1998. LUR is based on the principle that pollutant concentrations at a given location depend on the environmental characteristics of the surrounding area that influence or reflect emission intensity and dispersion efficiency. To achieve this concentrations measured at road-side monitoring locations were regressed against relevant environmental variables, with the resulting equation used to predict concentrations at unmeasured locales based on those predictor variables. Within their investigation, Gehring et al (2002) applied the LUR model constructed by the SAVIAH project using measurements of altitude, land cover and traffic volume (Briggs et al 2000), to provide a model fit ($R^2=0.67$) comparable with sophisticated dispersion models. After adjusting for social factors, an odds ratios of 1.32 [1.10-1.59] and 1.03

[0.90-1.18] were respectively reported per $1.5\mu\text{g}/\text{m}^3$ incremental in $\text{PM}_{2.5}$, for prevailing symptoms of cough without infection and respiratory infections in the first year of life; with such effects appeared to attenuate during the infant's second year of life (Gehring et al 2002).

Brauer et al (2002) conducted a comparable inquiry into the onset of pediatric asthma across a Dutch cohort of 4,135 infants aged 2 years, after designing a national $\text{PM}_{2.5}$ transport focused LUR model determined by land cover, traffic volume and region ($R^2=0.73$). After adjusting for social factors, odds ratios of 1.14 [0.98-1.34] and 1.20 [1.01-1.42] were respectively reported for self-reported infections of the upper respiratory tract and wheeze, per $3.2\mu\text{g}/\text{m}^3$ increase in $\text{PM}_{2.5}$ (Brauer et al 2002). In addition, odds ratios of 1.12 [0.84-1.50] and 1.04 [0.85-1.26] were respectively reported for cases of doctor-diagnosed asthma and bronchitis, per $3.2\mu\text{g}/\text{m}^3$ increase in $\text{PM}_{2.5}$ (Brauer et al 2002).

In a unique GIS study of Continental Europe, Kunzli et al (2009) acquired detailed industrial and road-transport 1x1km emission release inventories from Austrian, French and Swiss national agencies to assess the individual public-health impacts of outdoor and traffic-related air pollution components. In this context, PM_{10} was deemed the most appropriate indicator of fossil-fuel combustion sources, as a pollutant-by-pollutant estimate would grossly overestimate health impacts. Residential levels were linked to 1x1km PM_{10} emission grids for 1996, and the subsequent population exposure distributions were provided with appropriate meta-analytical exposure-response functions, calculated as the variance weighted average across the results of preceding epidemiological inquiries. Levels of outdoor air pollutants recorded by PM_{10} were annually associated with 48,000, 450,000 and 45,000 bronchial episodes in children aged 0-15 years, respectively residing in Austria, France and Switzerland; with contributions from road-transport held responsible in 43.75%, 55.55% and 53.33% of such cases (Kunzli et al 2000). Whilst the use of external coefficients fails to provide a conclusive assessment, the widespread coverage of emission inventory datasets and their uniform measures of environmental assignment, are considered to offer great potential in future explorations of environmental contributions specific to the transport sector.

2.4. ENVIRONMENTAL INJUSTICES OF AIR POLLUTION

A substantial quantity of published literature examining the short-term effects of air pollution on health events tends to derive from an abundance of time series, case-crossover and panel studies, which traditionally utilise average citywide ambient pollutant concentrations in order to estimate population exposure solely on a temporal scale. While ideal for assessing the

impacts of geographically wide pollution episodes and long-term general population ambient exposure, such studies overlook the fact that air pollutant concentrations often exhibit strong spatial patterns across the various micro environments located within the urban arena. Failure to consider these spatial variations at the sub-city level can lead to exposure misclassification and subsequent bias. Such considerations are of particular importance, especially when assessing the levels of personal exposure to environmental impacts. Therefore, it should be of little surprise that important questions even for the well-established short-term health effects of air pollutants prevail; the most important of which relate to the precise characterisation of the exposure-response relations generally and within specific population groups.

Conventionally vulnerable social groups comprising of the young and elderly, have been targeted for aiding in the definition of air quality standard exposure thresholds. However recent interest into the field of 'Environmental Justice' (EJ) by researchers and policymakers has highlighted an increased social patterning of society's vulnerable groups, with people of lower socioeconomic status often found living within areas experiencing elevated environmental burdens. The current US EPA definition of EJ originates from their 1995 'Environmental Justice Strategy', and remains the basis on which the US government may provide legal assistance:

"Environmental Justice is the fair treatment and meaningful involvement of all people regardless of race, colour, national origin, or income with respect to the development, implementation, and enforcement of environmental laws, regulations, and policies. Fair treatment means that no group of people should bear a disproportionate share of the negative environmental consequences resulting from industrial, governmental and commercial operations or policies. Meaningful involvement means that: [1] people have an opportunity to participate in decisions about activities that may affect their environment and/or health; [2] the public's contribution can influence the regulatory agency's decision; [3] their concerns will be considered in the decision making process; and [4] the decision makers seek out and facilitate the involvement of those potentially affected."

(US EPA 1995/2013 - Online)

Two important dimensions contained within this definition relate to the 'fair treatment' and 'meaningful involvement', which were considered in detail by the Scottish Executive (now Scottish Government) to form the first EU integration of EJ concerns in social policy. Within this debate, a distinct separation is made between the distributional and procedural aspects of this phenomenon:

“[1] The ‘distributive justice’ concern that no social group, especially if already deprived in other socio-economic respects, should suffer a disproportionate burden of negative environmental impacts;

[2] The ‘procedural justice’ concern that all communities should have access to the information and mechanisms to allow them to participate fully in decisions affecting their environment”

Scottish Executive 2004, p16

Whilst both definitions highlight distributional and procedural aspects, the European outlook places emphasis on how social conditions produce injustice, with American approaches focusing on the racial dimension of discrimination and exclusion during decision-making. These general dissimilarities in underlying philosophy relate to the geographically differences in public policy, determined by historic issues of class conflict (the need to correct overlying social processes) or civil rights movements (the need to uphold an individual’s natural rights). In paraphrasing Cutter (1995), Environmental Justice (EJ) may be defined as a principle guaranteeing the equal access to a clean environment and equal protection from possible environmental harm, irrespective of one’s race, income and or class (socio-ethnic status). Of significant importance is the fact that such equality measures embody mechanisms for assigning culpability, therefore shifting the burden of proof of contamination to the polluters not resident, a term coined as ‘The Polluter-Pays Principle’ (PPP). “Thus EJ research seeks to determine whether marginal and/or minority groups bear a disproportionate burden of environmental problems, and whether planning policy and practice affecting the environment are equitable and fair” (Mitchell et al 2003, p909).

The environmental justice debate in the USA has been explored at length, with origins in the civil rights movement regarding issues of landfills and polluting industries predominantly located within black communities or indigenous people’s reservations. However, such investigations have typically been plagued by difficulties of definition, assessment methodology and interpretation. In a review of the historic EJ literature, Bowen (2002) concludes that the empirical foundations are so underdeveloped, that little can be said with scientific authority concerning geographical patterns of inequality; with the direct use of existing literature potentially resulting in the creation of poorly conceptualised and harmful managerial decisions. Still, even where evidence has clearly pointed towards discrimination, court cases have often been unsuccessful in proving intentional conduct on the part of those held accountable (Hershenberg 2001). Nonetheless, EJ is now an important part of environmental and public health policy assessment in the USA, mandated by a presidential executive order in addition to a growing body of research (Clinton 1994). Conversely, the issue

is less developed in the UK, with significant research and policy interest only appearing at the turn of the millennium in the wake of investigations conducted by environmental pressure group Friends of the Earth; who assessed the proximity of deprived communities to industrial facilities regulated by the Environment Agency (McLaren et al 1999).

The existence of a social gradient in health is a well-established concept, recently reassessed via a meta-analysis of 155 relevant papers, of which 70% suggest that health is less good in societies where vast income differences exist (Wilkinson et al 2006). However despite the numerous socioeconomic factors already identified to affect health, some of these inequalities remain unexplained, leading to the hypothesis that environmental nuisances may also contribute to social health inequalities (Deguen & Zmirou-Navier 2010). “Increasingly, it has come to the attention of researchers and policy makers that the distribution of exposure to air pollution is not equitable, but this inequity has until recently received little formal epidemiologic attention” (Naess et al 2007, p686). Traditionally, epidemiological based studies of air pollution have treated socioeconomic positioning as a confounding influence, removable by any available indicator in an attempt to achieve burden estimates independent of the social environment. Conversely, few studies have looked carefully at how these factors interact with one another. Subsequently in recent times, researchers have taken particular interest in examining the associated double burden of deprivation and exposure to air pollution in relation to respiratory health (Crouse et al 2009, Kingham et al 2007, Naess et al 2007, Namdeo & Stringer 2008, Wheeler & Ben-Shlomo 2005).

In a paper aspiring to advancing the theory and methods surrounding the concept of health, wealth and air pollution, O’Neill et al (2003), outline three possible mechanisms to explain how exposure to air pollution may contribute to greater health effects among individuals of lower Socioeconomic Status (SES):

- 1)** Lower SES may increase susceptibility to air pollution-related health risks directly through increased levels of psychosocial stress, limited access to health care, or increased likelihood of living in lower quality housing;
- 2)** Some health conditions (e.g. asthma, diabetes, and cardiovascular diseases), behaviours (e.g. smoking) and genetic traits that increase susceptibility to effects of air pollution are distributed differentially by SES
- 3)** Populations with low SES may have more frequent or more intense exposures to air pollution than those with high SES due to environmental inequalities.

At present, it is suggested that disparities in exposure by SES are conceivably the least studied of the three outlined mechanisms through which air pollutants may contribute towards a social gradient in health outcomes (Crouse et al 2009, O'Neill et al 2003). Nevertheless, such issues are rapidly attaining recognition in the policy domain; with the Environment Agency's position statement on environmental inequalities declaring that those living in the most deprived parts of England experience the worst air quality (Environment Agency 2004). Such statements stipulate the need for further research investigating the current state of affairs of environmental inequity, thus opening the possibility for improving policy guidelines for tackling the associated double burden of deprivation and environmental hazards.

2.4.1. CONTEMPORARY STUDY DESIGNS & PROTOCOLS

Although the environmental justice movement in relation to air pollution has received increased attention in recent years, it should be noted that a majority of the early literature has tended to focus around the inequalities associated with industrial pollutants, particularly within a North American context (Morello-Frosch et al 2001, Hipp et al 2010). More recently, there has been increased attention paid to traffic pollution's role on defining urban air quality, likely brought about by attempts to incorporate issues of environmental equity into the sustainable transport debate (Feitelson 2002).

A significant proportion of the emerging literature assessing air pollution, social deprivation and health outcomes now follows the widely accepted environmental epidemiology time-series techniques (HEI 2003), focusing on the use of generalized additive models (GAMs). A Norwegian study, conducted by Naess et al 2007 utilised GAMs to combine monthly dispersion model pollutant estimates (1992-95), mortality figures (1992-98) and 1992 cohort SES data for elderly residents aged 50-74 within the municipality of Oslo. The derived statistical models were subsequently stratified across relevant sex and age adjusted bands, for analysis with both singular and a combination of SES covariates, with and without the associated air pollution concentrations. Findings from this study revealed deprivation at both the individual and neighbourhood level to be associated with air pollution, accounting for some of the excess mortality associated with air pollution in these neighbourhoods.

Lin et al 2004 employed non-parametric GAMs with natural cubic splines, to evaluate the associations between gaseous air pollutants and asthma hospitalisations (1987-98), for children aged 6-12 years, stratified by sex and SES, in the city of Vancouver, Canada. This particular investigation identified nitrogen dioxide (males only) and sulphur dioxide (females only) to be significantly and positively associated with asthma in the low SES group, but not in the high SES group. Conversely, an identically designed investigation across Strasbourg, France

(2000-05) revealed positive but not significant associations between asthma attacks and modelled PM₁₀, NO₂ and SO₂ concentrations, to be influentially independent of small-area variations in deprivation (Laurent et al 2008). Here, discrepancies between comparably designed studies only act to emphasise the continued requirement to consider such questions across additional study settings, through a more expansive range of modelling concepts.

Recently, Deguen & Zmirou-Navier (2010) conducted a review of papers assessing the relationship between social inequality and health risks linked to ambient air quality, within a European setting. In total 129 papers were identified to explore inequalities in exposure according to some measure of socio-economic status, of which only 23 investigated the extent to which such factors modified relationships between air pollution and some health event, often comprising of mortality rates. In general, the studies imply that poorer people experienced elevated exposures to air pollution, bar a few exceptions. Nevertheless overall patterns, irrespective of exposure, identified subjects of low SES to experience consistently greater health effects in relation to air pollution. To conclude, the authors identify two plausible directions for future research to address shortcomings in both localised and international research assessing the double burden of SES and air quality on health status. It was suggested that the most prolific observable improvements in future studies, could be attained through the application of improved personal exposure classification, a concept consistently recognised within a plethora of other contemporary research articles (Mitchell 2005, Wheeler & Ben-Shlomo 2005, Crouse et al 2009, Barcelo et al 2009). Secondly, in a forthcoming research ventures the researchers propose to address issues relating to a shortage of childhood-based studies detailing issues of SES, environmental pollutants and health. Such issues are of considerable importance, considering that poverty and deprivation at an early age may potentially cause adverse health consequences throughout a person's entire life (Deguen & Zmirou-Navier 2010).

In recent times, focus has started to shift to the inability of temporal and conventional multivariate regression techniques to efficiently measure localised variations of environmental equity. This is of particular concern, when considering that EJ is an explicitly spatial problem, concerning geographic elements rarely distributed in a uniform manner (Gilbert & Chakraborty 2011). In a distinguished EJ analysis on the health risks from automobiles, Chakraborty (2009) presents a selection of global regression models that can account for spatial dependence (if detected in the residuals of conventional multivariate models), through the addition of a spatial lag or error component. The spatial lag model assumes that autocorrelation is only in the dependent variable, resulting in an assessment focusing on the existence and strength of spatial interaction. In contrast, spatial error models consider residual formation to involve a

previously unmeasured geographical explanatory parameter, included to prevent modelling bias induced by spatial autocorrelation. Compared to the conventional regression models that indicated R^2 values of 0.35-0.40, spatial error model R^2 values ranged from 0.59-0.68, suggesting a considerable improvement in overall fit. In terms of environmental justice implications, Chakraborty (2009) observed persistent patterns of racial inequity involving the distribution of estimated health risks from vehicular emissions, across Tampa Bay, Florida. In contrast, a more complex relationship materialised for markers of poverty, with most models reporting no significant relation to cancer risk, or negative associations with respiratory risk that ceased to be significant under spatial schemes. Such observations emphasise the need for future investigations to consider the effect of spatial autocorrelation in environmental equity studies, in avoiding fictitious conclusions.

McLeod et al (2000) applied another class of global models incorporating spatial elements, known as multilevel models, in the first study of its kind to investigate the relationship between social class and air pollution concentrations in the UK, with specific focus on regional variation. Here, social class index (SCI) scores, population density and broad ethnic compositions were derived from the 1991 UK census, and combined with PM_{10} , SO_2 , and NO_2 levels interpolations of the 1997 1x1km UK National Atmospheric Emissions Inventory (NAEI) dataset, for 401 districts nested within 10 administrative regions. Initially a model was constructed from a fixed intercept representative of mean pollutant levels, and two random intercepts representative of district and regional level variations.

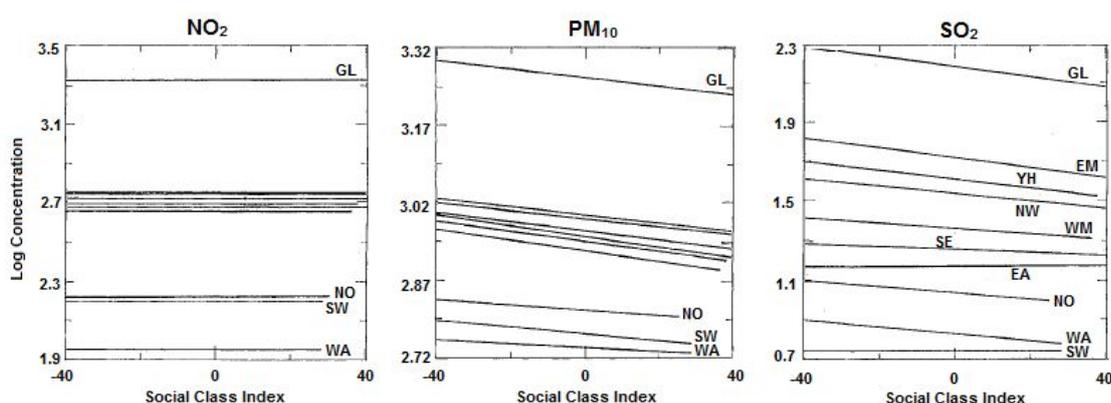


FIGURE 2.10: Predicted relationship across the UK between localised pollutant and social class index [-40 Deprived; +40 Affluent] by region (Adapted from McLeod et al 2000)

For all three pollutants, a greater degree of variability was observed at the regional level, with a strong urban bias between PM_{10} and NO_2 through their mutual source association, road-transport. A measurement of social status was then added, represented by a fixed parameter and a random slope parameter to describe regional variations. Negative associations between

social class index and pollutant concentrations were unanimously, with those most affluent experiencing lower levels of pollutants, although the magnitude of this relationship differed between each region. Interestingly, the higher the regional concentration of PM₁₀ the greater the social divide, whereas for NO₂ an equitable distribution of regional emissions was observed (Figure 2.10).

A third set of models also incorporating generalised shifts in ethnicity and population density, found positive relationships between pollutants and ethnic minorities. However, through individually exploring ethnic diversity, persons of higher social status now appeared more likely to be exposed to higher pollutant concentrations. McLeod et al (2000) concluded that whilst wealthier inhabitants consider a range of property characteristics prior to purchase, only a limited quantity of stock displays the required environmental and cultural amenities, with the latter option appearing most preferential. Thus, sweeping measures to improve air quality across urban locations, might actually decrease current levels of equity and produce injustice within certain regions.

More recently, a multilevel model approach was used to better assess the national cumulative health risk of public exposure from chemicals unique to industrial processes, across 65,166 census tracts (99.6% total) housed within 3,121 US counties (Young et al 2012). Here socioeconomic deprivation respectively increased a community's estimated cancer and respiratory exposure risk level by 6-20% and 12-27%, after adjustment for regional population, regional economic activity and local population density (Young et al 2012). Whilst the aforementioned spatial modelling approaches favour the removal of potentially confounding spatial elements from global relationships, others have actively looked to embrace these problematic localised variations, developing non-stationary relationships through a somewhat underused EJ technique known as Geographically Weighted Regression (GWR). Under this system individual ordinary least square models are conducted for each observation, with the influence of surrounding observations in each model weighted by proximity (adhering to Tobler's (1970) first law of geography), to provide geographically unique parameter estimates. Most significantly, GWR allows for a geographic disaggregation of community attitudes and characteristics to provide a greater sense of individuality, which is of importance considering that various sub-divisions of socio-ethnic class are often grouped into broad census categories. Through locating pockets of burdened communities, one may still identify global trends but through a means not limited to producing sweeping and potentially misleading conclusions.

Mennis & Jordan (2005), demonstrate the first application of GWR within an environmental equity analysis, in their assessment of the spatial distribution of air toxic release facilities across New Jersey, USA. Here, the density of toxic release inventory (TRI)

facilities within each census tract (theoretical exposure) described a level of hazard attributed to airborne toxic releases. The findings revealed a positively global and local relationship between ethnicity and air toxin release facilities, whereas the positive relationships of hazardous facilities with socioeconomic status appear to exist only in selective pockets; thus illustrating the importance of GWR in environmental equity assessments. To date, Gilbert & Chakraborty (2011) present the only EJ GWR study to assess the influence of social and environmental elements on areal health, indirectly defined as the risk of cancer from TRI exposures. Thus, it would be of interest for future GWR to develop upon such studies, applying measurements of actual health events and through exploring a wider range of cardiorespiratory conditions influenced by short-term exposures.

2.4.2. CONTEMPORARY TRANSPORT BASED ENVIRONMENTAL JUSTICE STUDIES

One of the earliest transport based environmental justice studies was conducted by Bae 1997, through the assessment of equity impacts brought about by Los Angeles' Air Quality Management Plan (AQMP). This remains one of the few studies to consider the net welfare impacts of air quality regulations, highlighting the direct health benefits to poorer members of society. In this methodology both beneficial (e.g. health improvements, housing costs) and detrimental (e.g. unemployment, transport taxation) impacts were weighed, in monetary terms, starting from the imposition of several uniform federal clean air acts from 1970 (amended in 1990) until their expected attainment date in 2010. Measures of statistical dispersion over 21 cities (population >100,000) in the form of Gini coefficients, respectively averaged pre and post AQMP values of 0.364 and 0.347, equating to a 4.7% improvement in the distribution of income after environmental intervention (Bae 1997). The generated models thus provided an alluring conclusion for policy makers, suggesting that a full implementation of the air quality standards outlined within AQMP could produce progressive benefits throughout the Los Angeles region, dismissing established preconceptions.

Recent UK based studies, investigating the relationship between policy driven air pollution changes on health and social deprivation in Leeds, have come in the form of two complementary studies conducted by Mitchell (2005) and Namdeo & Stringer (2008). In their study, Namdeo & Stringer (2008), examine how the relationship between air pollution, social deprivation and health would hypothetically alter through distance-based road user charging (RUC) scenarios. Under the base scenario, deprived (75th percentile) and affluent (25th percentile) communities on the Cumulative Deprivation Index were respectively exposed to NO₂ levels of 20.52 and 19.21µg/m³, indicating an unjust distribution of urban air quality. For

these two groups, a RUC set at 2 pence/km produced reductions of 3.4-3.6%, however it was not until a severe 10 pence/km scheme was implemented that a just situation materialised, with respective reductions of 10.9% and 14.3% in affluent and deprived locales (Namdeo & Stringer 2008). However, despite a strong relationship existing between social deprivation and general health status (R^2 0.47), only a weak study relationship was observed between air quality and health status, contradictory to the overall opinion of prior epidemiology studies.

Mitchell (2005) also reports social inequities to exist in relation to NO_2 distribution across Leeds, while assessing a plethora of alternative transport strategies. Nevertheless, in both cases the researchers agree that such findings cannot be used to state categorically that deprived communities bear a greater air quality dependent health burden, as other factors determining exposure are at present ignored. These include, the negligence of future land use and infrastructure in policy development, and the possibility of certain target groups (e.g. children) being more sensitive to pollutants than the general population. Yet, opportunities exist to improve exposure assessments, to address a wider range of transport measures affecting urban air quality, and to assess the generality of the findings reported here through extension to other cities (Mitchell 2005).

Through an appraisal of the London Congestion Charge Scheme (LCCS) Tonne et al 2008, directly calculated whether a fair distribution of air pollution and mortality benefits associated with the experimental transport scheme was achievable. Modelled pre (2003) and post (2007) LCCS concentrations assumed meteorology and vehicle fleet compositions to remain constant, allowing for the isolation of traffic flow and speed influences. The LCCS was identified to be responsible for a decrease in daily traffic levels of cars by 26% and heavy goods vehicles by 7% within charging zone wards, with no systematic change in traffic occurring across local roadways surrounding the zone (Tonne et al 2008). Across London, the greatest reductions in modelled pollutant concentrations occurred within the most deprived populace. For instance since the implementation of the LCCS, those most deprived communities were found to experience $0.24\mu\text{g}/\text{m}^3$ reductions in residential NO_2 levels, causing approximately 60 Years-of-life-gained per 100,000 persons over a 10-year period (YLG_{10}); meanwhile those least deprived only experienced a $0.02\mu\text{g}/\text{m}^3$ reduction in NO_2 equating to 5 YLG_{10} (Tonne et al 2008).

In contrast, Cesaroni et al's (2012) evaluation of two low-emission zones established in Rome across the period of 2001-2005, revealed well-off residents as experiencing the greatest level of health gains from zoning implementation. Here, residential reductions in NO_2 concentrations were observed to provide 687 YLG_{10} for communities of high socioeconomic position, compared to benefits of only 163 YLG_{10} experienced by residents of the most

deprived quintile (Cesaroni et al 2012). Whilst it should be the attention of policy workers to minimise pre-existing societal gradients, the Rome LEZ fails potentially introduces new issues, when considering that access to a car reflects social standing (see Carstairs Index). Transport surveys have consistently demonstrated those poorest and most socially disadvantaged within society to experience transport inequality. Within the 2012 UK National Travel Survey average levels of car ownership rests at around 75%, yet only 48% of the lowest income quintile households own private vehicles (DfT 2013). Furthermore, per person low-income households make 46% fewer trips and travel only 38% of the distance conducted by affluent households each year (DfT 2013). Pairing this information with the outputs from Cesaroni et al (2012), would suggest that Rome's LEZ has not only widened the gap in social health, but also increased the ability of the rich to shift their environmental contributions onto those most vulnerable members of society. In future it would be wise to place focus on locating and defining specific communities of interest (in terms of pollutant creation and exposures), in order to improve the ethical efficiency of future traffic management schemes, prior to implementation.

Taking a different approach, Mitchell & Dorling (2003) present the results of the first national study of air quality in Britain to consider the implications of its distribution over ten thousand local communities in terms of potential environmental injustice. Here, quintile plots of appropriate demographic data revealed community SES to be strongly related to NO_x emissions and ambient NO₂ concentrations (Figure 2.11). Interestingly, the study also signalled that communities with access to the fewest cars tended to suffer from the highest levels of air pollution, whereas those in which car ownership is greatest enjoy the cleanest air. Mitchell & Dorling (2002) note that UK air quality is predicted to improve further over the next decade or so, however the spatial distribution of pollution will remain much as it is now and hence inequity patterns are also likely to remain largely unchanged. If such a statement were to be true, it is of the utmost interest to develop these preliminary investigations, to advance our knowledge and avoid the environmentally unjust future we currently face. I believe this may be partially achieved through advanced spatial modelling techniques, utilising improved estimates of personal-transport emission contributions in conjunction with measures of personal exposure and social positioning. Through this approach, it becomes possible to locate and characterise patterns of mobility, which favour the development of localised solutions tailored to the needs of individual communities.

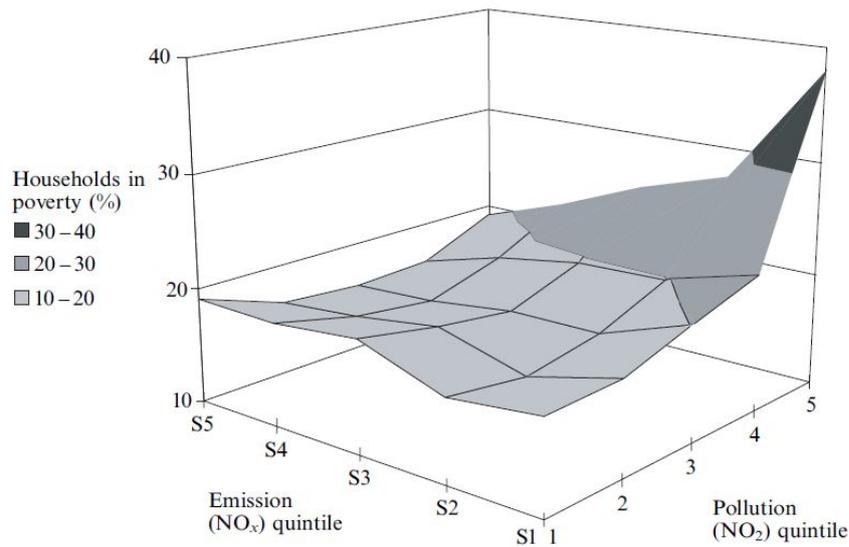


FIGURE 2.11: Poverty rate by NO_x emission and ambient air quality for 10 444 British wards in 1999 (Mitchell & Dorling 2003)

In terms of financial constraint, issues of mobility and the encouragement of 'green' transport are typically achieved through subsidies and concessionary fare schemes, whose cost and overall efficiency could be increased from a targeted approach. Here, Social exclusion becomes a useful term when discussing transport policy, with the success of schemes determined not only by affordability, but also through engaging marginalised groups to provide a local transport system that serves their needs (Hodgson & Turner 2003). Still, issues of poverty and social exclusion in transport policy are often entwined, with the availability, unreliability, high cost and time consuming nature of public transport (especially for local travel), reinforcing other dimensions of social exclusion to create 'no go' and 'no exit' communities (Kenyon et al 2002). Yet, the concept of developing neighbourhood transport systems challenges the traditional design philosophy, which seeks to maintain network integrity across the largest possible geographic scale. Nevertheless, Hodgson & Turner's (2003) practical implementation of neighbourhood policies for pockets of extreme poverty across Stockport, provide a template from which communities of socially excluded groups can be involved in the construction and management of future local transport systems. However, priority should initially focus on finding tools to locate and characterise community's, to understand the relation between mobility and local socio-environmental-health gradients; ultimately providing knowledge from which to better target and craft local policy.

In summary, to date there have been few investigations of the health effects of reduced air pollution resulting from policy interventions, with a significant proportion of current investigations consistently focusing on industrial operations. Furthermore, few intervention studies exist which focus on real world traffic management programmes, and

where conducted hypothetical rather than ground-truthed evidence exists quantifying the health benefits associated with reductions in traffic pollutants (Tonne et al 2008, Cesaroni et al 2012). Perhaps the use of more sensitive health measurements, consisting of localised hospital databases rather than modelled mortality ratios, will allow for a clearer assessment of current and impending air quality action plans.

2.5. CONCLUSIONS

Upon conducting this literature review, it has become apparent that there is a requirement for additional research within the emerging fields of environmental epidemiology, concerning the exposure inequities of particulate air pollution during childhood. To date, inquiries in this field have widely reported the existence of a 'double burden', in which mechanisms involving deprivation and exposure to air pollutants entwine to impede respiratory health. Yet this concept exists largely in a theoretical manner (Crouse et al 2009, Kingham et al 2007), with ground-truthed outcomes tending to involve crude indicators of health, recording levels of general well-being (Namdeo & Stringer 2008), modelled risk (Chakraborty 2009, Gilbert & Chakraborty 2011), or mortality (Naess et al 2007, Tonne et al 2008). Cases where sensitive measures of respiratory health are applied almost exclusively focused on asthma hospitalisations, and have reported contradictory results on the potential existence of a double burden (Lin et al 2004, Laurent et al 2008). These inadequacies stem from a much wider problem, concerning the general lack of epidemiological evidence reporting the relations between particulates and sensitive yet clinician diagnosed respiratory outcomes amongst children.

In terms of specific respiratory outcomes, perhaps focus should be shifted away from asthma in favour of a broader analysis of acute respiratory infections, which represent 74.6% of the overall respiratory burden; of which 77.7% occurs in the URT (Fry & Sandler 1993). Interestingly, it has been established that children in their first 10-years of life are most susceptible to suffer from a wide spectrum of frequent acute respiratory infections, caused by the heightened response of a developing immunological system. Yet in spite of their frequency, the causes of 'Childhood Cataract Syndrome' (CCS) remain uncertain and unproven, only partially explained by viral activity with underlying social factors likely at play (Fry 1993). In this project, it is considered that through analysing the spatial outbreak of URT and LRT infections, one may be able to shed light on the perceived involvement of various socio-

environmental stimuli on CCS, which until now have only been theorised in an extensive body of anecdotal research.

Whilst pollutants may directly aggravate the human respiratory tract, a wealth of laboratory based research has also linked exposure to an immunosuppressive response, leaving the host open to further damage from bacterial and viral infection (Gilmour et al 2001, Mushtaq et al 2011). Following this logic, one should therefore consider the possibility of socio-environmental stimuluses' largely acting to weaken the URT where contact is greatest, priming these locations for pathogenic colonisation. Subsequent exposures are then either likely to facilitate the passage of these pathogens towards the lungs, or have direct impact in the lower regions themselves caused by the hosts reduced immunosuppressive response. Prolonged or reoccurring exposure of the LRT during childhood is likely to stunt the development of vital cardiorespiratory organs, potentially inducing ailments that prevail into adulthood (Grigg 1999, Stick 2000). It is thus of interest for this research project, to also examine the role of socio-environmental stimuli in the decline of respiratory health along this infectious pathway within a real-world setting. This is in contrast to the majority of existing literature, which looks directly at the severest of acute LRT outcomes (asthma/bronchitis) and its links with chronic illness, without considering its initial onset.

Whilst a concern towards the involvement of air pollutants on public health has been widely publicised, few studies have successfully defined source specific impacts within the urban environment. Here, road-transport is of particular concern, emitting a concoction of air quality objective pollutants and carcinogenic hydrocarbons within close proximity to residential districts. Surrogate measures, in the form of residential proximity to major road links, have been previously used to explore outcomes most often at specific locations of extreme exposure; yet those same interactions across naturally occurring geographic frontiers of the entire cityscape remain unquantified. A void this project intends to fill through a boundary analysis approach, which is widely used within the fields of genetics and ecology to evaluate the geographic frequency and magnitude of intertwined processes on specified outcomes (Barbujani et al 1989, Hall 2008). A procedure, which appears to provide promising applications within the field of EJ, whose conclusions in the past have been plagued by inappropriate assessment practises.

In terms of a regression based approach, the use of temporal and conventional multivariate regression techniques have previously failed to evaluate localised variations of environmental equity in an effective manner. This is of particular concern, when considering that EJ is an explicitly spatial problem, regarding geographic elements rarely distributed in a uniform manner. As the application of spatial models is still in its infancy, Gilbert &

Chakraborty (2011) present the only GWR EJ study to assess the local influence of social and environmental elements on areal health, indirectly defined through risk. To date, Chakraborty (2009) present the sole EJ based spatial analysis of the respiratory burden specific to automobiles, once more restrained through its use of modelled risk rather than real-world health outcomes. It is therefore the intention of this project to extend the use of global and local spatial regression techniques into the largely theoretical EJ debate, through combining geographically detailed social, environmental and pediatric admission databases. In order to derive source specific outputs the project will follow in the steps of Kunzli et al (2009), whose direct use of emission inventories based on monitored vehicle flows, are considered to offer great potential in future explorations of environmental contributions specific to the transport sector. Primarily due to the vast geographic coverage of such environmental monitors, and the ease to which modelled outcomes may be transferred.

PROJECT DESIGN & DATA COLLECTION

OVERVIEW

This chapter opens with a description of the study area of Leicester UA, in terms of its geographic location and demographic characteristics, so that the reader may understand the relevance of such environmental inquiries. Following on from here the chapter describes the methodological approach adapted for this research based on findings presented within the preceding literature review, providing an overview of the project's design and data collection methods.

3.1. STUDY POPULATION AND SETTING

Leicester is a city of some 280,000 inhabitants spread across an area of 73.32km², located within the East Midlands of England. It is regarded as the British prototype for an ethnically harmonious multicultural city (Bonney & Le-Goff 2007, Vidal-Hall 2003). Population demographics from the 2001 UK Census reveal a relatively young population to inhabit Leicester, with 22.29% of residents under the age of 16 years (ONS 2003). Furthermore, 47.09% of children aged 0-15 years are from ethnic minority groups, of which 63.71% are identified to be of Indian ethnicity (ONS 2003). The city's other clearly defined ethnic minority groups are representative of contemporary UK migration trends, including children of Afro-Caribbean (5.69%), White Non-British (3.17%), and Other South Asian (12.49%) ethnicities (ONS 2003). In general Leicester is considered a relatively poor city, ranked as the 31st poorest out of 354 Local Authorities in England under the 2007 Indices of Multiple Deprivation (ONS 2008a). Such ethnically integrated yet deprived cities provide perhaps one of the greatest challenges for modern urban planners.

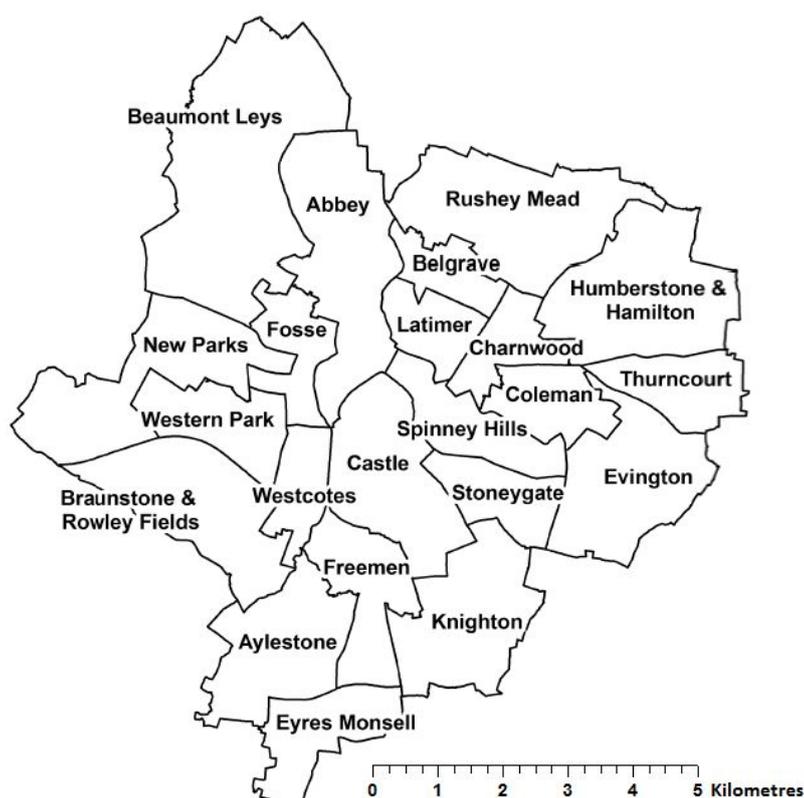


FIGURE 3.1: Definition of Leicester UA's major residential boroughs, as identified by CAS WARDS from the 2001 UK Census

The subsequent research project was conducted across Leicester Unitary Authority's (UA) 187 Lower Level Super Output Areas (LLSOA). LLSOA's are a new national geography unit for reporting statistics, containing on average 1,500 residents, and are of a higher spatial

resolution than traditionally explored Census Area Statistics (CAS) Wards. Across Leicester UA, 7-14 LLSOA's typically nest within each CAS Ward. LLSOA and CAS Ward maps were obtained through the ONS Census Geography Data Unit's access facilities provided by UKBORDERS (ONS 2001a, 2001b). Throughout this research project, model outputs are to be geographically referenced based upon the cities CAS Ward Structures (Figure 3.1), so as to simplify the reader's identification of key processes. Where information relating to the current land use of an area becomes of interest, further reference will involve the OS 1:50,000 Map of Leicester (Figure 3.2). Therefore, one is to advise the reader to take note of Figures 3.1 and 3.2 for future reference.



FIGURE 3.2: Ordnance Survey 1:50,000 scale Landranger Map for the City of Leicester (Adapted with permission from the EDINA Digimap supply service: © Crown copyright 2012)

3.2. KEY DEMOGRAPHIC AND ENVIRONMENTAL DATASETS

3.2.1. HOSPITAL ADMISSION DATABASE

A geocoded respiratory subset of NHS hospital admissions for children aged 0-15 years, residing within Leicester UA's 187 LLSOA's from 2000-09, was obtained through the Leicester City Primary Care Trust (PCT). The geocoded dataset was based on residential address, and contains details of admissions for patients regardless of whether they were admitted to a hospital within or outside of the Leicester UA catchment area.

During the 10-year study period, 24,556 visits to NHS hospitals were made by Leicester UA residents aged 0-15 years, whose primary diagnosis was recorded under the WHO International Classification of Diseases (ICD) as 'Diseases of the respiratory system' (ICD-10 J00-99). The greater proportion of these cases were classified as 'J00-06: Acute upper respiratory infections' (41.65%), 'J40-47: Chronic lower respiratory diseases' (25.92%), and 'J20-22: Other acute lower respiratory infections' (16.87%).

ICD10 Respiratory Subset	Hospital Admissions	% J00-99 Admissions	Cases Per 1,000 Children
ICD10: J00-06 [Acute URTI]	10,228	41.65	163.94
ICD10: J09-18 [Influenza & Pneumonia]	1,667	6.79	26.72
ICD10: J20-22 [Other Acute LRTI]	4,142	16.87	66.39
ICD10: J30-39 [Other Diseases URT]	3,627	14.77	58.14
ICD10: J40-47 [Chronic LRT Diseases]	6,364	25.92	102.01
ICD10: J60-70 [Lung Diseases By External Agents]	50	0.20	0.80
ICD10: J80-84 [Diseases Of The Interstitium]	17	0.07	0.27
ICD10: J85-86 [Suppurative/Necrotic LRT Disorder]	52	0.21	0.83
ICD10: J90-94 [Other Diseases Of The Pleura]	180	0.73	2.89
ICD10: J95-99 [Other Respiratory Diseases]	1,242	5.06	19.91

Note: Multiple respiratory symptoms may be responsible for an individual's hospital admission

TABLE 3.1: Aggregated children's respiratory hospital admissions (ICD-10: J00-99) experienced across Leicester UA, with patient symptoms indexed into major respiratory subsets: 2000-09

Routine UK hospital statistics classifying patients on discharge are generally thought to be of a high standard, which is maintained through a considerable expenditure of NHS resources on quality assurance activities. A systematic review of 12 studies comparing hospital episode statistics with medical records identified a median coding accuracy rate of 91% for diagnostics within England & Wales (Campbell et al 2001). Furthermore, in this review there appeared to be no significant differences in coding accuracy over time, condition type or rarity of codes being assessed. Similarly an assessment of multiple NHS hospitals during 2009/10 identified diagnosis and procedure coding errors to exist in only 11.3% patient records (Audit Commission 2010). Such levels of reported accuracy would suggest that the Leicester PCT dataset is sufficiently robust in supporting research and managerial decision-making processes.

However, in using NHS hospital episode statistics one should consider that children of affluent families might favour the use of privatised health care, although within the UK medical insurance remains viewed as a luxury rather than a necessity. A single private hospital exists within the city of Leicester, which is part of the 'Nuffield Health' group. Whilst, it remains 'good' practice and is expected of all medical partitions to record patient record updates, private health groups are not legal obliged to inform NHS trusts of any changes. Within the medical literature, no records currently exist exploring the scale to which medical procedures are perhaps under-reported in this manner. However, any under-reporting of health outcomes, in a few of the most affluent communities has the potential to mask the explanatory weight of an EJ investigation. Still, the use of private healthcare is perhaps of greater relevance to planned surgical care, in combatting patient waiting times, rather than emergency respiratory admissions where patient care occurs in a prompt fashion at national medical facilities. In terms of children's respiratory complaints, an absence of private health care is likely restricted to the primary care level, where insurance encourages the treatment of minor complaints to prevent any further deterioration. For this investigation, any confounding influence caused by the absence of admission records made to private services, is considered minimal, but remains of interest for future enquires.

For the purpose of this study, annual average LLSOA respiratory admissions were calculated by pooling together case counts across a 10-year period, with this number dividing by the number of respective years included and number of persons aged 0-15 years residing within each census area. Following this procedure a series of 1-year standardised hospital admission rates were obtained for specific subset and overall diseases of the respiratory system. In the construction of the regression models, select years within the middle of this 10-year period were omitted in-order to further evaluate model performance via R^2 cross-validation measures.

A major benefit of drawing case data from an extensive range of years in the construction of an annual admission rates, is the removal of potential temporal confounding influences, be they annual or seasonal specific events (i.e. disease outbreaks, viral epidemics). Still concern may arise in the evaluation of children's hospital cases across a decade, relating to the use of population count data, with marginal expansions or contractions potentially impeding ones analysis. On the 16th September 2010, the Office for National Statistics (ONS) released an online series of mid-year population estimates for LLSOA's across England and Wales, during 2002-2008 (ONS 2010). Across Leicester as a whole, levels of children age 0-15 years appear to have remained relatively stable, recorded at a figure of 62,387 in the 2001 UK Census and a level of 61,837 in the 2008 mid-year population estimates (ONS 2003, ONS 2010).

To evaluate the magnitude in which localised shifts in population levels might influence the dependent variables, a series of annual LLSOA J00-99 admission rates per 1,000 children were constructed using ground-truthed population counts from the 2001 UK Census and mid-year population estimates (Appendix B4). Upon comparing these surfaces one observes an exceedingly strong correlation between census and mid-year derived rates across the first half of the study period ($R^2 \geq 0.90$), appearing to slightly diminish yet remain at a significant level towards the tail end of the study period ($R^2 \geq 0.77$) (Appendix B4). This would suggest that the use of census counts across a 10-year period is a viable option, with population changes providing minimal influence on the overall trends captured.

One should also note that the ONS mid-year estimates are only experimental statistics, which as of yet do not meet the required quality standards of the National Statistics (ONS 2010). Whilst such theoretical demographic changes based upon information gathered at a national level may be of use in the provision of regional and or local authority decisions, a high level of uncertainty is expected to exist where interpretation occurs at a local level. Based on the information detailed above, it was decided that ground-truthed population counts from the 2001 UK Census were the most favourable option for the calculation of standardised admission rates across Leicester UA.

3.2.2. EXPLORING THE SMALL NUMBERS ISSUE IN HEALTH DATA

Continued improvements in the performance and availability of computing resources have fuelled our need to better understand the local relations between behaviour, environment and health; often exclusively experienced by specific subgroups of the post-industrial populace. Yet, this upsurge in demand for information about small populations is at odds with the need to preserve privacy and data confidentiality. Furthermore, the evaluation of small numbers may raise statistical issues concerning the accuracy, and thus usefulness, of the data. Whilst rates based on a full population count are not subject to errors in sampling variability, the influence of random variation may become substantial when a small number of events define the numerator. Typically, rates based on large numbers provide stable estimates of the true underlying rate, whereas rates based on small numbers may fluctuate dramatically spatially or temporally, even where no meaningful differences exist.

To explore the potential influence of random variation, one should view the raw annual hospital admission data in a temporal manner. A sample of which is provided for all respiratory cases and the two subsets of interest (Appendix's B1-B3). In terms of overall trends, citywide respiratory hospitalisation rates would appear to be at a comparable level at

the start and end of the 10-year study period. Yet, interestingly levels of respiratory hospitalisations would appear to have slightly diminished around 2004, a year which was subsequently selected to form part of the omitted data from the GWR models, so as to allow for a more critical cross-validation measure.

Specific to the small numbers issue, a standard deviation was calculated for each of the 187 LLSOA's as a measure of dispersion, summarising the amount to which 10-years of annually recorded case counts varied from the expected value of a specific locale. In terms of J00-99 admissions, a Leicester LLSOA was expected to experience 13.13 cases per annum, deviating temporally by 4.65 cases across the 10-year period. More importantly, standard deviation values <4 and >4 were respectively associated with a 10-year mean J00-99 case count of 8.09 and 16.29; indicating that a series of relatively stable temporal measurements are in existence. For J00-06 admissions, the expected LLSOA case count was recorded at 5.47 with a temporal deviation of 2.59. Here, a standard deviation of <2 and >2 were respectively associated with expected J00-06 case counts of 3.24 and 6.33. Finally, expected LLSOA J20-22 case counts were recorded at 2.22 with a temporal deviation of 1.69. Here, a standard deviation of <1 and >1 were respectively associated with expected J20-22 case counts of 0.9 and 2.5. This once again demonstrates that temporal fluctuations appear proportional to the magnitude of cases recorded within that area; therefore a minimal level of temporal random variation is to be expected.

Whilst one may note that annual case counts for J00-99 (2.1 - 32.8), J00-06 (0.8 - 16.9) and J20-22 (0.3 - 7.2) occur across a small-moderate range, a greater sense of variation is introduced to the data once weighted in relation to children's population levels. Across Leicester, each LLSOA is expected to house 333 children, potentially differing by a standard deviation value of 101 children. However, through this procedure an aspect of random variation may be spatially introduced if a small populace is introduced to define the numerator. As such, statistical smoothing algorithms in the form of 'Bayesian nearest neighbour' and 'Poisson kriging' are often implemented to filter local small-scale variations from mapped health rates, enhancing the larger-scale regional trends.

Bayesian methods depend on the prior distribution of the disease rates (from the data itself) multiplied by a likelihood function, to produce a posterior distribution from which the Bayesian rates are determined. Here, observed rates are shrunk towards a global or local mean in the case of a nearest neighbour approach, resulting in an estimate of the 'true' value through borrowing the strength of other spatial units. If a raw rate estimate has a small variance (based on a large population at risk) then it will remain essentially unchanged. In contrast to traditional smoothers, geostatistical techniques go beyond the filtering of noise

allowing for an intuitive decomposition of structured variability according to spatial scale, as modelled by the semivariogram. For the Leicester dataset, Pearson's R measures of 0.82, 0.75 and 0.73 respectively measured the level of association between raw admission rates and their outputs under a Bayesian first-order nearest neighbour scheme ($P \leq 0.01$). Comparable values of 0.79, 0.72 and 0.70 were also respectively produced when correlating these raw rates with a Poisson smoother ($P \leq 0.01$). Whilst some local change in the hospital rates had occurred, these measures demonstrate that the presence of random spatial variation has a minimal impact on the overall trend of the datasets.

Although these smoothing approaches are recommended for use by medical professionals distinguishing community health in a purely cartographic sense, their application within this body of research was deemed unnecessary for evaluating dose-response relations; where the applied models actively incorporated the spatial variation of both dependent and independent parameters. In reality, any spatial smoothing treatment may falsely increase the spatial nature of the independent datasets, forcefully causing aspects of over-interpretation particularly by the GWR models. Furthermore, reducing the spatial gradient of health outcomes not only removes one's ability to evaluate how transitions in socio-environmental determine boundaries in health, but also opens the possibility for a concealment of exclusively local information (i.e. outlying hot-spots in health marking junction specific areas of traffic congestion).

3.2.3. SOCIAL COVARIATES

Population counts, ethnic composition and variables of deprivation recorded within the 2001 UK Census were accessed from Casweb, hosted by the MIMAS data centre as part of the ESRC census programme. Ethnic minority groups of interest were selected to represent the major post war migration trends experienced within Leicester from Commonwealth and European Countries, as described in Table 3.4. Here, children classified as [Indian] exclusively represented Leicester's 'Indian' group; with [Pakistani] and [Bangladeshi] children forming the city's 'Other South Asian' category; and children of [White Irish] and [Other White] origins recording populations of 'White Non-British' children. Under a traditional classification, persons of Afro-Caribbean ethnicity are residents of the United Kingdom who are of West Indian background and whose ancestors were primarily indigenous to Africa. However, as immigration to the United Kingdom from Africa increased in the 1990s, the term has been used to also include UK residents solely of African origins (Cappuccio et al 1998, Reeves et al 2001); thus allowing for a more mobile account of this ethnic grouping. Looking from a

technical standpoint, the subjective criteria used within the 1991 and 2001 census coding, means that families with parental links to the Caribbean may still choose to define themselves in their census as Black African. It is on this basis that ‘Afro-Caribbean’ children study group, is constructed from both the [Black Caribbean] and [Black African] divisions of the 2001 UK census.

Measures of Deprivation were recorded using Carstairs Index scores of unweighted UK Census variables exploring unemployment, overcrowding, car ownership and defined levels of social class (Carstairs & Morris 1991).

Variable	2001 UK Census Table	LLSOA Component Calculation (x100)	LLSOA Mean	LLSOA Std.
Unemployment	KS09b	(Males 16-74 years / Unemployed Males)	6.39	3.02
Mobility	UV062	(Households No Vehicle / All Households)	37.45	13.93
Overcrowding	UV058	(1.0+ Person Per Room / All Households)	4.10	3.57
Low Social Class	UV050	(Social Class D & E / All Persons)	45.67	13.17
<i>LLSOA Carstairs Index Score: Summation of Z-Scores for the four variables</i>				

TABLE 3.2: Calculation of Leicester UA’s LLSOA Carstairs Index Scores through the use of appropriate ONS 2001 UK Census demographic variables

Carstairs Index scores have been extensively applied within spatial epidemiology (Maheswaran & Elliott 2003, Gregory 2009), and were favoured over the more detailed and frequently recorded Indices of Multiple Deprivation (IMD) due to these indices containing health and pollutant measurements within their calculation. Carstairs Index scores at the LLSOA level within Leicester UA range from -7.19 (affluent) to 9.14 (considerably deprived), and show good levels of correlation with IMD 2007 measurements ($R^2=0.80$), thus suggesting that patterns of deprivation in Leicester remain broadly unchanged from 2000-2009.

To account for confounding measurements of overall population health, levels of smoking prevalence and obesity for persons 16 years and older were obtained via the Office for National Statistics (ONS 2008b) ‘Healthy Lifestyle Behaviours: Model Based Estimates, 2003-2005’ geocoded to the LLSOA (Table 3.4).

Passive smoking in the family home is considered to pose a major risk to young children’s health. Jones et al (2011) in an updated systematic review and meta-analysis of 60 studies identified smoking by one parent (OR: 1.22), both parents (OR: 1.62) or other household members (OR: 1.54) to significantly increased the risk of childhood LRI’s. Under a WHO modelling framework, the annual global burden of disease from exposures to second-hand smoke for children is calculated at a value of 6,614,900 disability-adjusted life-years; a figure equal to 61% of the overall population’s burden from second-hand smoke (Oberge et al 2011). Likewise obesity is considered to be a well-known burden on respiratory health, as

recorded in relation to the onset of asthma (Beuther & Sutherland 2007, Chen et al 2013). Here, a recent 6 study meta-analysis of 18,760 children reported a dose-response effect of elevated BMI on asthma incidence, when comparing normal-weight subjects (BMI <25) to those deemed overweight (OR: 1.19) or obese with a BMI >30 (OR: 2.02) (Chen et al 2013).

However, one should consider that these modelled based estimates of obesity are only representative of the adult population, extrapolated in this study as a marker of childhood rates. Still, this is deemed to be a suitable proxy with parental obesity often identified as the predominant risk factor for childhood obesity, probably owing to a combination of genetic, social and environmental factors. For instance, a 16-year follow-up study of 4,788 mother-father-child trios from a Finnish birth cohort reported a strikingly high risk of becoming overweight for boys (OR: 5.66) and girls (OR: 14.84) where both parents were classified as exhibiting long-term weight issues (Jaaskelainen et al 2011). In terms of a hereditary link, quantitative genetic modelling within a UK sample of 5,092 twin pairs aged 8-11 years born during the current obesity pandemic, identified substantial heritability (77%) for BMI (Wardle et al 2008). Subsequently, it has been established that children with two copies of lower-risk alleles from the FTO genotype respectively eat 20.9% and 24.9% less in the absence of hunger than those with one or two higher risk alleles (Wardle et al 2009).

Yet, in following a US cohort of 2,913 normal-weight children over a 6-year period, Strauss & Knight (1999) also demonstrate the key role of community and household socio-environmental influences in the development of childhood obesity. Whilst an obese mother (OR 3.62) appeared the driving demographic factor; a low family income (2.91) and reduced cognitive stimulation at a child's residence (2.64) also posed substantial risks (Strauss & Knight 1999). Current research from the US examining the influence of obesogenic environments on 730 families, identify reduced levels of childhood obesity within neighbours that scored highly for metrics associated with 'physical activity' and 'healthy eating' when compared to neighbourhoods low on both measures (OR: 0.41); after adjusted for parent weight status and demographic factors (Saelens et al 2012). Likewise, a reduced level of adult obesity was detected within neighbourhoods supportive of physical activity and healthy eating (OR: 0.57) compared to neighbourhoods low on both measures (Saelens et al 2012). Based upon the knowledge that obesity is predominantly a hereditary phenomenon, and that the obesogenic environment influences adult and childhood weight in a similar manner, one may conclude the ONS adult obesity estimates offer a suitable proxy for this investigation.

The modelled healthy lifestyle estimates (ONS 2008b), were constructed from a pooling of individual respondent information contained within the 2003, 2004 and 2005 Health Surveys for England (HSfE). The project was commissioned by the NHS Information Centre for

health and social care, to meet user requirements for local level measurements in health domains where no suitable administrative sources exist. In total 28,993 adults provided a valid smoking status, with 7,024 participants reporting that they were current smokers; and 24,974 adults provided valid height and weight data, from which it was calculated that 5,874 were obese (Scholes et al 2007).

As the methodology used to produce estimates of local health remains relatively new, and is subject to further consultation, modification and development, one is advised to adopt these datasets with caution. Whilst it is strongly recommended that users view the model-based estimates in light of their broad confidence intervals, analysis has shown that the datasets remain suitable for distinguishing areas of high/low values (Scholes et al 2007). Still, they are unlikely to precisely mirror any available measures from local studies or surveys, and as such do not represent an estimate of the actual prevalence; although several validity documents have documented high levels of correlation (Scholes et al 2008a, 2008b).

Here, internal validation of local model outputs was achieved through: (I) Direct estimate comparison, (II) Residual analysis, (III) Calibration diagnosis, and (IV) Stability analysis (Scholes et al 2008a). In the 549 output areas containing >15 HSfE respondents, Pearson's correlation coefficients between the modelled and direct 2003-2005 HSfE estimates were respectively recorded at 0.37 and 0.61 for obesity and smoking. Modelled residuals were also observed to be randomly and evenly scattered around their expected mean value 0, indicating that no important relationships had been omitted. In their final construction stages, model-based estimates were locally calibrated so that a population-weighted output measure equalled previously published direct estimates for each Strategic Health Authority (SHA). Across England ratio adjustment factors of 1.00 and 1.02 were respectively reported for smoking and obesity, with the East Midlands adjustments of 1.02 were recorded for both lifestyle choices. The minimal amount of scaling required to directly match SHA surveys, thus demonstrates a substantial level of quality assurance. Finally, to check for the presence of spurious relations within the model the dataset was split into two halves at random, allowing for the re-estimation of model parameters. Here, respective correlations of 0.99 and 0.92 were respectively recorded for smoking and obesity, providing confidence in the robustness of the estimates.

External validation was initially achieved through comparing model-based estimates with records from the: (I) 2000-2002 HSfE, and (II) 2003 Merseyside Health Survey (Scholes et al 2008a). In the 577 output areas containing >15 HSfE respondents, correlation coefficients between the modelled and direct 2000-2002 HSfE estimates were respectively recorded at 0.41 and 0.55 for obesity and smoking. Yet in providing this independent test of quality, it

should be noted that comparisons over a different timeframe may confound results where changes have occurred. More suitably, the 2003 Merseyside Health Survey of 72 output areas containing >15 respondents, respectively record strong correlations of 0.50 and 0.55 for obesity and smoking. Overall this demonstrates that whilst the healthy lifestyle datasets are unable to record actual prevalence levels, they are capable of capturing the overall trends, allowing for this investigation to suitably account for the impact of such phenomenon.

3.2.4. ROAD-TRANSPORT EMISSIONS

In correlating measures of pollution with census data, an assumption is made that an individual's exposure occurs entirely within the relevant census unit, thus potentially causing a significant exposure bias. The Department for Transport (DfT) National Travel Survey: 2009, identifies that primary school pupils (aged 5-10) on average travel only 1.5 miles to school (DfT 2010). Therefore one may assume for the most part that outdoor exposures experienced by children at school would be similar to those experienced at their place of residence due to proximity.

Residential exposure to particulate matter up to 10 μ m in diameter generated by road-transport (TPM₁₀) was determined through the interpolation of 2,157 datum points contained within the '2008 1x1km Road Transport PM₁₀ Emission' map of Leicestershire, provided by the UK National Atmospheric Emissions Inventory (NAEI) (AEA Technology & DEFRA 2010). The NAEI emission models are derived through combining emission factors with annual average daily flows, which are obtained from direct counts of vehicle compositions along sections of major roads (A-roads and motorways), and through modelled flows along minor roads. Incremental amendments to the emission models methodology since its implementation, restricts the direct comparison of previous years models. Therefore data from the latest version of the '2008 1x1km Road Transport PM₁₀ Emission' map was implemented to broadly represent traffic emission levels during 2000-2009.

Here, fuel consumption and emission factors are expressed in grams of emissions per kilometre driven for each detailed vehicle class, derived from vehicle emission test data over different drive cycles (TRL 2009). Hot exhaust emission and the related fuel consumption estimates are calculated within the NAEI across 6 major vehicle classes, based on fleet composition contained within the DVLA's licensing database (Tsagatakis et al 2010). Vehicle fleet age profiles and fuel mix are fixed at a national level, whereas fleet mix varies geographically (urban, rural and motorway settings). Estimates of the distance travelled by vehicles whilst operating under cold start conditions are derived from the 2001 UK Census,

locating the origin and destination of trips conducted between residence and place of work, with levels of car ownership defining the level of trips from the home to other locations. These geographic trip distributions are reconciled with the outputs from Department for Transport TEMPro (Trip End Model Presentation Program) model (DfT 2009), and are provided with emissions estimates based on the COPERT III model for cold-starts (Ntziachristos & Samaras 2000). Finally, non-exhaust PM₁₀ contributions involving brake and tyre wear, in addition to the abrasion and general deterioration of road surfaces, are incorporated in the NAEI road-transport models (Tzagatakis et al 2010). Any national map like this will incorporate a number of assumptions that may influence its absolute local accuracy, which pose as a potential limitation for this study; although it should be recognised that this is currently the best available data source for this model input.

LLSOA centroid estimates of experienced road transport emissions were obtained through Ordinary Kriging (OrK) interpolation function within SpaceStat 3.5.6. The kriging process (Krige 1966) constructs an optimal interpolator for the variable of interest by minimizing the variance of the estimation error as enumerated by the spatial covariance, subject to unbiased conditions. The degree of spatial dependence of a spatial variable \mathbf{Z} (e.g. TPM₁₀ emissions) is described by the variogram function, $2\boldsymbol{\gamma}$, where the semivariance $\boldsymbol{\gamma}$ is defined by (Goovaerts 1997):

$$\boldsymbol{\gamma}(\mathbf{h}) = \frac{1}{2\mathbf{N}(\mathbf{h})} \sum_{\alpha=1}^{\mathbf{N}(\mathbf{h})} [\mathbf{Z}(\mathbf{u}_{\alpha}) - \mathbf{Z}(\mathbf{u}_{\alpha} + \mathbf{h})]^2 \quad (\text{Eq.1})$$

Where \mathbf{h} is a vector presenting the distance and relative position of the two observations $\mathbf{Z}(\mathbf{u}_{\alpha}) - \mathbf{Z}(\mathbf{u}_{\alpha} + \mathbf{h})$, and $\mathbf{N}(\mathbf{h})$ is the number of such paired observations for the distance band in question. Thus the variogram is a measure of the average dissimilarity between data as a function of their separation in geographical space. A variograms search vector \mathbf{h} may be defined in terms of purely distance (omnidirectional) or, alternatively across set distances limited by direction if a degree of anisotropy and knowledge of such trends exist (directional). In this analysis an omnidirectional variogram was favoured, on the basis that road traffic at an individual and or higher level, is likely to branch off from major routes in all directions in a relatively even manner, so as to maximise trip efficiency. Furthermore more sample pairs are used within the construction of an omnidirectional than any directional variogram, thus increasing the likelihood of creating a clearly interpretable structure. In the construction of Leicestershire's TPM₁₀ emission omnidirectional variogram, 40 lag counts (number of bins) separated by a lag distance of 600m (width of classes) and a lag tolerance of

300m, were used to define individual classes of data pairs. It should be noted that if a point pair falls within the tolerance of two lag bins, then the pair is excluded from the semi-variance calculation.

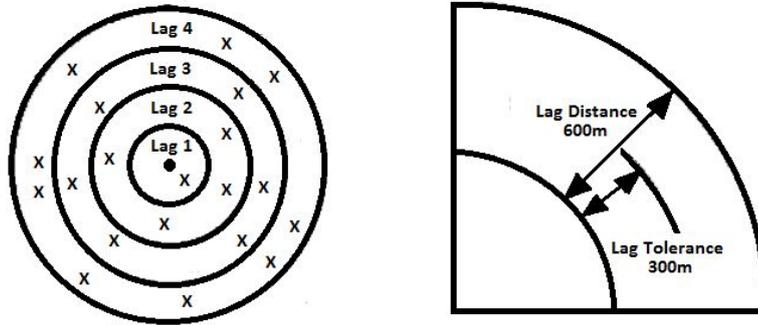


FIGURE 3.3: Graphical representation of the search strategy used by the omnidirectional variogram

TPM₁₀ emission semivariogram values γ across Leicestershire, for paired localities separated by a distance h , were approximated by the following equations (Webster & Oliver 2007):

$$\gamma(h) = \begin{cases} c_0 & \text{If } 0 \\ c_0 + c_1[1 - \exp(-h/a_1)] & \text{If } 0 < h \leq a_1 \\ c_0 + c_1 + c_2[1.5(h/a_2) - 0.5(h/a_2)^3] & \text{If } a_1 < h \leq a_2 \\ c_0 + c_1 + c_2 & \text{If } h > a_2 \end{cases} \quad (\text{Eq. 2})$$

Where the sill ($c_1 = 0.082$) and threshold ($a_1 = 1428.944\text{m}$) of the short-range component of the variation are represented by a negative exponential model, and the sill ($c_2 = 0.125$) and threshold ($a_2 = 18462.269\text{m}$) of the long-range component are described through a spherical model. The level of spatially uncorrelated noise within the modelled semivariogram, known as the nugget effect ($C_0 = 3.141 \times 10^{-10}$), was noted to have an insignificant influence on calculated levels of semivariance. The mathematical models (Eq.2) were identified to suitably fit the TPM₁₀ emission variogram ($R^2 = 0.99$), thus suggesting that the spatial structure of the data was sufficiently captured by the OrK interpolation. These fitted semivariogram model outputs in combination with a Lagrangian parameter were subsequently used to determine the optimal kriging weights λ_α , under unbiased constraints; a procedure described in greater detail within the successive section.

OrK LSOA centroid estimates of road-traffic emissions $\mathbf{Z}^*(\mathbf{u})$, were obtained through calculating the linear weighted moving averages of the $\mathbf{n}(\mathbf{u})$ observations of the road-traffic emission datum $\mathbf{Z}(\mathbf{u}_\alpha)$ plus the constant local mean $\mathbf{m}(\mathbf{u})$ within a given neighbourhood centred on an unsampled locality \mathbf{u} (Goovaerts 1997):

$$\mathbf{Z}^*(\mathbf{u}) = \sum_{\alpha=1}^{n(\mathbf{u})} \lambda_{\alpha}(\mathbf{u}) \cdot \mathbf{Z}(\mathbf{u}_{\alpha}) + \left[\mathbf{1} - \sum_{\alpha=1}^{n(\mathbf{u})} \lambda_{\alpha}(\mathbf{u}) \right] \mathbf{m}(\mathbf{u}) \quad (\text{Eq. 3})$$

Ordinary Kriging (OrK) is the most widely applied kriging method as it required neither awareness nor stationarity of the mean over the entire area. As such, the unknown local mean $\mathbf{m}(\mathbf{u})$ is filtered from the linear estimator through forcing the kriging weights λ_{α} to sum to 1, leading to an OrK estimator of (Goovaerts 1997):

$$\mathbf{Z}^*(\mathbf{u}) = \sum_{\alpha=1}^{n(\mathbf{u})} \lambda_{\alpha}(\mathbf{u}) \cdot \mathbf{Z}(\mathbf{u}_{\alpha}) \quad \text{with} \quad \sum_{\alpha=1}^{n(\mathbf{u})} \lambda_{\alpha}(\mathbf{u}) = \mathbf{1} \quad (\text{Eq. 4})$$

Through forcing the kriging weights to sum to 1, it is thus possible to minimise the estimation of error variance $\sigma_{\mathbf{E}}^2$ under the unbiased constraint common across kriging methods (Goovaerts 1997):

$$\begin{aligned} \sigma_{\mathbf{E}}^2(\mathbf{u}) &= \mathbf{Var} \{ \mathbf{Z}^*(\mathbf{u}) - \mathbf{Z}(\mathbf{u}) \} \\ \mathbf{0} &= \{ \mathbf{Z}^*(\mathbf{u}) - \mathbf{Z}(\mathbf{u}) \} \end{aligned} \quad (\text{Eq. 5})$$

The minimisation of the error variance under the unbiased constraint calls for the definition of a Lagrangian \mathbf{L} , which is a function of the data weights λ_{α} and a Lagrangian parameter $2\mu(\mathbf{u})$, under the following constraints (Goovaerts 1997):

$$\begin{aligned} \mathbf{L} &= \sigma_{\mathbf{E}}^2(\mathbf{u}) + 2\mu(\mathbf{u}) \cdot \left[\mathbf{1} - \sum_{\alpha=1}^{n(\mathbf{u})} \lambda_{\alpha}(\mathbf{u}) \right] \\ \frac{\mathbf{1}}{2} \cdot \frac{\partial \mathbf{L}(\mathbf{u})}{\partial \mu(\mathbf{u})} &= \mathbf{1} - \sum_{\alpha=1}^{n(\mathbf{u})} \lambda_{\alpha}(\mathbf{u}) = \mathbf{0} \end{aligned} \quad (\text{Eq. 6})$$

In the case of the OrK system of equations, expressed in terms of the variogram, the kriging weights turns out to be (Goovaerts 1997):

$$\begin{cases} \sum_{\beta=1}^{n(\mathbf{u})} \lambda_{\beta}(\mathbf{u}) \cdot \gamma(\mathbf{u}_{\alpha} - \mathbf{u}_{\beta}) - \mu(\mathbf{u}) = \gamma(\mathbf{u}_{\alpha} - \mathbf{u}) & \alpha = 1, \dots, n(\mathbf{u}) \\ \sum_{\beta=1}^{n(\mathbf{u})} \lambda_{\beta}(\mathbf{u}) = \mathbf{1} \end{cases} \quad (\text{Eq. 7})$$

The left hand side of the system describes the dissimilarities between the data points α and β , while the right hand shows the dissimilarities between each data point and the estimation point \mathbf{u} . Thus, the OrK system is solved through a standard linear optimisation approach to approximate the solution of over determined systems, by minimising the sum of the squares of the errors made in solving every single equation. Once the kriging weights (and Lagrange parameter) are obtained, the ordinary kriging error variance is given by (Van-Groenigen 2000):

$$\sigma_E^2(\mathbf{u}) = \sum_{\alpha=1}^{n(\mathbf{u})} \lambda_{\alpha}(\mathbf{u}) \cdot \gamma(\mathbf{u}_{\alpha} - \mathbf{u}) + \mu(\mathbf{u}) \tag{Eq. 8}$$

In theory, no other interpolation procedures can produce better estimates (being unbiased, with minimum error) than kriging techniques, if founded upon an accurately modelled variogram. Geostatistical methods with weights derived through a variogram provide a more accurate interpolation estimate than deterministic methods (i.e. Inverse Distance Weighting or Nearest Neighbour interpolation), as weights are not constructed as an arbitrary and uniform function of distance. Furthermore, deterministic methods unlike kriging techniques do not provide prediction standard error outputs, therefore justifying the use of such models may be problematic.

Model performance was validated through predicting an omitted proportion of the Leicestershire 1x1km NAEI dataset to directly compare with their real-world values (Appendix B5). Table 3.3 indicates over-smoothing to have only a minor impact on modelled outputs, and that an acceptable model performance may be achieved where datum is sparse. Thus it is believed that the model is an acceptable means of deriving TPM₁₀ emission levels at a more localised level than the 1x1km NAEI grid that data values currently exists in. OrK outputs defined through the theoretical two model semivariogram, accounting for the 8 nearest NAEI road transport emissions measurements, produced TPM₁₀ kriged estimates for Leicester UAs LSOA centroids ranging from 0.333-2.648 tonnes/year with a small average variance of 0.026 (Appendix B6).

R²: Real vs. Predicted Values of Omitted NAEI Locations (%)					
	10% Data	20% Data	30% Data	40% Data	50% Data
4 NAEI Observations	0.91	0.83	0.76	0.73	0.66
8 NAEI Observations	0.90	0.82	0.75	0.71	0.64
12 NAEI Observations	0.89	0.82	0.75	0.72	0.65

TABLE 3.3: OrK model validation achieved through comparing real-world with predicted TPM₁₀ levels accounting for different degrees of spatial smoothing and sampling intensities.

3.3. DATASET DISTRIBUTIONS

Table 3.4 provides a statistical summary of the social-lifestyle, health and environmental variables of interest previously discussed, with respect to their original sources. All variables are to be comprehensively incorporated within the projects spatial modelling strategy, with the exception of markers for ‘White British’ children, which are discussed with relation to pattern based analyses.

	Min.	Max.	Mean	Std. Dev.	Skewness	Kurtosis
LEICESTER CITY PCT DATASET: 2000-09						
<i>Annual Hospital Admissions Per 1,000 Children [Age 0-15 Years]:</i>						
▪ ICD10 J00-99	13.89	147.83	39.43	15.05	2.59	14.54
▪ ICD10 J00-06	5.08	95.65	16.82	8.59	4.79	38.34
▪ ICD10 J20-22	1.58	34.78	6.68	3.89	2.83	15.61
ONS 2001 UK CENSUS						
<i>Children’s Ethnic Groups [Age 0-15 Years]:</i>						
▪ % White British	4.41	93.71	53.69	30.72	-0.31	-1.44
▪ % White Non-British	0.00	7.07	1.56	1.45	1.23	1.40
▪ % Indian	0.00	90.07	28.80	26.57	0.71	-0.91
▪ % Other South Asian	0.00	35.71	5.74	6.87	1.81	3.66
▪ % Afro-Caribbean	0.00	33.33	2.82	3.85	4.19	25.69
<i>Deprivation Scores Across Leicester UA:</i>						
▪ Carstairs Index	-7.19	9.14	0.00	3.16	0.15	-0.48
ONS HEALTHY LIFESTYLE BEHAVIOURS [AGE 16+ YEARS]: 2003-05						
▪ % Smoking Prevalence	11.10	53.50	27.11	11.50	0.50	-1.01
▪ % Obesity Prevalence	12.10	30.30	24.30	4.72	-1.13	0.48
UK 1x1KM NAEI [INTERPOLATED]: 2008						
▪ TPM ₁₀ Emissions (t/yr.)	0.33	2.65	1.04	0.38	1.41	3.03

TABLE 3.4: Descriptive statistics for social-lifestyle, health and environmental factors recorded across Leicester UA

Of traditional statistical interest are measurements of skewness and kurtosis, which are fundamental in comparing dataset distributions to those experienced under normal circumstances. Here, skewness is a measure of symmetry or more precisely the lack of symmetry within the dataset. Meanwhile, kurtosis measures whether the data in question is peaked or flat relative to that of a normal distribution. Both of these are considered important credentials under classical statistical requirements governed by normality assumptions. Table 3.4 shows an excessive level of kurtosis (>3) to be particularly strong within the hospital admission datasets and, for markers of ‘Other South Asian and ‘Afro-Caribbean’ children’. For these five variables in question, a positive measure of skewness also indicates that their

distributions are skewed to the right. To better understand the collective distributions of all twelve variables, a series of Box-Whisker plots were subsequently constructed, following the normalisation of each individual distribution on a scale of 0-1 (Appendix B7).

Favourably, Box-Whisker plots are capable of providing a measure of central location (median) and two measures of spread or variation (range and inter-quartile range), in addition to visually identifying the orientation of the median relative to the quartiles (skewness). More specifically, the box itself represents the middle 50% of the data bounded by the first and third quartiles, which respectively mark where 25% of the data fall below and above.

As indicated by the statistical summary of Table 3.4, all three measures of respiratory health are associated with a strong positive skew, following a Leptokurtic distribution (consisting of a tall thin peak). Here 25% of the data is distributed along a heavy right tail, some of which are traditionally deemed to be extreme outliers under traditional definitions (Appendix B7). Similar trends are to be found for markers of ethnic minorities, with the exception of 'Indian' children whose distribution remains defined by a positive skew, but across a considerably larger spread. Still one should note that high outliers are present across all ethnic minority groups, in-line with those distributions for children's respiratory hospital admissions. Values of deprivation appear relatively normally distributed, as one might expect from a summation of z-scores. Levels of adult smoking prevalence are also defined by a relatively centralised box, but one should note that a mild positive skew is present here. Only measures of obesity and 'White British' children are characterised by a strong negative distribution.

From these traditional statistical summary measures one can conclude that the datasets in question appear represented by relatively unique distributions, affected by outlying values. However, perhaps of far greater importance is the manner in which traditional distribution measures are unable to consider the spatial arrangements of the dataset. Thus the decision to discard extreme values must be made with particular care, especially when evaluating interactions of a spatial nature, as these values are typically of greater interest. In some circumstances the spatial location of extreme values may be helpful in detecting erroneous data if values are isolated, although one would consider this a highly unlikely situation, in view of the high sense of scrutiny that census and medical statistics experience within the UK. Thus, if there is no physical reason for discarding extreme values or treating them separately, one may wish to reduce their influence via a data transformation procedure.

Unfortunately, when dealing with data of a spatial nature the user is unlikely to be fully aware of the consequences of such transformations. In reality, the back-transform may erase most of the benefits of satisfying more robust statistics, with such actions requiring

careful consideration, calling for far more than a simplistic desire to meet traditional distribution measures (Goovaerts 1997). In the literature review it was also shown that EJ is an explicitly spatial problem. On this basis the spatial distribution of variables is considered to be of primary importance, something which the transformation of datasets may potentially distort. For these reasons, the use of regression strategies specifically designed to either account for or filter out the spatial components of a dataset (inclusive of GWR and Multilevel models), were favoured over traditional transformations.

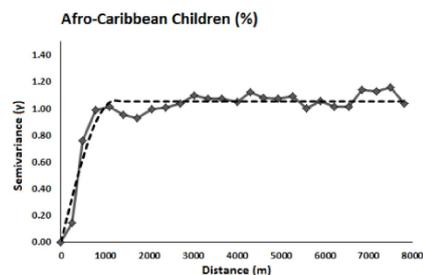
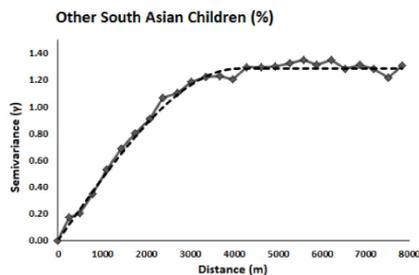
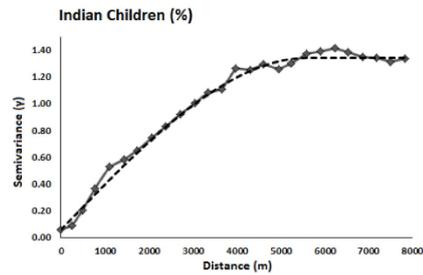
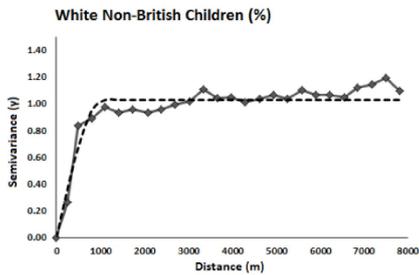
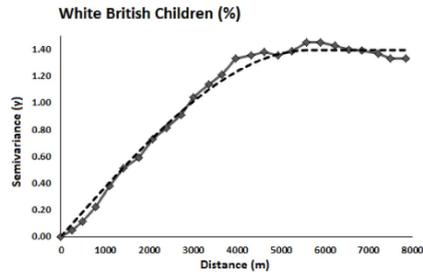
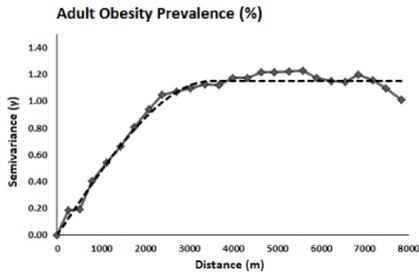
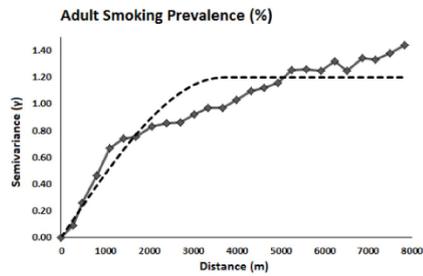
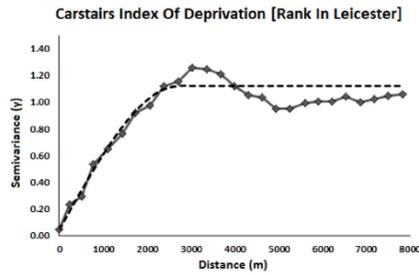
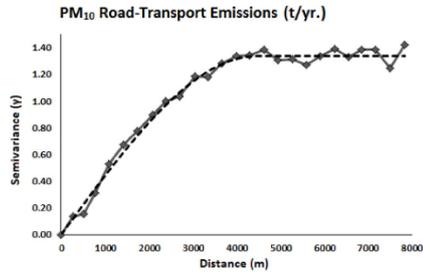
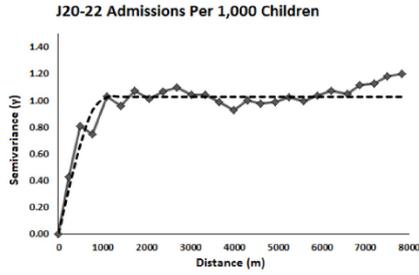
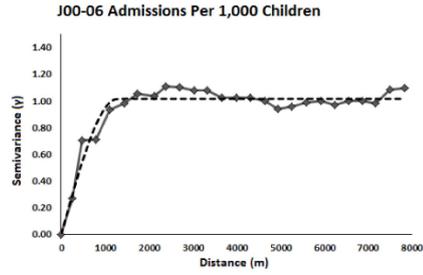
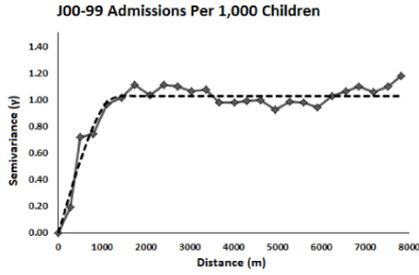
To confirm the spatial distribution of the Leicester UA dataset, a series of variograms were constructed to determine the rate and nature of change in citywide levels of spatial continuity, amongst the aforementioned variables. Here, the variogram is shown to be of interest in its own right, as well as a component of the kriging procedure. To provide a direct comparison of spatial distributions a standardised variogram model, rescaling the magnitude of dissimilarity terms of variance at specified lags (**h**) was favoured, so as to correct for the preferential sampling of high values. The rescaling accounts for the large change in variance from one lag to the next, minimising the likelihood for erratic fluctuations to occur in addition to providing a more accurate estimate of the short-range variability (Goovaerts et al 2005):

$$\gamma(\mathbf{h}) = \frac{1}{2N(\mathbf{h}) \cdot \sigma^2(\mathbf{h})} \sum_{\alpha=1}^{N(\mathbf{h})} [Z(\mathbf{u}_{\alpha}) - Z(\mathbf{u}_{\alpha} + \mathbf{h})]^2$$

(Eq.9)

Upon viewing the variogram plots (adjusted for intra lag variation) one may observe that a common degree of spatial continuity, and thus distribution, exists between the health and socio-environmental factors of interest across Leicester UA (Figure 3.4). This mutually strong sense of spatial continuity may be verified through the plotting of theoretical model estimations against those real-world measurements of all other variables across Leicester (Appendix B8). Generally theoretical models were found to accurately describe their variograms original data points, with R-square values ranging from 0.88-0.99. Theoretical models for children's J00-99 admissions were observed to fit moderately well to those variogram plots describing the spatial distributions of socio-environmental influences across the city ($R^2 \geq 0.54$).

FIGURE 3.4 [PAGE 86]: Variogram plots (adjusting for intra lag variation), each fitted with a singular spherical theoretical model describing the variables rate of spatial continuity across Leicester UA. Omnidirectional variograms were characterised to include 40 lags, each separated by 320m.



3.4. COMMUNITY CREATED PRIVATE-TRANSPORT EMISSIONS

Annual LLSOA estimates of private road-transport PM₁₀ emissions created from individual communities were derived by combining vehicle fleet compositions with workforce trips, which were assumed to represent the significant proportion of population movements. Such a procedure was applied within the later sections of the projects analysis; exploring the environmental accountability and general social attitudes of specific communities (non-regression based enquiry).

Vehicle compositions (%) within each LLSOA were derived from a summary of privately owned vehicles registered with the Driver Vehicle Licensing Agency (DVLA) in 2010, provided by Callcredit Information Group. Vehicle counts were disaggregated into their corresponding LLSOA's, from 4 postal sectors for vehicles older than Euro III, and 22 postal districts for vehicles Euro III onwards (Appendix B9). This disaggregation procedure was achieved through the use of the Postcode Best Fit (PBF) Methodology, developed by the ONS to produce population estimates for a range of different geographies which are entirely consistent with each other, regardless of whether target estimates may be formally aggregated into their respective source geographies (non-overlapping). The online facilities to conduct the PBF methodology were accessed through the MIMAS service GeoConvert, as part of the ESRC census programme.

The PBF methodology involves a population weighted ratio transformation, founded on the initial apportionment of LLSOA census population measurements (approximately 1,560 persons) to an individual postcode (approximately 40 persons), using age and sex information contained within NHS GP postcode level patient registers (ONS 2011). A preliminary enquiry of the PBF methodology assessing the transferability of population geographies from 11,103 Wards (1991 census) to 2,780 Postal Districts (1999), recorded a high degree of fit (91.4%) despite the low degree of spatial hierarchy (46.5%) in the datasets (Simpson 2002). Meanwhile respective levels of hierarchy and fit of 17.2% and 78.8% were recorded for the conversion of 11,103 Wards into 9,252 Postal Sectors (Simpson & Yu 2003). Although the PBF methodology has been formed with demographic principles in mind, the project also encourages a wider level of uptake, facilitating the use of more innovative and historical datasets.

An early application of such procedures is recorded by Debenham et al 2001, who combined 1991 census data with updated postal sector unemployment claimant records, to explore workplace-based characteristics and commuting linkages across Yorkshire and the Humber. More recently, Norman et al 2011 transformed pseudo postcode sector (Scotland) and ward (rest of UK) vital statistic records as well as 1991 census data for the Townsend Index, into their updated 2001 boundary units, when examining the relationship between

rising premature mortality and persistent deprivation. In a UK exploration of pathways to obesity, Stafford et al 2007 converted ward based National Health Survey (1994-1999) registers across England to postal sector units, conforming to their Scottish counterparts. In addition, records of social disorder (crime, policing, physical dereliction) were converted from a Local Authority level, for use alongside potential explanatory measures of local infrastructure and physical characteristics sourced at post sectors. Thus demonstrating how population-weighted restructuring procedures may be applied to match demographic and environmental datasets with loose ties to population structures, sourced across several spatial scales.

As car-ownership is thought to share a noteworthy tie with elements of population structuring, the PBF methodology was considered the most appropriate method for disaggregating the Leicestershire DVLA summary dataset. In its initial data preparation the DVLA data summary was split into 25 vehicle groups defined by vehicle age (Pre-Euro, Euro I, Euro II, Euro III, Euro IV, Euro V) category (car, commercial vehicle, motorcycle) and fuel type (petrol, diesel). Motorcycles were uniformly classified due to low levels of ownership. The 25 designated vehicle groups were taken to account for a proportion of 2001 UK Census recorded LLSOA trips to work, by mode of transport for persons aged 16-74 in employment (ONS 2003). Each group was assigned a distance based PM_{10} emission factor (g/km) for urban driving conditions, taken from the Department for Transport (DfT) Emission Factors 2009. These emission rates were then combined with a commissioned ONS 2001 UK Census dataset, detailing the daily method of travel to work by the average Euclidean distance (km) travelled within each LLSOA.

3.5. MODELLING STRATEGY

Quantitative modelling techniques used within this research project to describe the influence of socio-environmental stimuli on respiratory outcomes, fall into three broad categories, which include: (I) Pattern detection, (II) Dose-response evaluation, and (III) Distance-response evaluation (Figure 3.5).

Initially indicators of statistical association are used to determine whether linear or non-linear relations exist, so as to assist with the selection of independent variables in the successive stages of model development. Here two suitable test were identified, Pearson's R which measures linear relations within the raw data, or Spearman's Rho which ranks observations so that monotonic relations may subsequently be explored.

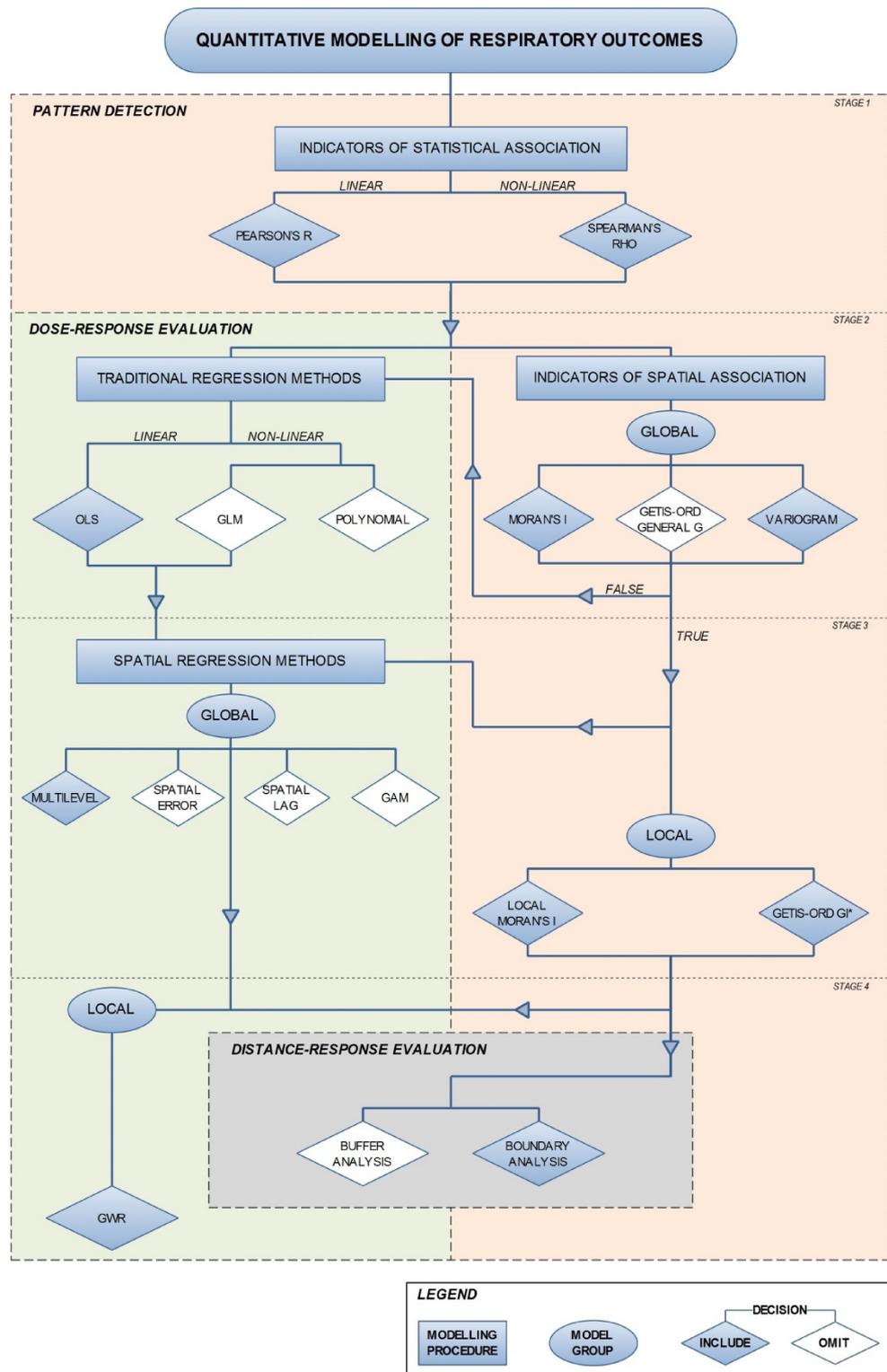


FIGURE 3.5: Proposed quantitative spatial modelling strategy for describing the influence of socio-environmental stimuli on respiratory outcomes

The second stage of this modelling strategy intends to develop upon this exploratory data analysis through the use of global indicators of spatial association, which define a variable's magnitude of spatial continuity across the entire study area of interest. Here, the variogram uses a graphical format to describe the measures in variance as a function of distance between

all pairs of sampled locations. Meanwhile, the Moran's I and Getis-Ord General G return a single index measure of clustering, across user defined sets of census units deemed to be neighbouring. If conducted in an incremental manner (i.e. first-order, second-order, etc.) test index measures may be plotted in the form of a correlogram to describe the relation as a function of distance. The Moran's I is purely a global pattern detector informing whether clustering, dispersion or random spatial distributions exist, and therefore was favoured over the General G test which only detects spatial clustering. If spatial structuring is deemed to be absent or minimalistic at a global level, traditional regression methods are recommended for conducting a dose-response evaluation. Where linear statistical association is observed between dependent and independent variables in stage one an OLS model may prove favourable.

If spatial structuring is recorded at a global level, this may be handled in two ways at stage three of this modelling strategy; the first of which involves the application of spatial regression methods of a global nature. These models supplement the OLS and link-function (GLM) models with an additional term that incorporates the spatial autocorrelation structure of a given dataset to uphold the assumption that independent observations exist. The spatial lag model assumes that autocorrelation is only present in the dependent variable, addressed through the inclusion of an n^{th} -order neighbourhood weight matrix as an independent parameter. This method is most appropriate when the focus is on the assessment of the existence and strength of spatial interaction. In contrast, the spatial error model assumes that regression errors are spatially dependent and that the included explanatory variables do not fully explain spatial autocorrelation. Here, a residual spatial matrix at the n^{th} -order neighbourhood acts to improve the precision of regression coefficients. However a more flexible way of accounting for spatial autocorrelation at a global model may be achieved via multilevel models. Rather than being restricted by an n^{th} -order approach, weight matrixes are classified by potentially more appropriate user defined geographical distributions (Jephcote & Chen 2013). Whilst GAM's have also been demonstrated to offer a flexible approach to incorporating the influence of a spatial surface (Vieira et al 2005), this project decided against the use of such a modelling procedure in light of the previously documented time-series controversies (see chapter 1).

The second block of analysis conducted at stage 3 involves the use of local indicators of spatial association, which allow the user to geographically locate, distinguish and match (between variables) individual processes of a spatially dependent nature. The two tests of interest here appear in the form of the Local Moran's I and Getis-Ord G_i^* , and are deconstructions of their global counterparts. For each observational point, an individual test is

conducted which measures the magnitude of similarity or dissimilarity to its neighbours. Here, the local mean used within the Local Moran's I test only includes neighbouring features, whereas the local mean for Getis-Ord G_i^* includes neighbouring features in addition to the locale in question. As such the G_i^* statistic may only be used to detect geographically unique hot and cold spots, whereas the Local Moran's I is capable of identifying such clusters as well as high/low spatially outlying features. Still, the computational differences of the G_i^* statistic serve for a suitable cross-validatory measure in the detection of local patterns (Anselin 1995, Burra et al 2002).

Stage four of this modelling strategy offers two different approaches of analysis, to handle datasets in the presence of local spatial structures. Here, a dose-response evaluation involves a recently developed local regression technique, known as GWR, which generates a separate regression equation for each feature analysed in a sample dataset as a means of addressing spatial variation. Beneficially this allows for a much more flexible capture of numerical and geographical trends, in the presence of non-stationarity. To complement this analysis it is also possible to conduct a distance-response evaluation, viewed as an intermediate between pattern and dose-response evaluators of a local nature. Under this type of modelling falls the widely utilised buffer analysis procedure, which maps coordinate referenced self/clinician diagnosed ailments, to obtain the level of respiratory risk associated at incremental distances from a major roadway after adjusting for social covariates (see chapter 2.3.2). However, such an analysis is unfeasible across Leicester UA, where respiratory case files are geocoded at a census unit level, to preserve patient confidentiality.

Still, it is possible to achieve an alternative approach within this block of statistics, known as a boundary analysis. This series of statistical measures share closer ties with pattern detection approaches, and as such, do not define a level of risk within each zone. Rather they provide a singular unbiased critical distance threshold (not restricted to a predefined locale), which may be used to uniquely assess both environmental and social factors. Of further benefit is the manner in which these measures can explore how health and theoretical boundaries constructed from multiple socio-environmental influences interact. Subsequently, it becomes possible to locate and measure in terms of proximity how individual stimulus-responses interrelate, to cause a health response of greater magnitude (Pearce et al's 2010). As such, boundary analysis approaches are a means of validating how the different stimulus-response relations defined by GWR modelling interact, under a difference branch of statistics. For ecological datasets, a combination of GWR and boundary approaches allows for a more complete image of the dataset, meeting and potentially surpassing the outputs of a traditional buffer analysis (which is not a current option).

3.6. INDICATORS OF STATISTICAL ASSOCIATION

The identification of statistical dependency refers to the presence of a relationship, in which two variables fluctuate in a proportionate manner throughout the dataset. Whilst such relations are often suspected and visually observed, correlation indices are a means of definitively quantifying the direction and strength of this covariation. The two most popular of these traditional measures consist of the Pearson's product-moment (Pearson 1985) and Spearman's rank correlation (Spearman 1904) coefficient.

3.6.1. PEARSON'S CORRELATION COEFFICIENT

The Pearson product-moment correlation coefficient (r) is a dimensionless statistical measurement of the correlation between two variables, invariant to linear transformations of either variable. Subsequently it is widely used in the sciences as a method of measuring of the strength of linear dependence between two variables. The Pearson's correlation test may provide outputs ranging from +1 to -1, with a correlation of +1 meaning that there is a perfect positive linear relationship between the variables. A value of 0 indicates there to be no significant linear relationship between the variables.

If we have a series of n measurements of X and Y written as X_i and Y_i where $i = 1, 2, \dots, n$, and sample means of X and Y denoted as \bar{X} and \bar{Y} , then the Pearson product-moment correlation coefficient (r) between X and Y is portrayed as (Rodgers & Nicewander 1988):

$$r = \frac{\sum(X_i - \bar{X})(Y_i - \bar{Y})}{\sqrt{\sum(X_i - \bar{X})^2 \sum(Y_i - \bar{Y})^2}} \quad (\text{Eq.10})$$

A t-test is required to test the significance of the correlation, where the calculated t-value is compared to a standard table for a two-tailed Students t; here the critical value for 187 observations with a 95% confidence interval is 1.97 (Mitchell 2004):

$$t = r \sqrt{(n - 2) / (1 - r^2)} \quad (\text{Eq.11})$$

3.6.2. SPEARMAN'S RANK COEFFICIENT

In contrast, the Spearman's rho coefficient is a nonparametric (distribution-free) rank statistic proposed as a measure of the strength of the association between two variables. It is a measure of a monotone association that is used when the distribution of data (non-linear)

makes Pearson's correlation coefficient misleading. It assesses how well an arbitrary monotonic function can describe the relationship between two variables, without making any assumptions about the frequency distribution of the variables. In principle, the Spearman's rho is simply a special case of Pearson's product-moment coefficient in which the data is converted to ranks prior to calculation (Mitchell 2004):

$$\text{rho} = 1 - \frac{6 \cdot \sum D^2}{n^3 - n} \quad (\text{Eq.12})$$

Here, recorded values are ranked from highest (1) to lowest (n) for each individual variable, with **D** representing the difference between a pair of rank values recorded at each feature. Like the Pearson's *r*, coefficient values range from 1 (perfect direct correlation) to -1 (perfect inverse correlation), with significance recorded by a t-test output.

3.7. INDICATORS OF SPATIAL ASSOCIATION

The ability to visualise spatial data beneficially allows for the quick identification of any obvious patterns, which may be classified as regular, random or clustered, with the term 'clustered' used to describe a spatial aggregation of events. Besag & Newell (1991) initially classified the different methods for analysing clusters as either specific or non-specific, with such terms later being coined by epidemiologists as 'local' and 'global' cluster analyses. Global (non-specific) clustering methods are used to assess whether clustering is apparent throughout the study region but do not identify the location of clusters, rather such methods produce a single statistic measuring the extent of general spatial association. In contrast, local (specific) methods of cluster detection define the locations, extent and nature of such spatial associations, which may vary across the study area in question.

3.7.1. GLOBAL INDICATORS OF SPATIAL ASSOCIATION: MORAN'S I

The Moran's I coefficient (Moran 1948) is a well-known test for spatial autocorrelation of aggregated data, providing the user with an estimate of the degree of spatial similarity observed among neighbouring values of a specified attribute across the entire study area. A fundamental aspect of all autocorrelation statistics is the weights matrix, which is used to define the spatial relationships of the regions so that those in close spatial proximity are given greater weight in the calculation than those that are distant.

The Moran's I coefficient of autocorrelation is similar to Pearson's correlation coefficient which measures correlation (linear dependence) between two variables X and Y, giving a value between +1 and -1 inclusive. However the Moran's I coefficient differs from this test through quantifying the similarity of an outcome variable among areas that are defined as spatially related (Moran 1948). The Moran's I statistic is given by (Mitchell 2004):

$$I = \frac{n \sum_i \sum_j W_{ij} (X_i - \bar{X})(X_j - \bar{X})}{(\sum_i \sum_j W_{ij}) \sum_i (X_i - \bar{X})^2} \quad \text{(Eq.13)}$$

Here the mean of the variable \bar{X} is subtracted from the value of the target feature X_i and the value of its neighbour X_j , the differences are multiplied by each other and by the weight of that pair W_{ij} , where they are added to form the sum of all features. A value divided by the variance σ^2 defined as $[\sum_i (X_i - \bar{X})^2 / n]$ multiplied by the sum of the weights.

The Moran's I coefficient is approximately normally distributed and has an expected value of $-1 / (N - 1)$, when no correlation exists between neighbouring values. Consequently the expected value of the coefficient is expected to approach 0 as the number of spatial units (N) increases. Although the Moran's I coefficient generally lies between +1 and -1, it is not bound by these limits unlike Pearson's correlation coefficient. A Moran's I output of zero indicates the null hypothesis of no clustering to be true, whereas a positive Moran's I result signals that a positive spatial autocorrelation exists within the dataset i.e. clustering of areas of similar attribute high or low values. In contrast a negative coefficient indicates negative spatial autocorrelation resulting in the neighbouring areas tending to demonstrate characteristically dissimilar attribute values.

Several disadvantages of such autocorrelation tests are identified by Moran (1948), including, that the test assumes that the population at risk is evenly distributed within the study area and that the correlation or covariance is the same in all directions (isotropic). Such factors are of particular concern especially when observing clusters across both geographically large and demographically diverse areas, where local effects may be obscured. Such disadvantages may be overcome through the use of local statistics which scan the entire dataset measuring dependence across user specified portions of the study area, thus aiding in the identification of a clusters locality, dimension and intensity.

3.7.2. LOCAL INDICATORS OF SPATIAL ASSOCIATION: ANSELIN'S LOCAL MORAN'S I

A localised version of the Moran's I test was initially devised by Anselin (1995) in order to detect local levels of spatial autocorrelation within aggregated data. This was achieved through separating the Moran's I statistic, so that it could review individual contributions for each area within a selected study region. Anselin's Local Moran I statistic is one of several developed Local Indicators of Spatial Association (LISA), and is specifically used to detect spatial patterns that compare (cluster) or deviate (spatial outlier) extremely from neighbouring elements (Anselin 1995):

$$I_i = \frac{(X_i - \bar{X})}{\sigma^2} \cdot \sum_j^n W_{ij} (X_j - \bar{X})$$

$$I_i = Z_i \cdot \sum_j^n W_{ij} Z_j \quad j \neq i$$

(Eq.14)

Where, Z_i is the z-score value for the attributed of interest at the ego location i , and Z_j is the z-score value for the attributed of interest at neighbouring observations j . For the Leicester UA datasets, spatial weights indicating the strength of connection between the paired LLSOA features of i and j are represented by W_{ij} . Only immediate adjacent geographic features were defined to have spatial weighting which were standardised by neighbour count, thus preventing individual areal units from having a greater impact than any other neighbouring features. Univariate Local Moran's I statistics were conducted on the annual rates of children's respiratory hospital admissions and potentially influential social-ethnic and environmental factors of interest. Tests for spatial patterning across two variables within the same time period were also conducted though the Bivariate Local Moran's I statistic, where the ego (location i) is defined by variable 1 and its neighbours (locations j) by variable 2.

The significance of the Local Moran statistic values were obtained through 9999 Monte Carlo conditional randomisations, with the resultant p-values experiencing an adjustment in the form of the Simes correction (Simes 1986):

$$p'_i = (n + 1 - a)p_i$$

(Eq.15)

Here n is the number of p-values being considered (the number of neighbours and the central location p_i), and a is the index (lowest value starting at 1) indicating the location in the sorted vector of the p-values for a location and its neighbours. The Simes adjustment is a less conservative form of the Bonferroni correction, and is a procedure often used when

conducting tests of multiple significance to minimise the extent of Type I (false positives) and Type II errors (false negatives).

In a preliminary exploration of conflict patterns in Africa, Anselin (1995) fully verified the effectiveness of the local Moran test, through the identification of the same four countries recognised by another popular local clustering technique, in the form Getis-Ord G_i^* statistic. Common conception proclaims that the two techniques are best used in a complementary fashion, for validatory purposes.

3.7.3. LOCAL INDICATORS OF SPATIAL ASSOCIATION: GETIS-ORD LOCAL G_i^*

The Getis-Ord local statistics are an indicator of local clustering that measure the concentration of a spatially distributed attribute variable (Getis & Ord 1992, Ord & Getis 1995). This is of particular contrast to the previously mentioned Moran's I and Anselin's local I statistics, both of which solely measure the correlation between the characteristics of interest in adjacent areas, without investigating the clusters magnitude.

The Getis and Ord local statistics are additive in nature, focusing on the sum of the j values in the vicinity of i in the form of two statistics, thus allowing the researchers to choose hypotheses based on proximity (G_i) or on clustering (G_i^*). The Getis-Ord G_i^* is written as (Ord & Getis 1995):

$$G_i^* = \frac{\sum_{j=1}^n W_{ij}X_j - W_i^*\bar{X}}{\sigma \cdot \sqrt{(n S_{1i}^* - W_i^{*2}) / (n - 1)}} \quad \text{for all } j \quad \text{(Eq.16)}$$

Where the sum of the weights including that of the ego W_i^* and the sum of the squared weights including ego S_{1i}^* are defined by (Ord & Getis 1995):

$$W_i^* = W_i + W_{ii} \quad \text{and} \quad S_{1i}^* = \sum_{j=1}^n W_{ij}^2 \quad \text{for all } j \quad \text{(Eq.17)}$$

Akin to the Local Moran statistic, the output of the G_i^* function is presented in the form of a z-score for each feature based on the process of Randomisation Null Hypothesis computation. The subsequent z-score values are associated with a normal distribution thus attaching them with levels of significance and confidence. As previously, significance was obtained through 9999 Monte Carlo conditional randomisations, with the resultant p-values experiencing an adjustment in the form of the Simes correction (Simes 1986).

To summarise, when given a set of weighted features, the G_i^* statistic identifies the presence of statistically significant hot and cold spots inclusive of the target locale. In contrast, the Local Moran's I statistic is capable of providing an image of greater detail, recognising not only hot and cold spots, but also the presence of spatial outliers. Under the G_i^* statistic it is conceivable that a high valued target feature can show up as a hot-spot even when surrounded by low values. Still one drawback of the local Moran's analysis is that the focal point of prospective elements of clustering are not accounted for. Despite its simplistic spatial description, the G_i^* statistic has been successfully implemented to predict 86.1% of all new landslide occurrences (Chu et al 2009) and work well in conjunction with the Global Moran's I to analyse health care hotspots (Tsai et al 2009), across Taiwan. As such validation of more advanced pattern recognition techniques is recommended through the use of the G_i^* statistic.

3.8. SPATIAL REGRESSION ANALYSIS

Ordinary Least Squares (OLS) regression methods are traditionally used to define the variation of a dependant variable in terms of a fixed response gradient for each individual explanatory variable:

$$y = \beta_0 + \beta_1 X_1 + \beta_2 X_2 + \dots + \beta_N X_N + \varepsilon \quad (\text{Eq.18})$$

Where y is the dependent variable (Hospital Admissions), $X_1, X_2 \dots X_N$ are the independent variables (TPM_{10} , Carstairs Index, etc.), ε is the residual value, β_0 is the intercept, and $\beta_1, \beta_2 \dots \beta_N$ are regression coefficients relating to their respective independent variables. As such, OLS models describe average (or global) parameter estimates, which are assumed to operate uniformly across space. Yet, the assumption of a uniform modelled relationship over space would be quite misleading, if such relationships being are intrinsically different across space, as is the case in Leicester. Following on from the literature review, presented in chapter 2, it was decided that the spatial elements within the study area of Leicester UA should be addressed and/or accounted from through the use of global (Multilevel) and local (GWR) spatial regression procedures.

3.8.1. MULTILEVEL MODELLING

Global regression procedures, in the form of multilevel modelling were initially conducted in order to explore the chief factors influencing children's respiratory health across Leicester. Multilevel regression models are a class of statistical models developed for the analysis of data

structures with nested (hierarchical) sources of variability. Observations made within a cluster are usually assumed to be dependent, whereas clusters themselves are assumed to be independent of one another. The general idea of a multilevel model is that this hierarchy is taken into account, through the addition of random affects to traditional regression models, so as to define the covariance structure of the data. In essence, the random effects remove unmeasurable population subset influences (white noise) away from the fixed parameter estimates.

To address issues of spatial nonstationarity, Leicester's 187 Lower Level Super Output Areas (LLSOA's) (*i*) were first nested into quantiles (*j*) of J00-99 children's hospital admissions (dependent variable). This was conducted under the assumption that LLSOA observations experiencing similar hospital rates contain populations with comparable disease tolerance levels. In this two-level response model the unmeasured disease tolerance effect was included through the addition of a second intercept which differed only across each quantile. The linear random intercept multilevel model of Leicester is defined as:

$$\mathbf{Y}_{ij} = \mathbf{X}_{ij} * \boldsymbol{\beta} + \mathbf{Z}_{ij} * \mathbf{b}_j + \boldsymbol{\varepsilon}_{ij} \quad \begin{array}{l} \mathbf{i} = \text{LLSOA Observations} \quad (\text{Level 1}) \\ \mathbf{j} = \text{Quantile Data Cluster} \quad (\text{Level 2}) \end{array} \quad (\text{Eq.19})$$

Where, **Y** represents the dependent variable recorded as children's respiratory hospital admissions, **X₀, X₁, X₂...X_N** are the fixed independent variables (e.g. Intercept, Carstairs Index, TPM₁₀ etc.) with correspondent fixed effects parameter estimates **β₀, β₁, β₂...β_N**. The random effects occurring at the upper community level are described through the variable **Z**, which has a random effect parameter estimate **b**. In this instance a single random effect is included in the form of a random intercept, which is allowed to change across quantiles, in effect accounting for the influence of white noise across similar outcome values. Residual values of the complete model are recorded as **ε**.

It is assumed that **b** and **ε** are uncorrelated random variables with zero means and covariance matrices **G** and **R**, respectively. Thus, the expectation and variance **V** of the observation vector **Y** are (Brown & Prescott 2006):

$$\begin{aligned} E[\mathbf{Y}] &= \mathbf{X}\boldsymbol{\beta} \\ \text{Var}[\mathbf{Y}] &= \mathbf{V} = \mathbf{ZGZ}^T + \mathbf{R} \end{aligned} \quad (\text{Eq.20})$$

Unbiased estimates of variance and covariance parameters were obtained through the restricted maximum likelihood (REML) estimation procedure (Brown & Prescott 2006, Section

2.2.1, p47), optimised through iterations of the Newton-Raphson expectation-maximisation algorithm. Upon defining suitable variance and covariance parameters it is possible to obtain $\hat{\beta}$ which is the 'best linear unbiased estimator' of β , and \hat{b} the 'best linear unbiased predictor' of b (Brown & Prescott 2006):

$$\begin{aligned}\hat{\beta} &= (\mathbf{X}^T \mathbf{V}^{-1} \mathbf{X})^{-1} \mathbf{X}^T \mathbf{V}^{-1} \mathbf{Y} \\ \hat{b} &= \mathbf{GZ}^T \mathbf{V}^{-1} (\mathbf{Y} - \mathbf{X} \hat{\beta})\end{aligned}\tag{Eq.21}$$

Whilst multilevel methods are traditionally used for predictive purposes, their application within this project is to be viewed more as a descriptive measure; through the placement of greater focus on respiratory outcomes restricted to the study period, rather than that of the explanatory influences. As such this global modelling strategy of incorporating broad yet localised spatial structures based on respiratory outcomes is considered conservative in nature. In this respect, where strong socio-environmental signals prevail, one may conclude their presence as a considerable driving force of health. The outcomes of which, are to be fully quantified through the use of local regression measures.

3.8.2. GEOGRAPHICALLY WEIGHTED REGRESSION (GWR)

As previously discussed, OLS models describe average (or global) parameter estimates, which are assumed to operate uniformly across space. However in reality non-stationary relationships are likely to exist as a consequence of: (I) Sampling variations within the data; (II) Contextual issues that produce spatially differing responses to the same stimuli; and or (III) Model misspecification (Fotheringham et al 1998). Such datasets thus pose a significant dilemma for traditional regression models, which assume observations to be independent of one another. Thus the nature of a model must alter over space to reflect the structure within the data.

Rather than calibrating a single regression equation (Eq.18), GWR generates individual regression equations for each of Leicester UA's 187 LLSOA census units, applying different weightings for the observations contained within the dataset (Fotheringham et al 1998):

$$y_i = \beta_0(\mathbf{u}_i) + \beta_1(\mathbf{u}_i)X_{i1} + \beta_2(\mathbf{u}_i)X_{i2} + \dots + \beta_N(\mathbf{u}_i)X_{iN} + \varepsilon_i(\mathbf{u}_i)\tag{Eq.22}$$

Where (\mathbf{u}_i) represents the location of observation i , and thus $\beta_1(\mathbf{u}_i)$ indicates that the regression coefficient β_1 defines a relationship specific to location i . The weight assigned to all other observations is based on a distance decay function, centred on the centroid of LLSOA

observation i . The calculation of the GWR model coefficients may be expressed as (Fotheringham et al 1998):

$$\beta(\mathbf{u}_i) = (\mathbf{X}^T \mathbf{W}(\mathbf{u}_i) \mathbf{X})^{-1} \mathbf{X}^T \mathbf{W}(\mathbf{u}_i) \mathbf{y} \quad (\text{Eq.23})$$

Where the superscript T denotes the transposition of a matrix, and $\mathbf{W}(\mathbf{u}_i)$ is the weight to be applied to locality i , derived from a proximity based geographical weight matrix of locality i and its neighbouring elements $J_{1..N}$. Through placing higher weightings based on proximity, GWR clearly adheres to the first law of geography, which states “everything is related to everything else, but near things are more related than distant things” (Tobler 1970, p236). Under this premise, sampling variations, issues of independence between observations, and response variations are likely to be addressed.

The distance decay function, which may take several mathematical forms, is modified by a bandwidth setting, allocating a distance at which the weight assigned rapidly approaches zero (Appendix B10). The GWR model weighting is computed through the use of either a fixed or adaptive kernels setting the value for any observation whose distance is greater than the bandwidth to zero, thus excluding them from the local calibration. The bi-square weighting of observation i and its neighbour j can be expressed as a function of the distance \mathbf{d} between localities and the applied bandwidth \mathbf{b} (Fotheringham et al 2002):

$$\mathbf{W}_{ij} = \begin{cases} [1 - (\mathbf{d}_{ij}/\mathbf{b})^2]^2 & \text{if } \mathbf{d}_{ij} < \mathbf{b} \\ = 0 & \text{if } \mathbf{d}_{ij} \geq \mathbf{b} \end{cases} \quad (\text{Eq.24})$$

One may argue that the kernels should always be allowed to vary spatially. For instance, if a fixed spatial kernel is applied where data is sparse, locally calibrated models will provide parameter estimate surfaces which are under-smoothed, defined by high levels of local variation and large standard errors. Whereas across densely sampled areas, fixed spatial kernels may cause over-smoothing, to provide a series of parameters similar in value, once more failing to realistically incorporate spatial elements of particular study areas (see Appendix B10).

GWR models exploring the respiratory subsets of interest were created within SpaceStat 3.5.6, using bi-square adaptive bandwidths, to allow for optimal weighting adjustment in accordance to the density of available data at each regression location. Models were constructed using various adaptive bandwidth schemes, defined by the consistent inclusion of the nearest 20, 40, 60 or N nearest neighbours in each local model. Model performance was identified to improve through the use of adaptive kernels, an issue which has

been observed in other studies dealing with census areas of variable size (Mennis 2006, Gilbert and Chakraborty 2011). Localised GWR parameter estimates and their correspondent p-value outputs were subsequently used to explore the intra-urban relationship between children's respiratory hospitalisations caused by residential exposure to traffic emissions, after adjustment to various social factors.

Traditionally GWR regression parameter standard errors are calculated using the global error variance, defined as the Residual Sum-of-Squares (RSS) at each of the target points (Fotheringham et al 2002). However, SpaceStat uses the local variance defined as the RSS from the regression calculation at the source points to calculate local standard errors, and hence local p-values for the regression parameters. It is believed that this approach should more accurately reflect the degree of non-stationarity encapsulated in the geographical weighted calculation.

Primary validation of GWR models was therefore achieved by conducting three ANOVA based generalised degrees of freedom F-tests using the 'spgwr 0.6-14' [R] package. The FBC-F derived by Fotheringham et al (2002) uses the effective degrees of freedom derived from the models RSS to calculate an approximate likelihood ratio test to compare GWR and OLS model abilities of reproducing the original dataset. The F-value is obtained via the OLS-RSS/GWR-RSS ratio with (df1, df2) denoting the respective OLS and GWR models degrees of freedom.

$$\begin{aligned} \text{OLS RSS} &= (\mathbf{n} - \mathbf{k}) \sigma^2 \\ \text{GWR RSS} &= (\mathbf{n} - [\mathbf{2tr}(\mathbf{S}) - \mathbf{tr}(\mathbf{S}^T\mathbf{S})]) \sigma^2 \end{aligned} \tag{Eq.25}$$

The effective number of parameters in GWR is given by $\mathbf{2tr}(\mathbf{S}) - \mathbf{tr}(\mathbf{S}^T\mathbf{S})$, where the hat matrix \mathbf{S} describes the influence of each observed \mathbf{y} on each fitted $\hat{\mathbf{y}}$ of the GWR model through the notation: $\hat{\mathbf{y}} = \mathbf{S}\mathbf{y}$. The effective number of parameters in a GWR is often not an integer but varies between the traditionally defined number of parameters \mathbf{k} (when the bandwidth tends to infinity) and \mathbf{n} (when the bandwidth tends to zero); In many cases, $\mathbf{tr}(\mathbf{S})$ is very close to $\mathbf{tr}(\mathbf{S}^T\mathbf{S})$ so an approximate value for the effective number of parameters is $\mathbf{tr}(\mathbf{S})$ (Fotheringham et al 2002).

Models received additional validated through the use of Leung et al's (2000) LMZ-F1 and LMZ-F2 tests, which apply different techniques to obtain the F-ratio and GWR effective degrees of freedom. One should also note that 2-3 years of children's respiratory admissions were omitted from the calculation of average annual admission rates experienced across the 10-year period. This additional validation step was incorporated to take account of R^2 cross-

validation, and is therefore considered a more comprehensive measure of performance across spatial models.

3.9. BOUNDARY ANALYSIS

3.9.1. BOUNDARY DETECTION: CRISP AREAL WOMBLING

Areal wombling is interested in the exploration of whether two adjacent census areas with a common border, defined as a Candidate Boundary Element (CBE), experience dramatically different observed response values. Methods for delineating boundaries of difference are collectively coined as wombling techniques, after Womble's (1951) initial quantification of spatial surface gradients in raster structures. Substantial differences between values of neighbouring localities are thus thought to denote the edge of homogeneous areas, such as the introduction of a pollution plume or group of persons with heightened susceptibility to health issues. In terms of health outcomes, boundaries are of particular importance in understanding whether the presence or disappearance of extreme values in influential socio-environmental phenomenon(s) may drastically increase levels of risk. Magnitudes of difference are derived through the assignment of a Boundary Likelihood Value (BLV) to each CBE using a Euclidean dissimilarity metric:

$$D_{ij} = (Z_i - Z_j)^2 \quad (\text{Eq. 26})$$

Where D_{ij} records the magnitude of dissimilarity between the two LLSOA census units, and Z is a measurement of the attribute value of interest at the LLSOA census units i and j . It should be noted that the raw dataset values recorded at each LLSOA observation are normalised on a scale of 0-1, prior to calculating the Squared Euclidean dissimilarities of the applicable CBE's. As a result, levels of dissimilarity for a univariate boundary analysis may never exceed a value of 1, with such a procedure allowing for a direct comparison of dissimilarity strength across multiple variables.

CBE's form an official boundary, known as a Boundary Element (BE), when their BLV's exceed established thresholds. A stringent BLV cut-off value was selected in accordance to prior wombling studies which traditionally define the upper 5th percentile as representing boundaries (Barbujani et al 1989, Fortin & Drapeau 1995). Some recent studies of boundary analysis have set BLV thresholds at the 20th percentile (Hall 2008, Jacquez & Greiling 2003), which would propose the detected boundaries within this study to be conservative in nature.

Univariate crisp polygon wombling techniques as detailed above were applied to detect boundaries across children’s respiratory hospital admissions, traffic emissions and to other individual social variables within BoundarySeer 1.3.13. Multivariate boundaries were obtained through equally weighting the variables dissimilarity metric surface-gradients. Within the Leicester UA dataset approximately 25 out of the 508 CBE’s were defined as BE’s for each individual or set of socio-environmental variables examined.

3.9.2. OVERLAP ANALYSIS

Overlap statistics were subsequently employed to establish whether the boundaries defined by areal wombling across different variables, occupied similar localities at a level greater than what would occur by chance. The extent and likelihood of boundary overlap was evaluated via the application of four overlap statistics based on the average minimum distance from boundaries in one variable to the nearest boundary in the other variable of interest (Jacquez 1995, Jacquez & Greiling 2003).

Assume two variables G (i.e. TPM₁₀ emissions) and H (i.e. J00-99 hospital admission rates) with **B_G** and **B_H** representing their corresponding boundary elements (BE). As detailed earlier BE’s within this study were classified to only include the top 5% of candidate boundary elements (CBE’s), representing the highest magnitudes of dissimilarity between two directly adjacent LLSOA’s. Upon defining the existence and location of the total boundary locations **N_{B_G}** and **N_{B_H}**, it is then possible to construct a distance matrix, with elements **d_{ij}** representing the distances between location i in the boundary for location G and location j in the boundary for variable H. Within the constructed distance matrix **min(d_{i.})** defines the smallest distance in column j of the matrix and **min(d_{.j})** represents the smallest distance in row i of the matrix. The overlap statistics may therefore be described as (Jacquez 1995):

$$\begin{aligned}
 O_S &= \text{card}(\mathbf{B}_G \cap \mathbf{B}_H) \\
 O_G &= \frac{\sum_{i=1}^{N_{B_G}} \min(\mathbf{d}_{i.})}{N_{B_G}} \\
 O_H &= \frac{\sum_{j=1}^{N_{B_H}} \min(\mathbf{d}_{.j})}{N_{B_H}} \\
 O_{GH} &= \frac{\sum_{i=1}^{N_{B_G}} \min(\mathbf{d}_{i.}) + \sum_{j=1}^{N_{B_H}} \min(\mathbf{d}_{.j})}{N_{B_G} + N_{B_H}}
 \end{aligned}$$

(Eq. 27-30)

The overlap statistic O_s measures the frequency of which boundaries within two datasets intersect, and is thus defined as the number (cardinality count) of elements that are located within in both boundary sets G and H . O_G is the mean distance from any location in the boundaries for G to the nearest location in the boundaries for H . O_H is the mean distance from any location in the boundaries for H to the nearest location in the boundaries for G . O_{GH} is the average distance from the location in the G or H boundaries to the nearest location in the other (G or H as appropriate) boundary.

The p-values of the four overlap statistics were defined through comparing observed values to their null distributions as generated by 499 Monte Carlo randomisations. Upper and lower p-values provide a sense of how extreme the observed values of the overlap statistics are compared to the reference distribution of values obtained by randomisation (Jacquez & Greiling 2003). The combined runtime of the boundary creation process and these four statistics, testing the strength of overlap between each pair of variables G and H , totalled approximately 16.5 hours on a PC with the following specifications: Windows XP, Intel Pentium Dual Core 1.8GHZ Processor, 4GB DDR2 Ram.

**ENVIRONMENTAL INJUSTICES OF CHILDREN'S RESIDENTIAL EXPOSURE
TO ROAD-TRANSPORT EMISSIONS: LEICESTER UA 2000-09**

OVERVIEW

This chapter concentrates on the first stage of this research project, which examines the beneficial impacts and/or burdens placed on a child's overall respiratory health by influential socio-environmental factors. Global and Local Indicators of Spatial Autocorrelation (GISA, LISA) statistically describe and illustrate the spatial nature of such socio-environmental influences and average annual hospital admission rates associated with a respiratory condition (ICD-10: J00-99) experienced by children residing within Leicester UA from 2000-09. Spatially appropriate modelling procedures, accounting for underlying geographical structures within the datasets, were then applied to define the extent to which socio-environmental variables of interest individually influenced respiratory health during childhood at global and local scales. This chapter covers objectives 1, 2, 3 and 4 of this project outlined in Chapter 1.

4.1. INTRODUCTION

The significant contribution of road-transport to air pollution within the urban arena is widely acknowledged, and traditionally explored in relation to health outcomes across a temporal scale. However, the structure of the urban environment is also of importance in dictating the existence of extremely variable traffic pollutant levels, which often tend to be linked with social disparities. Nevertheless 'Environmental Justice' studies have rarely tackled the adverse health implications of exposures from mobile sources (Chakraborty 2009), or have applied statistical techniques that are appropriate for such spatial data (Mennis & Jordan 2005, Chakraborty 2009, Gilbert & Chakraborty 2011).

Childhood is a critical period for the development and maturation of the delicate spongy organs of the cardiorespiratory system, which are particularly susceptible to the absorption of external environmental agents experienced within the urban arena. Compared to adults, a child's lung surface area is also considerably larger in relation to their body mass, with children potentially breathing up to 50% more air per kilogram of body weight (Schwartz 2004). Another factor that makes children more sensible to air pollution by comparison to adults is their tendency to spend longer periods outside, especially in evenings, over the summer months, and to be highly active during these periods (Cooper et al 2010, Steele et al 2010).

An assessment of residential exposures to traffic pollutants and 8-year lung development within Californian children using proximity to major road links as a surrogate to exposure, identified a decline in FVC of -63ml in residents $\leq 500\text{m}$ from freeways, diminishing to -19ml at distances of 1000-1500m (Gauderman et al 2007). Similar studies have also indicated self-reported respiratory conditions to increase among children and adolescents residing along streets with high road-transport activity (Duhme et al 1996, Oosterlee et al 1996). While surrogate measures have been related to hefty health effects, it should still be considered that such techniques are highly prone to exposure misclassification. Such issues may be mitigated through Geographic Information Systems (GIS), which offer a means of estimating personal exposures to traffic pollutants across vast populations (Kunzli et al 2000, Gehring et al 2002).

Increasingly, it has come to the attention of researchers and policy makers that the distribution of exposure to air pollution is not equitable, but this inequity has until recently received little formal epidemiologic attention (Naess et al 2007, p686). Traditionally, epidemiological based studies of air pollution have treated socioeconomic positioning as a confounding influence, with relatively few studies looking carefully at how these factors interact with one another, specifically with relation to mobile pollutant sources (Kingham et al

2007, Tonne et al 2008). The importance of such socio-environmental interactions are detailed within Environmental Justice (EJ) research, which consistently report the 'double-burden' of deprivation and air pollutant exposure as a key explanatory factor in defining health disparities (Crouse et al 2009, Kingham et al 2007, Naess et al 2007, Wheeler & Ben-Shlomo 2005). Enhancing upon this concept, Pearce et al 2010, describe area level health across UK CAS Wards to degrade with measurements of multiple environmental disamenities, an effect most pronounced within severely deprived localities. Such findings act to highlight the importance of the physical environment in shaping health, with the recommendation of future research considering this 'triple jeopardy' of social, health and environmental inequalities (Pearce et al 2010).

Traditionally EJ research has faced a plethora of challenges in causally associating environmental pollutants with adverse health outcomes, yielded through the absence of standardised assessment techniques and a tendency of measuring exposure via proximity to source rather than through actual pollutant distributions. Furthermore, conventional multivariate regression techniques are unable to account for non-stationary relationships, and are therefore prone to obscure local variations of environmental equity. This is of particular concern, when considering that EJ is an explicitly spatial problem, concerning geographic elements rarely distributed in a uniform manner (Gilbert & Chakraborty 2011).

In a distinguished EJ analysis on the health risks from automobiles, Chakraborty (2009) presents the application of global regression models that can account for issues of spatial dependence, through the addition of a singular spatial lag or error component. While ethnic differences were identified as a persistent explanatory role in the distribution of health risks, relationships between socioeconomic statuses appeared complex in nature, highlighting the need for consideration of spatial autocorrelation in future environmental equity studies. In addition, the implementation of a singular spatial component may capture contextual factors but it is more likely that the generalisation of such elements will inherently under-report localised variations within the dataset, specific to individual variables across a defined area. It is recommended that such issues be addressed through the application of locally weighted models which directly assign weight structures to the individual variables of interest, therefore allowing for their relationships to independently alter where elements of non-stationarity are observed. Logic would therefore dictate that the localised variation associated with the complex relationships between health, social and environmental factors should be explored through an underused EJ technique known as Geographically Weighted Regression (GWR). To date, Gilbert & Chakraborty (2011) present the only EJ GWR study assessing the influence of social and environmental elements on areal health, defined as the risk of cancer from

exposure to US EPA modelled TRI emissions. Thus, it would be of interest for future GWR investigations to advance such practices through applying measurements of actual health events and by exploring a wider range of cardiorespiratory conditions influenced by short-term exposures.

This chapter addresses the aforementioned research gaps, through exploring the association between residential exposures to road-transport emissions (TPM₁₀), and the incidence of children's respiratory hospital admissions within an EJ context, across Leicester from 2000-09. Unlike traditional epidemiological studies, such issues will be tackled in a spatial manner through the application of geospatial tools, thus providing a global and local analysis of Leicester's communities. Initially, 'Exploratory Spatial Data Analysis' (ESDA) techniques, consisting of global and local indicators of spatial association, will statistically evaluate the spatial arrangements of respiratory health and potentially associated socio-ethnic and environmental features. Potential relationships are then summarised at a global level through the application of multilevel models, which incorporate broad upper-level nesting structures to account for common elements of clustering found across Leicester. GWR models will then be used to locate and define local variations in complex relationships, illustrating whether multiple burdens are interrelated within specific inter-urban locations. To the authors knowledge this is the first time that GWR techniques have been applied to investigate the impacts of road-transport on health. It is anticipated that GWR model outputs will assist within the development of the first dose-response relationship between children's health and local road-transport emissions.

4.2. EXPLORATORY SPATIAL DATA ANALYSIS (ESDA)

In the 1970's, American statistician John Tukey originally formulated the concept of conducting 'Exploratory Data Analysis' (EDA) procedures as a means of statistically analysing a datasets principle characteristics, thus removing the necessity of conducting statistical modelling or formulating preconceived hypotheses (Tukey 1977). Developing upon such ideologies, 'Exploratory Spatial Data Analysis' (ESDA) represents a fundamental approach towards the statistical description of phenomena distributed within a spatial context, placing focus upon geographic arrangement and the proximity of interactions.

Location may cause two specific spatial effects of interest, coined as spatial dependence (auto-correlation) and spatial heterogeneity. Spatial dependence, illustrates the direct application of the 'First Law of Geography': in which "everything is related to everything

else, but near things are more related than distant things” (Tobler 1970, p236). From a geographic perspective, the clustering of similar elements is the rule rather than the exception, yet such formations violate the requirements of standardised statistical techniques, which assume independence amongst observations (Anselin 1993). Locational effects may also manifest in the form of spatial heterogeneity, which represents the variation of a process (non-stationarity) with respect to location. Unless a space is uniform, each location will have some degree of intrinsic uniqueness relative to its neighbours. The magnitude of spatial heterogeneity determines whether parameters estimated for the entire system may adequately describe the process at any given location.

4.2.1. CHILDREN’S RESPIRATORY HEALTH & COMMUNITY INFLUENCE

Global Indices of Spatial Association (GISA), in the form of the Global Moran’s I coefficient of spatial autocorrelation (Moran 1948), were conducted upon datasets containing respiratory hospitalisation rates and socio-environmental influences of interest across Leicester UA. Akin to the Pearson Product-Moment Correlation Coefficient test values range from 1 (clustered) to -1 (dispersed), with 0 indicating that the feature of interest has a poorly defined spatial relationship (random positioning). The magnitude of global autocorrelation was explored across several row standardised contiguity weighting schemes, the first of which placed weighting solely on first order neighbours (1 Queens Ring), with later tests placing weights up to and including fifth order observations (5 Queens Rings). To position these contiguity weights into context, Euclidean metrics observed nearest neighbour observations to be on average separated by a distance of 455m. One may approximate that this measurement represents the sequential increase in radial distance between each Queens order, weighting scheme.

Subsequently, the Global Moran’s I outputs are presented in a manner that allows one to estimate the strength of correlation between observations as a function of distance (Correlogram). This has been achieved in-order to (a) score citywide levels of autocorrelation amongst immediately neighbouring LLSOA’s; (b) define the spatial extent to which autocorrelation amongst observations occurs, if applicable; and (c) to assist in selecting appropriate spatial weighting schemes for the proceeding ‘Local Indicators of Spatial Association’ (LISA) analysis.

A Global Moran's I value of 0.39 ($P < 0.001$) was recorded for children’s respiratory hospitalisation rates (ICD-10: J00-99) during 2000-09 under a first order weighting scheme, identifying moderate citywide levels of spatial correlation between directly adjacent LLSOA communities. Test values reveal comparable children’s respiratory hospitalisation rates to occur at a fairly localised scale (Figure 4.1), with noticeable clustering appeared to exist up to

second-order LLSOA communities, recorded at a far weaker level ($I=0.14$, $P<0.001$). As previously mentioned in Chapter 3, test significance was evaluated via 9999 Monte Carlo permutations, to produce a comparable reference distribution from which a pseudo significance level is computed. For a significance level of 0.05, the derived Z-Score would have to be less than -1.96 (dispersed) or greater than 1.96 (clustered). Global Moran's Z-Score's for children's respiratory admissions confirms the existence of significant spatial clustering to exist, when placing weighting on observations separated by a second order distance or lower (Figure 4.1). From this combined information, children's respiratory cases are shown to be location specific within Leicester UA, with their radius of spatial dependence following a rapid exponentially decaying relationship across a distance of approximately <910m.

It would appear that community based measurements of healthy lifestyle choices share this theme of spatial dependence, albeit at a far greater magnitude. Levels of smoking prevalence in adults, symbolising the likelihood of a child experiencing frequent passive smoking events, recorded strong spatial dataset clusters at first order observations ($I=0.65$, $P<0.001$) which gradually decline but maintain a degree of spatial dependence even across fifth order LLSOA neighbours ($I=0.22$, $P<0.001$). Although levels of correlation are relatively low amongst fifth order observations, Monte Carlo simulations dictate that the likelihood of such relations to occur by chance is negligible. Thus, a broad community influence appears to determine the residential uptake and exposure to the effects of passive smoking. Levels of adulthood obesity, used to record a child's likelihood of receiving a balanced diet, appear to show strong spatial trends across first order LLSOA neighbours ($I=0.75$, $P<0.001$), with spatial dependence declining in a linear manner reaching relative insignificance after third order observations (radius of 1365m). Still, it should be noted that elements of clustering are of significance across all of the spatial scales explored (Figure 4.1).

In terms of socio-environmental influences of interest, PM_{10} road-transport emissions (TPM_{10}) and levels of 'White British' children, once more show strong levels of spatial dependency deteriorating moderately with distance in a linear fashion. Under a first order weighting scheme TPM_{10} emissions and levels of 'White British' children produce correspondent Global Moran's I values of 0.83 and 0.82 ($P<0.001$), which respectively decline to values of 0.28 and 0.40 when exploring levels of spatial autocorrelation across LLSOA observations separated up to and including a fourth order distance ($P<0.001$). Levels of deprivation, scored by the Carstairs Index of Leicester, show signs of moderate spatial dependency across immediately neighbouring observations ($I=0.56$, $P<0.001$), with the magnitude of correlation mildly decaying in an exponential style losing substance beyond third order LLSOA's ($I=0.22$, $P<0.001$). As with obesity prevalence, the Global Moran's Z-Score's

recorded for deprivation, TPM₁₀ emissions and levels of 'White British' children recorded significantly greater clustering than expected by chance across locations of moderate proximity. Noteworthy levels of spatial dependency for TPM₁₀ emissions were observed to exist up to and including third order observations (radius $\geq 1365\text{m}$), with dependency occurring at fourth order observation for levels of 'White British' children (radius $\geq 1820\text{m}$). For deprivation, a substantial level of location dependency existed for LLSOA observations of the second order (radius $\geq 910\text{m}$).

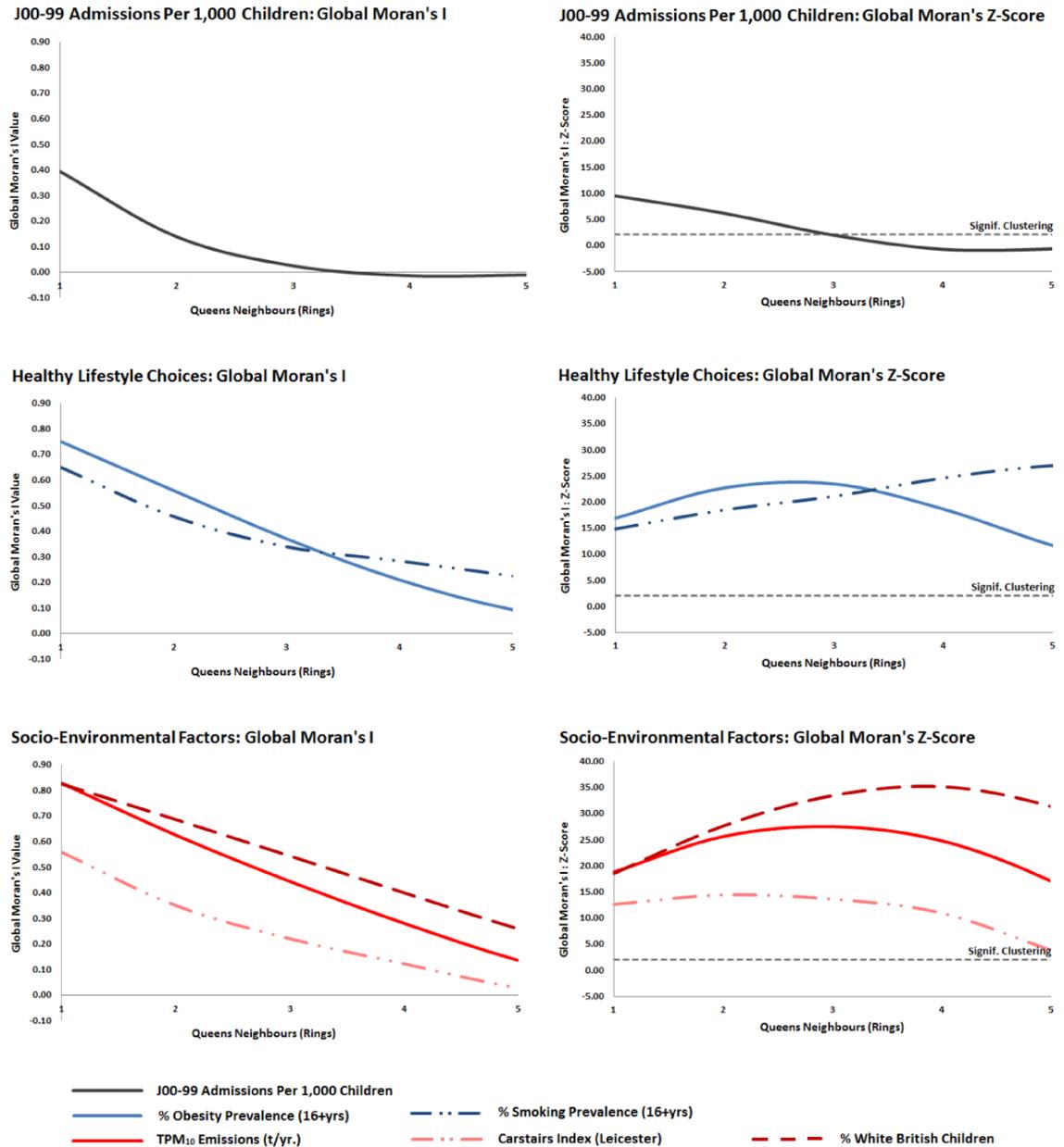


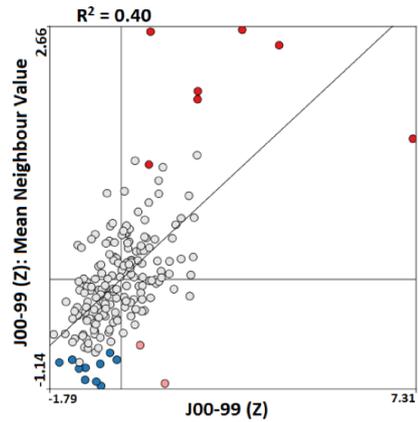
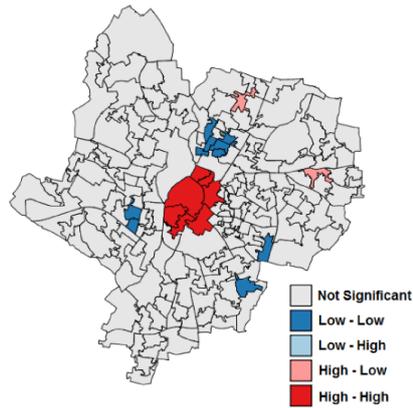
FIGURE 4.1: Spatial correlograms of Global Moran's statistical outputs, portraying the level of decay in autocorrelation between neighbouring LLSOA community's health, social and environmental influences, as a function of distance.

To summarise, Global Moran's I statistics individually identified significantly noteworthy levels of spatial autocorrelation to exist across children's respiratory admission rates, healthy lifestyle choices and socio-environmental influences. Children's J00-99 hospital admission rates portrayed moderate levels of spatial dependency with first order neighbouring LLSOA's (450m). All influential factors of interest shared a strong sense of location dependency with adjacent LLSOA communities, with moderate correlation remaining up to third order observations for measures of obesity prevalence and deprivation (1350m). Moderate correlation was found to persist within fourth order observations for levels of smoking prevalence, TPM₁₀ emissions and 'White British' Children (1820m). While such lifestyle, social and environmental influences share common characteristics across wide areas of Leicester UA, it should be recognised that a considerably greater magnitude of correlation occurs across directly adjacent LLSOA communities. It is this first order correlation that is of particular importance when investigating the presence of an association between children's respiratory hospitalisation rates, which exclusively operates across localised bands of distance.

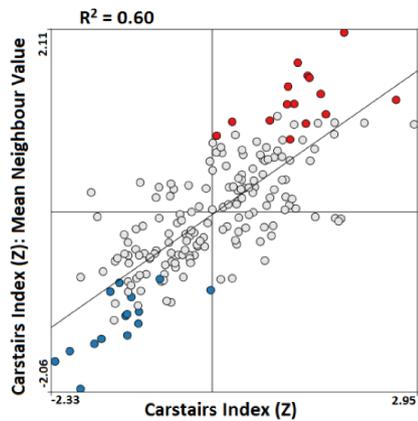
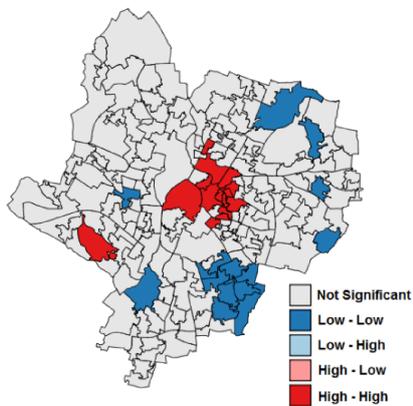
As a composite index, the Global Moran I coefficient is the measurement of overall clustering of the aforementioned data, used to evaluate the overall extent of spatial association within the study area of interest. In yielding a singular coefficient to define the entire study area, GISA's operate under the assumption of spatial homogeneity, which is a false assumption to hold when understanding that structural change across space (spatial heterogeneity) is a known feature of geographical research. Favourably, 'Local Indices of Spatial Association' (LISA) such as the Local Moran's I coefficient (Anselin 1995), dissect their global counterparts thus allowing for one to explore such previously hidden local variations. Furthermore, it is possible to observe clusters at a local level using LISA techniques where global autocorrelation was previously unreported, particularly in cases where homogeneous pockets diverge from the global trend. LISA's therefore allow for the identification of spatial outliers, in addition to locating and describing the direction of influence individual homogeneous areas exhibit in relation to surrounding observations (i.e. hot-spots or cold-spots). In essence, the Local Moran's I statistic allows for a comprehensive location specific analysis of the dataset, complementing its global counterpart and therefore providing an initial understanding of the spatial structures in play.

LISA's placing a row standardised weight solely upon first order observations were subsequently employed as part of this ESDA of respiratory hospital admissions and potentially associated social-ethnic and environmental influences of interest (Figure 4.2).

J00-99 Admissions Per 1,000 Children



Carstairs Index (Leicester)



TPM₁₀ Emissions (t/yr.)

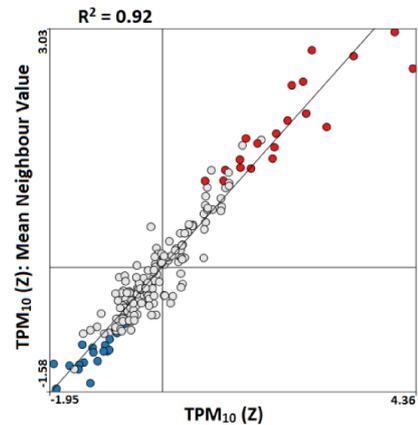
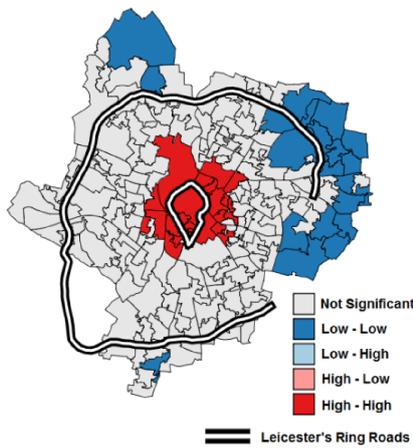


FIGURE 4.2: Local Moran's I cluster and outlier analysis of 2000-09 annual average children's respiratory admissions (ICD-10: J00-99), socioeconomic status and levels of TPM₁₀ emissions

The Local Moran's I statistical test identified the existence of a significantly high clustering of average annual respiratory hospital admissions (ICD-10: J00-99) across inner city children aged 0-15 (Figure 4.2). Observed annual J00-99 hospitalisation rates within this zone of high clustering range from 49.39 to 147.83 admissions per 1,000 children, which is considerably

higher than Leicester UAs average rate of 39.43 admissions. Notably similar patterns of clustering may be observed across yearly average output area levels of residentially experienced TPM_{10} emissions and Carstairs Index measured levels of deprivation, thus presenting the potential for a double burden of social and environmental issues collectively influencing a person's wellbeing (Figure 4.2). This highlighted area of interest contains the cities inner ring road (A594) and several key arterial roads linked to the cities outer ring road (A563) and the national road network. Residents within these respective inner city clusters were identified to on average experience an extra 0.78 tonnes/year of PM_{10} road-transport emissions, and be subject to Carstairs Index deprivation values 4.17 scores above typical citywide values.

The case for environmental equity may be further brought into question upon examining the distribution of Leicester's 'White British' ethnic group, which is identified to heavily populate the cities southern and western peripheries (Appendix C1). Significantly low levels of clustering for persons of 'White British' origin within and adjacent to the east of the city centre, present a potential scenario in which ethnic minority groups bear a disproportionate burden of environmental and social problems. Compared to Leicester's citywide ethnic demographics, it is observed that children of 'Afro-Caribbean', 'Indian', 'Other South Asian' and 'White Non-British' origins respectively constitute an additional 13.11%, 7.29%, 5.61% and 1.41% of the children's population within the designated inner city respiratory zone of concern. In contrast, across Leicester 52.91% of all children are identified as 'White-British', however this ethnic group only forms 23.13% of the designated inner city respiratory zones respective populace.

The Local Moran's I statistical test on children's respiratory admissions also identified the existence of two individual outlying LLSOA's experiencing elevated children's respiratory hospital rates, numerically distant from those at surrounding localities. Interestingly the outlier to the east of Leicester incorporates the incomplete section of Leicester's outer ring road known as the proposed 'Eastern District Distributor Road', which is yet to materialise despite the routes continued safeguarding within local transport plans (LCC 2009). At present traffic has to leave the outer ring road and join onto a single carriageway, thus causing regular congested at peak hours (Appendix C2). The location of nearby important private services in the form of the Nuffield Health Leicester Hospital may also contribute to local traffic disruption.

Similarly the northern outlier also identifies the existence of a major road junction for a primary radial corridor (A47: Melton Road) intersecting the outer ring road. This particular radial corridor contains a diverse range of retail stores and restaurants, indicative of Leicester's

multicultural heritage, that attract both locals and tourists to the area. Once again this hot-spot is observed across a section of the roadway which changes from a dual to a single carriageway as traffic heads towards the city centre, potentially designated the site as a second bottleneck in the flow (Appendix C2). Thus, it is of little surprise that this corridor is recognised in 'Leicester's Local Transport Plan' for 2011-26 as a key problem area, frequently experiencing both general traffic and bus delays. Such localised areas of congestion and their associated increase in traffic emission levels appear to have gone undetected by the 1x1km resolution modelled traffic flows of the NAEI PM₁₀ road-transport emission dataset.

Areas experiencing high levels of smoking prevalence, presented as likely candidates for passive smoking amongst children, tended to exist along the cities western periphery predominantly occupied by the 'White British' populace (Appendix C1). Similarly, obesity hot-spots are observed to exist towards the cities peripheries predominantly occupied by children of 'White British' ethnicity, whom reside far away from the inner city area of concern for respiratory complaints. It should be noted that low smoking and obesity rates are found within and around the aforementioned respiratory hot-spot of concern. This would suggest that these healthy lifestyle measures, either have a limited influence on respiratory health during childhood, or they are overshadowed by greater socio-environmental forces at play.

4.2.2. LEICESTER'S ETHNIC MINORITY GROUPS

The demographic characteristics of residents within Leicester UA reveal that approximately 60.54% of children aged 0-15years are of 'White British' ethnicity, at the time of the 2001 UK Census. Other key ethnic groups for this age range include children of 'White Non-British' (3.32%), 'Indian' (25.73%), 'Other South Asian' (4.18%), and 'Afro-Caribbean' (2.87%) ethnicity. The remaining 3.36% of children are either from ethnic groups that are undefined, or are of other minor ethnic factions relatively unrecognisable within Leicester UA.

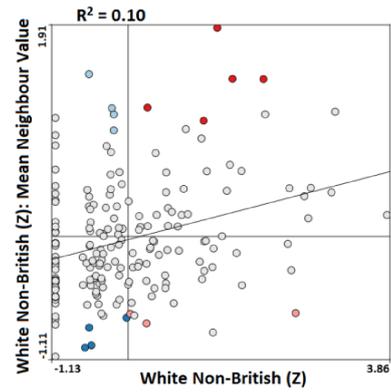
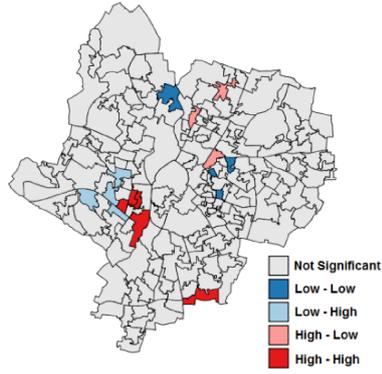
When applying first order weighting schemes, a common Global Moran's I value of 0.78 ($P < 0.001$) was observed for LLSOA community levels of 'Indian' and 'Other South Asian' children, indicating that the selection of residency amongst such groups is of particular importance (Appendix C3). In contrast, low levels of global spatial patterning by children of 'White Non-British' and 'Afro-Caribbean' ethnicity, illustrates a limited importance of residential location when observing their distribution at a citywide scale (Appendix C3). Yet, respective Global Moran's I values of 0.16 and 0.29 under a first order weighting scheme ($P < 0.001$), would suggest that homogeneous pockets are likely to exist, particularly relating to the residential patterning of 'Afro-Caribbean' children. It should also be recalled that

structures of autocorrelation may be unreported at the global scale, particularly in cases where homogeneous pockets diverge from global trends.

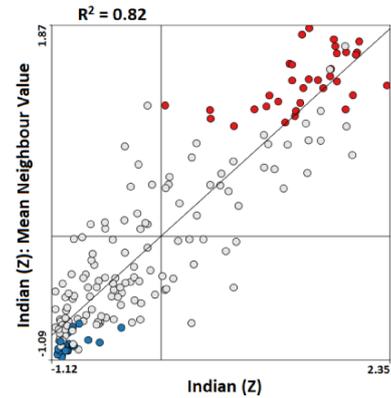
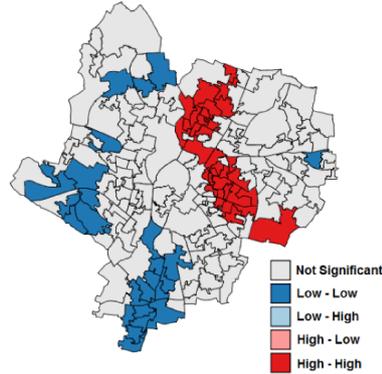
As previously mentioned, children's respiratory cases are particularly location specific across Leicester UA, with moderate levels of correlation occurring only with directly adjacent LLSOA communities. A similar exponentially decaying relationship would appear to define the distribution of 'Afro-Caribbean' children, whereas spatial patterning for the residency of 'White Non-British' children appears relatively inconsequential despite its recorded significance level (Appendix C3). Similar to the spatial distribution of 'White British' children, residential levels of 'Indian' and 'Other South Asian' children declines in a linear fashion with distance, maintaining a moderate degree of correlation with observations separated by distances of up to four orders (radius $\geq 1820\text{m}$). Likewise, Global Moran's Z-Score's for 'Indian' and 'Other South Asian' children recorded significantly greater clustering than expected by chance across locations of moderate proximity.

LISA's placing a row-standardised weight solely upon first order observations were subsequently conducted on Leicester's primary ethnic minority groups in order to detail the strongest elements of spatial positioning provided by each social group (Figure 4.3). In exploring the spatial distribution of ethnic minority groups, one may once again question the potential existence of environmental equality because 'Afro-Caribbean' and 'Other South Asians' tend to reside within inner city areas experiencing elevated levels of deprivation and TPM_{10} emissions. Interestingly the Local Moran's I statistical test identified hot-spots of 'Indian' residents exclusively across eastern Leicester, well outside of the respiratory hot-spot of interest, with this ethnic group appearing to fringe locations heavily populated by children of 'Other South Asian' and 'Afro-Caribbean' ethnicity. Furthermore, cold-spots of 'Indian' residency appear across southern Leicester and along the cities western periphery. Following this statement, one may presume that the lifestyle choices of Indian residents may have a role to play in maintaining a good level of respiratory health.

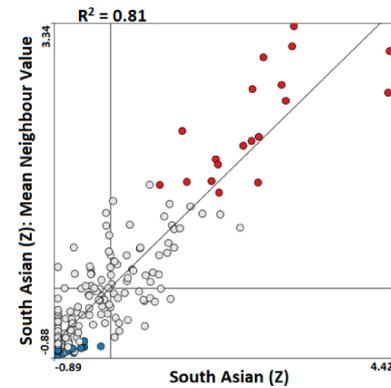
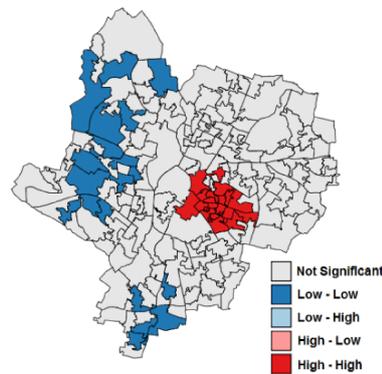
(%) White Non-British Children



(%) Indian Children



(%) Other South Asian Children



(%) Afro-Caribbean Children

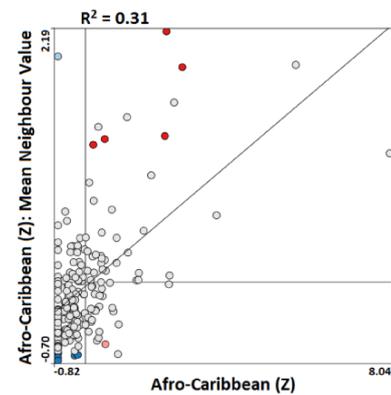
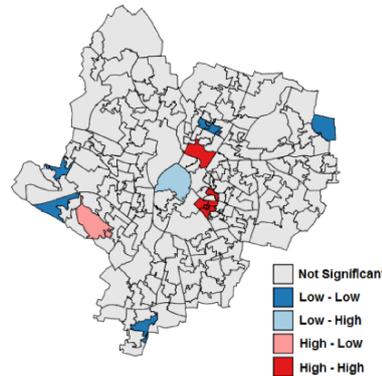


FIGURE 4.3: Local Moran's I cluster and outlier analysis of Leicester UA's key ethnic minority groups, drawn from the 2001 UK Census

4.2.3. NUMERICAL COMPARISON OF SPATIAL EXTREMES

LLSOA's contained within the inner city respiratory hot-spot were observed to experience annual average J00-99 admission rates of 80.65 cases per 1,000 children, whereas communities positioned within cold-spots appeared to experience 1.9 times fewer respiratory hospitalisation rates (27.64 cases per 1,000 children). Substantial socio-environmental differences appear to occur between the inner city hot-spot and cold-spots, with residents from the respiratory pocket of concern experiencing Carstairs Index deprivation scores 4.24 higher, in addition to being exposed to an extra 1.06 t/yr. of TPM_{10} emissions than their cold-spot counterparts. On average an extra 15.14% of residents in J00-99 hot-spot LLSOA's were recorded to smoke, with such communities respectively housing 6.90% and 11.33% more children of 'Other South Asian' and 'Afro-Caribbean' ethnicity. In contrast, 32.78% fewer children of Indian ethnicity occupied hot-spot localities, with such children tending to reside within areas where levels of reported respiratory complaints are lower. It would therefore appear that deprivation, exposure to road-transport emissions and the lifestyle of some (not all) ethnic minorities increases the likelihood of respiratory hospitalisations during childhood (Appendix C4, C5). It is plausible that the lifestyle choices of the 'Indian' minority group actively prevent the occurrence of respiratory hospitalisations, /or that this social group has the relevant knowledge to access public services, mitigating the severity and extent of such complaints.

Upon exploring respiratory admission rates across all socio-environmental influences and ethnic minority groups of interest, it would appear that hot-spots of deprivation, TPM_{10} emissions, obesity, and children of 'White British' and 'Afro-Caribbean' ethnicities are to a certain extent associated with adverse respiratory outcomes (Appendix C4, C5). In particular, hot-spots of deprivation, TPM_{10} emissions and smoking prevalence were detected to respectively experience an extra 20.05, 18.63 and 16.06 admissions per 1,000 children. Such figures correspond to respiratory rates 0.65, 0.48 and 0.51 greater than their equivalent cold-spot localities. In contrast, LLSOA's described as hot-spots of 'Indian' and 'White Non-British' children were respectively identified to experience 13.00 and 9.17 fewer admissions per 1,000 children. Such figures provide corresponding respiratory rates, which are 0.29 and 0.24 lower than what is experienced in LLSOA's which are sparsely populated by children of such ethnic groups. Negligible differences in respiratory outcomes were reported across LLSOA's either densely or thinly populated by children of 'Other South Asian' ethnicities.

Positioned within the centre of the inner-city respiratory hot-spot lies a LLSOA community of particular interest, as revealed by a potentially outlying Local Moran's Z-score of 7.22, which is substantially greater than its mean neighbouring LLSOA value of 1.48 (Figure

4.2). It would appear that this locality is the only LLSOA to be encased entirely by the inner-city ring-road. Here, other respiratory hotspots directly surround this location, with exception to an adjacent north-westerly site. Such locations of interest clearly follow a dispersion gradient in accordance to the UK's prevailing south-westerly wind direction, identifying the importance of road-side emissions on a child's respiratory incidents. Meteorological conditions recorded by Leicester University on a daily basis from 1996-2006 at their land-surface station (MIDAS Station ID: 24942) would appear to be in agreement with the prior statement, with wind speeds averaging 5.07 knots along an trajectory of 200.50 degrees (UK Meteorological Office 2006). Leicester's wind speed values confirm a widespread understanding of Midlands's area being one of the more sheltered parts of the UK, thus offering explanation to why the potential health effects of roadside pollutants only carry across to directly adjacent communities; in cases where considerable quantities of pollutants are emitted. This sharp dispersion gradient may offer some explanation to why an annual respiratory rate of 147.83 admissions per 1,000 children occurs within the inner-city hot-spots focal point, rapidly declining to 49.39-97.96 within the immediately surrounding vicinity. Earlier within this section, it was previously recognised that other unfavourable social influences also have strong elements of spatial dependency, with these influences tending to peak across mutual inner-city localities. Therefore, it is highly likely that multiple burdens collectively impede children's respiratory health within certain residential districts of Leicester.

Residents of TPM_{10} hot-spots are on average burdened with an additional 1.06t/yr. of TPM_{10} emissions than their corresponding cold-spot LLSOA's; a figure almost twice the value of citywide average residential exposure levels (Appendix C4, C5). TPM_{10} hot-spots would also appear to house deprived communities (+4.72 Carstairs Index scores), and respectively house 3.42 and 3.40 times more children of 'Other South Asian' and 'Afro-Caribbean' ethnicities. Such knowledge presents the case of inequalities in exposure prevailing within the post-industrial cityscape. Carstairs Index hot and cold-spot clusters were observed to differ by 8.17 scores of deprivation, with substantially deprived communities experiencing elevated TPM_{10} emissions (+0.77 t/yr.), in addition to 'choosing' an unhealthy lifestyle (+7.27% smoking prevalence, +8.21% obesity prevalence). Deprivation would also appear to have a strong association with ethnic minorities, with 46.51% fewer 'White British' children residing in hot-spot localities. In contrast, children of 'Indian', 'Other South Asian' and 'Afro-Caribbean' ethnicity were found to be 1.69, 3.05 and 2.38 times more likely to reside in a deprived rather than relatively affluent area. Although this relationship is not exclusive, as illustrated by the existence of a deprivation hot-spot within Leicester's western periphery predominantly housing children of 'White British' ethnicity. Elevated levels of smoking were associated with

an increase in deprivation (+3.01 Carstairs Index scores), with smoking prevalence hot-spots housing considerably more children of 'White British' ethnicity (+62.44%). Pockets experiencing elevated community smoking prevalence levels were found to correspondingly house 0.94, 0.92 and 0.22 times fewer children of 'Indian', 'Other South Asian' and 'Afro-Caribbean' ethnicities. Similarly, fewer ethnic minority groups appeared to show associations with obesity prevalence, although the condition appears less exclusive to 'White British' residents (+15% in obesity hot-spots) than what was observed for smoking prevalence.

In exploring Leicester's ethnic minorities (Appendix C4, C5), it was observed that LLSOA communities classified as hot-spots of 'Afro-Caribbean' residents, housed 12.91 times more children of such ethnic origins (+7.87%), than what was observed across their respective cold-spot localities. Extreme differences in community composition levels for this ethnic group appeared related to sizeable changes in TPM₁₀ exposures (+0.70t/yr.), whereas only moderate discrepancies in deprivation (+1.00 Carstairs Index score) were associated with increased levels of 'Afro-Caribbean' residency. Nevertheless, deprivation levels were recorded 2.06 and 3.06 scores above expected citywide values within this social group's respective cold and hot-spots, would imply that 'Afro-Caribbean' citizens are universally afflicted with issues of deprivation. LLSOA 'Afro-Caribbean' hot-spots also appeared to be accompanied by considerable declines in 'White British' residency levels (-48.73%), thus resulting in such LLSOA communities to beneficially experience relatively fewer cases of obesity and smoking.

In contrast, communities densely populated by 'Afro-Caribbean' residents tended to contain 0.50 and 14.53 times more children of 'Indian' (+14.15%) and 'Other South Asian' (+23.78%) ethnicities. Meanwhile, hot-spots of 'Other South Asian' were recorded to house 16.01 times more children of such ethnic origins (+20.00%) than their cold-spot counterparts. Areas densely populated by children of 'Other South Asian' ethnicity were also identified to experience slightly elevated levels of deprivation (+1.77 Carstairs Index score) and TPM₁₀ emission burdens (0.48t/yr.) at their place of residency. However, deprivation levels experienced by 'Other South Asian' residents were generally lower than those experienced by children of 'Afro-Caribbean ethnicity'. Once again, 'Other South Asian' residents were associated with a considerable fall in 'White British' residency levels (-69.09%), with such locations housing 7.11 and 1.94 times more children of Indian (+46.40%) and 'Afro-Caribbean' (+3.69%) ethnicities.

As with Leicester's other key ethnic minority groups, communities coined as hot-spots of 'Indian' residents by and large remained constructed from minorities (+63.40%), reconfirming the importance of spatial dependency amongst Leicester's ethnic groups. Hot-spots of 'Indian' children are surprisingly associated with mild to moderate increases in

deprivation (+1.51 Carstairs Index score) and residentially experienced TPM_{10} (+0.25t/yr.). Both factors have previously appeared to be considerably detrimental to respiratory health during childhood, yet 'Indian' residents are associated with providing a beneficial influence on a community's respiratory health. Out of all the key ethnic minority groups, 'Indian' residents appear to have the strongest disassociation with Leicester's indigenous population, with LLSOA hot-spot communities housing 73.92% fewer children of 'White British' origins.

Subsequently areas densely populated by 'Indian' residents have considerably lower smoking prevalence levels (-21.60%). Nevertheless, low levels of smoking prevalence in communities densely populated by Indian's (17.11%) are unlikely to provide substantial reasoning for this group's beneficial influence on a community's respiratory health, with comparatively low levels of smoking also being connected to hot-spots of 'Other South Asian' (19.53%) and 'Afro-Caribbean' (17.86%) ethnicities. Therefore, as previously thought, it would appear that the social lifestyle choices potentially have a significant role to play in actively preventing and/or mitigating the onset of severe respiratory complaints. Upon comparing the extreme spatial distributions, hot-spots of 'Indian' residents appear to have close ties with the 'Other South Asian' populace. In-fact localities densely populated by 'Indian' residents housed 12.72 times more children of 'Other South Asian' (+12.72%) heritage, whereas only a minor rise in 'Afro-Caribbean' residency levels was reported (+1.71%) between such areas.

Interestingly, the interactions between 'Afro-Caribbean', 'Other South Asian', and 'Indian' ethnic groups and their relationships with respiratory admissions, potentially illustrate a three-stages of the social climb that migrant groups face. For instance, a substantial proportion of Leicester's ethnic minority groups have traditionally occupied low skilled manual labour jobs, with an influx of migrants from the Afro-Caribbean and Asian colonies originally occurring during Britain's post-war reconstruction. These migrant communities settled in the older inner-city areas, where cheap housing was available in the wake of the departure of English residents offered the chance to escape the decay by moving to council estates around the city. (Vidal-Hall 2003). It is conceivable that the collapse of Leicester's manufacturing industries in the 1970's and 1980's would have significantly affected these migrant communities, potentially explaining any long-term residency within deprived areas. Furthermore, a sizeable level Afro-Caribbean migration has also occurred within the last decade through the movement of Somalis from the Netherlands, as low skilled economic migrants (Bonney & Le Goff 2007). These economically disadvantaged and recently arrived ethnic groups characteristically possess a limited knowledge of and access to public services, which would be of importance in explaining adverse health outcomes (COMPAS 2006, DCLG 2006). Although, outputs from the ESDA for 'Other South Asian' communities would suggest

that later generation families of post war migrants have acquired some of the relevant skills to improve their social standing and wellbeing.

In contrast, Leicester's Indian migrants largely comprise of 'twice migrants', whose families previous emigrated from India to East Africa, where they had occupied positions as businessmen and entrepreneurs. Although expelled under 'Africanisation' policies in the 1970's, this wave of Indian migrants were professionally skilled and had prior knowledge on how to successfully integrate, bypassing many of the socio-economic complexities migrants tend to experience (Bonney & Le Goff 2007). ESDA outputs reveal this group as financially able to reside within relatively affluent districts of the city, chose healthy lifestyle choices, and possibly have an advanced knowledge of both public and private services. However, it would be beneficial to err on the side of caution upon interpreting observations constructed from community characteristics that are not representative of the individual, to avoid any ecological fallacy. Still, these somewhat crude social structures open an interesting forum exploring the complexities of societal integration, requiring exploration in successive research.

It would appear that a certain degree of fluidity occurs between these three stages of a migrant's societal climb, occurring in an incremental manner (Appendix C4, C5). For instance, 'Afro-Caribbean' residents respectively represent 8.48%, 5.60% and 2.47% of the children within their own, 'Other South Asian' or 'Indian' LLSOA hot-spot communities. Meanwhile, 'Indian' residents respectively represent 67.31% and 41.60% of the children within their own, or 'Afro-Caribbean' LLSOA hot-spot communities. Whilst ethnic minority groups fluidly move between shared residential locations, a strong sense of segregation with the 'White British' population is still in existence.

Outside research, reveals a widening gap in health inequalities across England and Wales least and most deprived areas during 1971-91; primarily caused by healthier individuals migrating away from deprived areas, whereas persons with poor health show tendencies of sliding into socially disadvantaged communities (Norman et al 2005). Within Leicester it is believed that relocating to a new area constructed from residents whom have increased social and financial choice, would increase ones social ambitions and places peer pressure to 'Keep up with the Joneses'. Yet research has shown that upwardly mobile adults in England and Wales appear to exhibit increased risks of mortality, by comparison to that observed within socially stable demographics who already occupy their class of destination (Blane et al 1999). It was concluded that health gradient constraints continue to prevail, and that social mobility allows one to only moderate, rather than create or amplify, social class differences in health (Blane et al 1999). This same information is likely to hold for exposures during childhood, whereby if social and environmental burdens are allowed to persist then a child may be burdened with

long-term health issues, which can only be mitigated at a later date. This may provide reasoning for why 'Other South Asian' children are associated with a relatively insubstantial influence on community's respiratory admissions, despite their gradual positioning within LLSOA's predominantly occupied by 'Indian' residents.

The ethnic minority group defined as 'White Non-British' were found to predominantly reside within areas experiencing lower levels of deprivation (-5.32 Carstairs Index scores), and experiences TPM_{10} emissions of 1.23 t/yr. This ethnic group appears associated with good levels of respiratory health, with a tendency of sharing locations predominantly occupied by children of 'White British' origins (52.00%). Furthermore, hot-spots were identified to house 1.00, 0.57 and 0.50 times fewer children of 'Afro-Caribbean', 'Other South Asian' and 'Indian' ethnicities. This ethnic group would therefore appear to have integrated into Leicester, via an alternative route, exhibiting similar positive characteristics to 'Indian' residents. Nevertheless the mild spatial dependency of this ethnic group would suggest that comparisons between extreme pockets of residential occupancy are not be taken at face value.

4.2.4. BIVARIATE CORRELATION OF SOCIO-ENVIRONMENTAL INFLUENCES & HEALTH

Traditional dataset correlation tests were conducted to statistically determine whether relationships between individual socio-environmental influences and cases of children's respiratory hospitalisations (ICD-10: J00-99) exist within Leicester's LLSOA communities. The Pearson's Correlation statistic is computed on true values and depicts linear relationships, whereas the Spearman's Rho is computed on ranks and so depicts monotonic relationships. Both tests can vary in magnitude from -1 to 1, with 0 indicating no relationship, and values above 0.3 and 0.7 respectively indicating moderate and strong positive associations between the two variables in question.

Pearson's R-values identified several moderate linear correlations to exist between children's J00-99 hospitalisations and deprivation ($R=0.40$), TPM_{10} emission ($R=0.37$) and residents of 'Afro-Caribbean' ethnicity ($R=0.39$), with significance at the 99% confidence level. Moderate levels of non-linear correlation were also observed to occur between J00-99 admissions and levels of 'Indian' children ($Rho=0.38$) and smoking prevalence ($Rho=0.48$), as denoted by Spearman's Rho values recorded at the 99% confidence level. Carstairs Index values of deprivation across Leicester were observed to solely yield significant linear correlations of interest with TPM_{10} emissions ($R=0.40$), obesity rates ($R=0.51$) and community levels of 'Afro-Caribbean' children ($R=0.30$). TPM_{10} emissions also exhibited linear correlations with residents of 'Afro-Caribbean' ($R=0.42$) and 'Other South Asian' ($R=0.43$) ethnicities.

	Correlation Statistic	J00-99 Admissions	Carstairs Index	TPM ₁₀ Emissions	Smoking Prevalence	Obesity Prevalence	White Non-British	Indian	Other South Asian
Children's J00-99 Admissions	Pearson R								
	Spearman's Rho								
Carstairs Index (Leicester)	Pearson R	0.40**							
	Spearman's Rho	0.39**							
TPM₁₀ Emissions	Pearson R	0.37**	0.40**						
	Spearman's Rho	0.09	0.37**						
Smoking Prevalence	Pearson R	0.36**	0.33**	0.04					
	Spearman's Rho	0.48**	0.31**	0.00					
Obesity Prevalence	Pearson R	0.11	0.51**	-0.27**	0.35**				
	Spearman's Rho	0.30**	0.51**	-0.20**	0.46**				
White Non-British Children	Pearson R	0.05	-0.12	0.11	0.06	-0.27**			
	Spearman's Rho	0.03	-0.15*	0.05	0.06	-0.18*			
Indian Children	Pearson R	-0.29**	0.17*	0.17*	-0.68**	0.01	-0.24**		
	Spearman's Rho	-0.38**	0.06	0.22**	-0.75**	-0.236**	-0.16*		
Other South Asian Children	Pearson R	0.17*	0.29**	0.43**	-0.41**	-0.14	-0.08	0.52**	
	Spearman's Rho	0.00	0.22**	0.36**	-0.50**	-0.246**	-0.08	0.73**	
Afro-Caribbean Children	Pearson R	0.39**	0.30**	0.42**	-0.04	-0.03	0.25**	-0.01	0.35**
	Spearman's Rho	0.17*	0.15*	0.28**	0.01	-0.14	0.12	0.13	0.31**

Significance Levels: *P ≤0.05, **P ≤0.01

TABLE 4.1: Traditional linear (Pearson's R) and non-linear (Spearman's Rho) dataset correlations of children's respiratory health and socio-environmental influences, experienced by residents of Leicester UA: 2000-09

Meanwhile children of ‘Other South Asian’ origins shared a strong non-linearly correlation with ‘Indian’ children ($Rho=0.73$), yet followed a linear correlation with levels of ‘Afro-Caribbean’ residency ($R=0.35$). Such correlation statistics would appear to broadly complement the aforementioned outputs of the Local Moran’s I statistics.

Bivariate Local Moran’s I statistics were conducted in a manner, which held children’s J00-99 admissions at ego locations (i) and placed individual influences of interest at neighbouring LLSOA’s (j). With test outputs therefore highlighting the spatial relationship between respiratory cases and surrounding social-environmental influences, recorded across first-order locations. Relationships of particular spatial interest appear to involve respiratory cases and levels of deprivation ($R^2=0.29$), TPM_{10} emissions ($R^2=0.35$), smoking prevalence ($R^2=0.30$), ‘Indian’ ($R^2=0.18$) and ‘Afro-Caribbean’ ($R^2=0.46$) ethnicities.

	Local Moran’s Statistic		
	R ²	I Value	P Value
Carstairs Index (Leicester)	0.29	0.22	0.00
TPM₁₀ Emissions (t/yr.)	0.35	0.28	0.00
% Smoking Prevalence 16yrs+	0.30	0.24	0.00
% Obesity Prevalence 16yrs+	0.05	0.08	0.04
% 0-15y White Non-British	0.01	-0.01	0.52
% 0-15y Indian	-0.18	-0.17	0.00
% 0-15y Other South Asian	0.15	0.11	0.01
% 0-15y Afro-Caribbean	0.46	0.21	0.00

TABLE 4.2: Summary of the Bivariate Local Moran's I analysis, revealing the spatial associations between children's respiratory admissions and surrounding socio-environmental influences

It would appear, as previously discovered, that levels of deprivation encompass and inhabit inner city LLSOA’s within the central portion of Leicester’s respiratory hot-spot (Figure 4.4). Furthermore, relatively affluent areas towards the cities south and eastern periphery’s, particularly around the Knighton district, would appear spatially associated with reduced levels of severe respiratory symptoms. Likewise, substantial positive correlations between respiratory symptoms and TPM_{10} emission levels appear to inhabit inner city LLSOAs, encompassed and adjacent to the inner city ring road and its northern arterial roads. With correlation, occurring in a less focused spatial manner to that observed in the relationship between relative poverty and respiratory admissions.

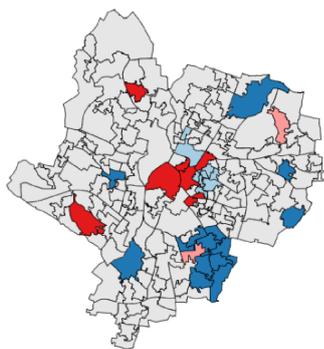
Interestingly, low respiratory incidents accompany reduced TPM_{10} emission levels along Leicester’s eastern periphery, within the wards of Evington and Thurncourt, which house the missing link in the cities outer ring road. Consequently, traffic is forced to leave the outer ring road and enter the heart of the city, producing a bottleneck to the north of these wards.

However, emissions associated with localised areas of congestion appear to have gone undetected by the 1x1km resolution traffic flows of the NAEI PM₁₀ road-transport emission dataset; thus offering some form of explanation for the outlying cases of high respiratory rates within the wards of Humberstone and Hamilton.

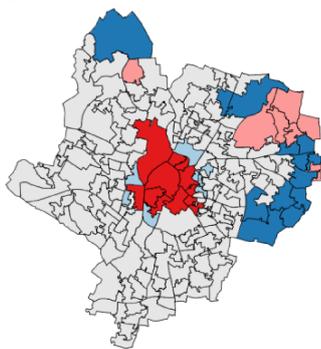
Bivariate Local Moran's I:

(i) J00-99 Admissions Per 1,000 Children; (j) ...

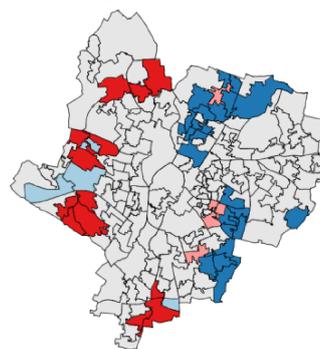
(j) Carstairs Index [Leicester]



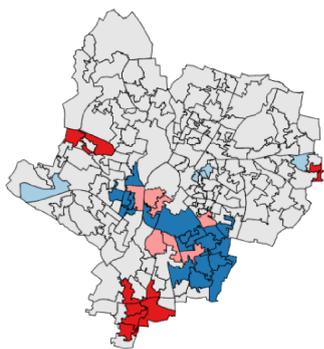
(j) TPM₁₀ Emissions



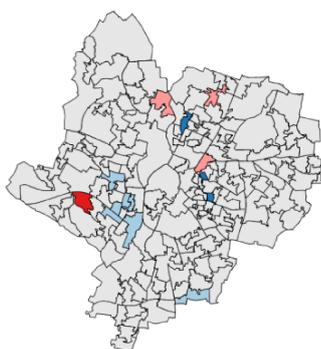
(j) Smoking Prevalence



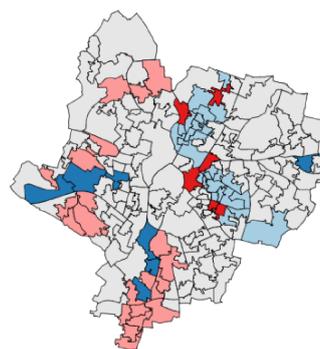
(j) Obesity Prevalence



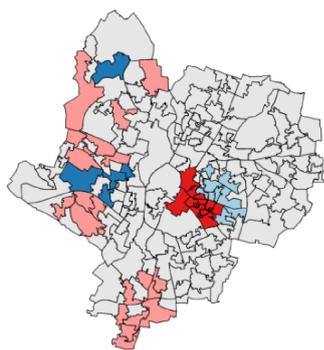
(j) White Non-British Ethnicity



(j) Indian Ethnicity



(j) Other South Asian Ethnicity



(j) Afro-Caribbean Ethnicity

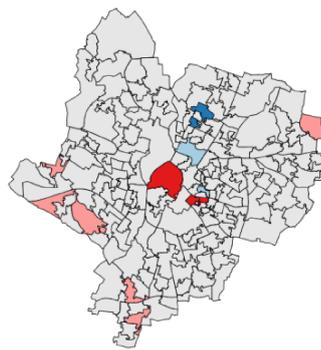


FIGURE 4.4: Bivariate Local Moran's I cluster and outlier analysis of 2000-09 annual average children's respiratory admissions (ICD-10: J00-99) and neighbouring socio-environmental influences of interest

As previously discussed, children of Indian ethnicity seem to be negatively associated with severe respiratory incidents across inner-city areas. Here, Bivariate Local Moran's tests for this ethnic group would also appear to show a strong disassociation with high respiratory

admission rates along Leicester's west and southern peripheries, where 'White British' residency is high. Whilst an extremely low level of 'Indian' residency is behind the formation of these peripheral patterns, the moderately high admission rate (averaging 49.66 per 1,000 children) of these communities is still 10.23 cases above the citywide average.

The substantial relationship between smoking prevalence and residents of white ethnicity would appear to shed light on why children within Leicester's western periphery experience just above the expected quantity of respiratory admissions. One may observe that children of 'Afro-Caribbean' ethnicity also tend to reside away from these localities on the city's western periphery, which experience above average levels of respiratory complaints associated with smoking prevalence and minor deprivation pockets. However, it would appear that this ethnic group has strong ties with adverse respiratory outcomes, through tending to reside within and around LLSOA's at the focal point of the inner-city hotspot of interest.

4.3. GLOBAL REGRESSION: MULTILEVEL MODELLING

In the previous section, measurements of social, environmental and health were individually observed to primarily form localised pockets of a homogeneous nature, confirming that spatial structures are in operation across the City of Leicester. Furthermore, bivariate spatial comparisons of respiratory health outcomes and specific socio-environmental influences reveal the presence of heterogeneous pockets within certain city sectors. It is likely that such spatially deviating responses to the same stimuli are a manifestation of contextual issues within individual intra-urban communities.

However, traditional regression procedures are grounded in the implicit assumption that the variables contained within the model are of a stationary disposition. Yet, the assumption of a uniform modelled relationship over space would be quite misleading, if such relationships were intrinsically different across space, as is the case in Leicester. While Geographical Weighted Regression is perhaps the most promising spatial method in attempting to explain local variation in complex relationships, one may still favour the familiar summarised outputs of a global regression model to ascertain initial knowledge of the study area in question.

Multilevel regression models are a class of global models developed for the analysis of data structures with nested (hierarchical) sources of variability. Observations made within a cluster are usually assumed to be dependent, whereas clusters themselves are assumed

independent of one another. To address dataset issues of spatial nonstationarity, Leicester's LLSOA communities (Level 1: Observations), were nested into quantiles (Level 2: Classification) of the dependent variable J00-99 children's hospital admissions. This was conducted under the assumption that LLSOA observations experiencing similar hospital rates contain populations with comparable disease tolerance levels. In this two-level response model, the unmeasured disease tolerance effect was included through the addition of a level 2 intercept, which differed only across each quantile. By nesting the data points via their respective differences in disease tolerance levels it should be possible to capture the spatial structures associated with admission rates, previously identified by ESDA statistics. As expected, cartographic displays of the quantile J00-99 admission groups identified neighbouring observations to share common nesting structures. Multilevel models, capturing fluctuating levels of susceptibility to respiratory conditions during childhood, were therefore deemed appropriate in addressing issues of spatial dependency amongst the Leicester dataset (Appendix C6).

In the construction of the multilevel models, children's respiratory admissions from 2004-05 were omitted from the calculation of average annual admission rates experienced across the 10-year period of 2000-09. Such a procedure was conducted in order to evaluate model performance through cross-validation measures. Two-tier multilevel regression models, including all 187 LLSOA observations and an upper level structure constructed from 4, 5, or 6-quantile group classification of hospitalisation rates, were applied to globally examine the contemporaneous effect of socio-environmental constraints on children's respiratory admissions. Traditional OLS (Model A) and multilevel (Model B, C, D) linear regression results are summarised in Table 4.3. From here, an identical series of spatial models (E-H) were constructed using 186 locations, omitting the potentially outlying community at the focal point of the inner-city respiratory hot-spot (Table 4.4).

	Model A: OLS Linear Level 1: 187 Observations		Model B: Linear Multilevel Level 1: 187 Observations Level 2: 4-Quantiles		Model C: Linear Multilevel Level 1: 187 Observations Level 2: 5-Quantiles		Model D: Linear Multilevel Level 1: 187 Observations Level 2: 6-Quantiles	
	<i>Estimate</i>	<i>Std. Error</i>	<i>Estimate</i>	<i>Std. Error</i>	<i>Estimate</i>	<i>Std. Error</i>	<i>Estimate</i>	<i>Std. Error</i>
I. Fixed Effects (Normalised 0-1):								
Intercept	28.81***	4.20	34.41***	6.22	33.73***	5.82	34.42***	5.47
Carstairs Index (Leicester)	19.03**	7.98	2.90	5.51	3.69	5.15	2.96	5.04
TPM ₁₀ Emissions (t/yr.)	20.59**	8.14	24.13***	5.53	23.17***	5.17	21.56***	5.13
% Smoking Prevalence (16+yrs)	1.82	6.47	-3.87	4.45	-1.67	4.11	-1.22	4.10
% Obesity Prevalence (16+yrs)	2.01	5.35	0.53	3.63	0.27	3.41	0.24	3.33
% White Non-British Children	-6.96	4.81	-2.59	3.29	-2.89	3.08	-3.59	3.06
% Indian Children	-24.07***	5.60	-5.62	4.06	-3.38	3.86	-2.57	3.72
% Other South Asian Children	13.91**	6.51	-1.41	4.58	-3.12	4.35	-4.70	4.26
% Afro-Caribbean Children	21.07**	9.77	9.36	6.72	5.61	6.28	6.84	6.19
II. Covariance Parameters:								
Intercept			121.94*	88.00	133.42*	86.09	136.81**	80.76
Variance			66.82***	6.99	58.53***	6.14	56.17***	5.91
III. Fit Statistics:								
Akaike (AIC)	1474.10		1356.30		1335.97		1332.06	
Schwarz(BIC)	1506.41		1349.55		1331.68		1329.77	
R ²	0.40		0.72		0.76		0.77	
Cross-Validation R ² (2004-05 Admins)	0.35		0.48		0.48		0.50	

Significance Levels: *P ≤ 0.1, **P ≤ 0.05, ***P ≤ 0.01

TABLE 4.3: Linear OLS and multilevel models of annual J00-99 hospital admissions per 1,000 children aged 0-15 years within Leicester UA: 2000-09

	Model E: OLS Linear Level 1: 186 Observations		Model F: Linear Multilevel Level 1: 186 Observations Level 2: 4-Quantiles		Model G: Linear Multilevel Level 1: 186 Observations Level 2: 5-Quantiles		Model H: Linear Multilevel Level 1: 186 Observations Level 2: 6-Quantiles	
	<i>Estimate</i>	<i>Std. Error</i>	<i>Estimate</i>	<i>Std. Error</i>	<i>Estimate</i>	<i>Std. Error</i>	<i>Estimate</i>	<i>Std. Error</i>
I. Fixed Effects (Normalised 0-1):								
Intercept	29.77***	3.32	34.90***	5.46	34.37***	5.06	35.10***	4.68
Carstairs Index (Leicester)	17.80***	6.31	2.82	3.34	3.81*	1.94	3.03*	1.74
TPM ₁₀ Emissions (t/yr.)	1.85	6.69	7.18**	3.49	6.62**	3.07	4.89*	2.90
% Smoking Prevalence (16+yrs)	0.81	5.12	-4.35	2.70	-2.52	2.35	-2.25	2.23
% Obesity Prevalence (16+yrs)	5.29	4.24	3.35	2.21	2.88	1.95	3.03*	1.81
% White Non-British Children	-2.37	3.83	1.28	2.01	1.06	1.77	0.63	1.67
% Indian Children	-21.66***	4.44	-4.23*	2.47	-2.21	2.21	-1.83	2.02
% Other South Asian Children	13.67***	5.15	-1.04	2.78	-2.57	2.48	-4.16*	2.31
% Afro-Caribbean Children	34.76***	7.84	22.75***	4.15	18.69***	3.65	20.26***	3.42
II. Covariance Parameters:								
Intercept			107.22*	76.48	116.36*	74.16	119.01**	69.24
Variance			24.56***	2.58	19.08***	2.01	16.56***	1.75
III. Fit Statistics:								
Akaike (AIC)	1379.03		1166.55		1125.45		1104.42	
Schwarz(BIC)	1411.28		1159.80		1121.15		1102.13	
R ²	0.48		0.86		0.89		0.90	
Cross-Validation R ² (2004-05 Admins)	0.41		0.51		0.51		0.53	

Significance Levels: *P ≤ 0.1, **P ≤ 0.05, ***P ≤ 0.01

TABLE 4.4: Linear OLS and multilevel models of annual J00-99 hospital admissions per 1,000 children aged 0-15 years within Leicester UA: 2000-09 (Outlier Removed)

Traditional multivariate OLS linear regression procedures constructed from all 187 LLSOA communities (Model A) were deemed to provide a moderate goodness-of-fit to the Leicester dataset (R^2 0.40), with ANOVA F-test's indicating the complete model to be of statistical significance ($P < 0.01$). Model A appears to be fashioned from a particularly high intercept value, which would advocate that citywide base levels of respiratory health are accountable for a substantial proportion of children's respiratory hospitalisation cases. Such background levels of respiratory health are shown to be annually accountable for 1,797 (71.74%) children's J00-99 admissions across Leicester UA. Parameter estimates indicate that average output area rates of PM_{10} road-transport emissions and deprivation are positively associated with children's respiratory hospital admissions. Furthermore, regression estimates for Leicester's major ethnic minority groups indicate the proportion of Indian residents within an area to be significantly and negatively related to risk of respiratory-based hospital admissions, whereas elevated levels of Afro-Caribbean and Other South Asian residents are significantly and positively associated with such admissions.

Across Leicester, deprivation was annually associated with 558 admissions (+22.29%), TPM_{10} emissions with 378 cases (+15.10%) and levels of 'Afro-Caribbean' and 'Other South Asian' residency were recorded to respectively influence 106 (+4.23%) and 142 (+5.67%) children's respiratory hospitalisations across Leicester UA (Appendix C8). In contrast, elevated levels of 'Indian' residency were associated with substantial respiratory benefits, resulting in 498 fewer children's annual J00-99 admissions (-19.89%). Traditional multivariate regression analysis thus provides strong evidence to support ESDA concepts, which suggested that cumulative respiratory-based hospitalisation incidences are distributed disproportionately with respect to socioeconomic status (SES), specific ethnic minorities and environmental exposures within Leicester UA's 0-15 year age group.

Covariance parameters of the multilevel models exploring all 187 of Leicester's LLSOA's (Models B-D) were identified to capture common intra-urban structures of spatial dependency caused by alterations in levels of childhood disease prevalence, below the 90% significance level. Model D, constructed from a 6-Quantile upper level structure of J00-99 admissions, best captured the general magnitude of spatial dependency experienced within pockets of comparable respiratory status distributed throughout Leicester. It is believed that the constructed upper level nesting structures account for the variations in respiratory disease tolerance across higher population sub-sets. Under a scheme incorporating six levels of tolerance, disease susceptibility influenced annual respiratory episodes by -16.26 to 20.88 hospitalisation cases per 1,000 children (Model D). Across Leicester, the random intercept of

the multilevel model, representing unmeasured structures of disease prevalence, accounted for 21 respiratory cases per year (0.85%).

An R-squared value of 0.77 indicates a satisfactory goodness-of-fit for Model D, as confirmed through cross-validation measures with the 2004-05 annual average J00-99 hospitalisation rates omitted from the models construction ($CV-R^2 = 0.50$). Furthermore, respective reductions in AIC and BIC test scores of -148 and -177, compared to its OLS counterpart (Model A), would confirm that multilevel modelling procedures add an element of increased accuracy despite the increasing the level of model complexity. Through incorporating an upper level spatial structure representative of disease tolerance, the multilevel models appear to place a greater emphasis on the responsibility of background levels (fixed intercept) in determining a LLSOA community's respiratory health. For instance, Model D shows that 2,148 admissions (85.72%) are accountable by citywide background levels, a shift of +13.98% above the base rate calculated in Model A. As with the OLS counterpart model, normalised increases in TPM_{10} emission levels were observed to be the driving explanatory force of socio-environmental related respiratory outcomes, causing up to 21.56 admissions per 1,000 children. In comparison, the maximum influence of other socio-environmental variables of interest ranged from only -4.70 to 6.84 admissions per 1,000 children.

On a citywide scale, multilevel modelling of all 187 LLSOA communities identified TPM_{10} emissions to contribute 396 (15.81%) respiratory hospitalisations per annum ($P \leq 0.01$), a figure 0.71% above its counterpart OLS model (Appendix C8). Meanwhile, the impact of other detrimental influences in multilevel models would appear to have severely diminished from their values recorded by traditional multivariate modelling procedures. Levels of deprivation and 'Afro-Caribbean' residency correspondingly influenced 3.47% and 1.37% of children's citywide J00-99 admissions, with such rates respectively initiating 18.82% and 2.86% fewer citywide cases than predicted by Model A. However, the respiratory impact of such social influences was found to not be of significance at the 90% confidence level. Placing lower responsibility on socio-environmental influences also dramatically reduced the beneficial influence of Indian and White Non-British residency, which respectively prevented 17.76% and 1.76% fewer citywide hospitalisations than Model A; although significance was above the 90% confidence level. Nevertheless, multilevel regression estimates for Leicester's major ethnic minority groups would once again indicate the divisive role that community construction has on respiratory health, with some minority groups associated with significantly positive health risks, while others seem to diminish such levels.

Distribution plots of the residuals produced from Models A-D consistently indicate the presence of an outlying location contributing a particularly high residual value, thus causing the distributions to skew to the left (Appendix C9). Furthermore, Kolmogorov-Smirnov (K-S) test scores of the multilevel models (Models B-D) were found to reject the presence of a normal distribution at the 99% significance level (Table 4.5). Meanwhile the K-S score rejected the likelihood of a normal distribution describing the residuals produced from the OLS model (Model A) at the 95% significance level. The lack of normality occurring within the residuals of the multivariate OLS linear regression model (Model A) may raise questions regarding overall model performance, and/or appropriateness, as the ANOVA F-test used for validation is sensitive to the presence of non-normality. However the Central Limit Theorem (CLT) states that given a sufficiently large sample size (e.g. $n=30$), the mean of all samples from the same population will be approximately equal to the mean of the population, following an approximate normal distribution pattern, with all variances being approximately equal to the variance of the population divided by each sample's size (Urdan 2005). In applying the CLT, one may therefore use probabilities associated with the normal curve to answer questions about the means of sufficiently large samples, as are contained in Models A-D.

Arguably, when dealing with datasets of a spatial nature it is of a far greater importance, when necessary, to produce spatially independent residuals. Thus, indicating that spatial structures within the dataset have been sufficiently captured from a correctly specified model. Furthermore, the presence of spatial autocorrelation within the residuals is considered a violation of one of the fundamental assumptions of OLS models, which assume observations to be independent of one-another (Longley & Tobon 2004, Ibeas et al 2012). Global Moran's I: Z-scores identify clustering within the OLS model residuals (Model A), confirming that traditional regression techniques are inappropriate for modelling the non-stationary processes occurring across Leicester. In contrast, Global Moran's I: Z-scores identify no significant spatial patterning for multilevel models B-D, suggesting that elements of spatial dependency amongst observations have been removed. Nevertheless, a new range of linear multivariate OLS and multilevel models were also constructed (Models E-H) in-order to explore the magnitude of such socio-environmental influences upon the removal of the outlying city centre LLSOA.

	Kolmogorov-Smirnov (K-S) Statistic: Residuals Normal Distribution			Global Moran's I: Residuals	
	K-S Value	P ≤ 0.05	P ≤ 0.01	Z-Score	Spatial Pattern
Model A (OLS)	0.11	Reject	Accept	3.66	Clustered
Model B	0.16	Reject	Reject	1.83	No Pattern
Model C	0.17	Reject	Reject	1.12	No Pattern
Model D	0.17	Reject	Reject	1.24	No Pattern
Model E (OLS)	0.08	Accept	Accept	2.28	Clustered
Model F	0.09	Accept	Accept	0.32	No Pattern
Model G	0.09	Accept	Accept	-0.81	No Pattern
Model H	0.10	Accept	Accept	-0.42	No Pattern

TABLE 4.5: Empirical and spatial assessments of normality in residuals from Models A-H

Traditional multivariate OLS linear regression procedures constructed from 186 out of Leicester's 187 LLSOA communities (Model E) were deemed to provide a moderate goodness-of-fit to the Leicester dataset (R^2 0.48), with ANOVA F test's indicating the complete model to be of statistical significance ($F=10.27$, $P<0.01$). A slight improvement in goodness-of-fit was observed through excluding the city centre focal point of elevated respiratory concern, as denoted through respective R^2 and $CV-R^2$ values +0.08 and +0.06 above the outputs recorded by Model A. As with prior models, citywide base levels of respiratory health would appear to account for a substantial proportion of children's respiratory hospitalisation cases. Such background levels of respiratory health are shown to be annually accountable for 74.20% of children's J00-99 admissions across Leicester UA; a value 2.46% above what was recorded via the OLS model constructed from all 187 LLSOA communities.

In removing the one outlying community, normalised incremental increases in levels of TPM_{10} emissions considerably reduced from 20.59 to 1.85 admissions per 1,000 children. Furthermore, TPM_{10} emissions were no longer deemed to alter a child's respiratory status in a manner deemed to be of global significance ($P>0.1$). Across Leicester, Model E holds TPM_{10} accountable for only +1.36% of children's total annual respiratory admissions, a figure markedly beneath the rate of +15.10% recorded in Model A. This would imply that TPM_{10} emissions have a major role to play on the deteriorating health of inner city communities, particularly within the excluded LLSOA that was previously noted to be contained by Leicester's inner-city ring-road. In conducting local spatial regression procedures, issues of outlying observations are addressed through applying location specific weighting rather than a singular or several generalised schemes; consequently one may maintain all observations positioned within the study area of interest. Logic would therefore dictate that such localised relationships should be explored in further detail within the ensuing sections of this chapter.

Model E parameter estimates assigned to all other socio-environmental influences barely digress from those estimates of Model A, with the exception of respiratory rates associated with normalised increments of Afro-Caribbean residency rising from 21.07 to 34.76 admissions per 1,000 children ($P \leq 0.05$). ESDA had previously located a pocket of elevated levels of 'Afro-Caribbean' residency within inner-city localities, which would suggest that hospitalisation influence transferred from TPM_{10} emissions to the lifestyle of this ethnic minority. Across Leicester, deprivation was annually associated to increase the number of respiratory cases by 20.87% ($P \leq 0.01$), and levels of 'Afro-Caribbean' and 'Other South Asian' residency were recorded to respectively influence 6.99% ($P \leq 0.01$) and 5.58% ($P \leq 0.01$) of children's respiratory hospitalisations (Table 4.4, Appendix C8). These citywide values only deviate from corresponding estimates contained within Model A by -1.42%, +2.76% and -0.09%. Levels of 'Indian' residency within a LLSOA community were once again associated with substantial benefits to health, reducing admission levels across Leicester by 17.91% ($P \leq 0.01$).

Covariance parameters of the multilevel models exploring 186 out of Leicester's 187 LLSOA's (Models F-H) were shown to capture common intra-urban structures of spatial dependency caused by alterations in levels of childhood disease prevalence, below the 90% significance level. As before, a multilevel model constructed from a 6-Quantile upper level structure of J00-99 admissions (Model H) was found to provide the best account of previously unmeasured variations in disease tolerance experienced by upper level population sub-sets. In comparing the random intercept values of multilevel models constructed from mutual nesting structures, one may see that the outlying datum point omitted from Models F-H has a limited influence in determining the impact of disease prevalence experienced by upper level population sub-sets. Across Leicester, unmeasured structures of disease prevalence defined by a 6-Quantile J00-99 nesting structure (Model H) were estimated to influence 0.86% of all annual respiratory cases affecting children.

An R-squared value of 0.90 indicates a satisfactory goodness-of-fit for Model H, as confirmed through cross-validation measures with the 2004-05 annual average J00-99 hospitalisation rates omitted from the models construction ($CV-R^2 = 0.53$). Model H appears to provide a noteworthy level of improvement upon its corresponding OLS model, producing superior R^2 and $CV-R^2$ values by respective magnitudes of +0.42 and +0.12 above what Model E's diagnostic tests provided. Furthermore, a reduction in the AIC test score value of Model E by -274.61 again confirms that multilevel modelling procedures add an element of increased accuracy despite the increasing the level of model complexity. Multilevel models omitting the outlying inner-city LLSOA, also appear to provide marked improvements on models containing

all 187 LLSOA's constructed from mutual upper level nesting schemes. As demonstrated by the corresponding improvements in R^2 , $CV-R^2$ and AIC values of +0.13, +0.03 and -227.64 between Model's D and H. As before, through incorporating an upper level spatial structure representative of disease tolerance, the multilevel models appear to place a greater emphasis on the responsibility of background levels (fixed intercept) in determining a LLSOA community's respiratory health. Model H indicates that 87.50% of children's admissions can be accounted by citywide background levels of respiratory health, a level +13.30% above the base rate calculated by its equivalent multivariate OLS model (Model E). In removing the outlying LLSOA community, background respiratory levels of Model H also appeared to explain 1.78% more admission cases than what was previously calculated under the same nesting scheme (Model D).

Model H, displays proportional increases in levels of 'Afro-Caribbean' residency as having the greatest influence on a child's respiratory health ($P \leq 0.01$), 3.14 times greater than the next major influence detrimental to respiratory health, identified as TPM_{10} emissions. In addition, incremental increases in the levels of TPM_{10} emissions, obesity and residential levels of 'Other South Asian' children were identified to have minor influences of respiratory health at the 90% significance level. Across Leicester UA, levels of Afro-Caribbean and 'Other South Asian' residency were found to influence the total amount of respiratory cases by +4.07% and -1.70% respectively. Meanwhile, TPM_{10} emissions were thought to contribute to 3.57% of cases, with levels of obesity influencing 5.44% of Leicester's respiratory hospitalisations during childhood. Compared to multilevel Model D, respiratory cases associated with TPM_{10} emissions, obesity, 'Afro-Caribbean' residency, and 'Other South Asian' residency were found to correspondingly differ by -12.24%, +5.01%, +2.70% and -1.70%. Although levels of 'Indian' residency were not found to be of significance ($P > 0.1$), this ethnic minority group was still associated with reducing respiratory hospital admissions by 1.52%, suggesting a similar influence to what was previously portrayed in Model D.

Distribution plots of residuals formed from the OLS and multilevel models with the city centre outlier removed (Models E-H) appeared to be of a normal distribution as confirmed by K-S scores at the 95% significance level. However Global Moran's I Z-scores reveal the OLS residuals from Model E to cluster, confirming the belief that traditional regression techniques are inappropriate for modelling the non-stationary processes occurring across Leicester. Global Moran's I Z-scores identified no signs of significant spatial patterning for multilevel models omitting the aforementioned city centre outlier (Model's F-H).

To summarise, both traditional OLS and multilevel regression analyses provide strong evidence to support ESDA concepts, which suggested that cumulative respiratory based hospitalisation incidences are distributed disproportionately with respect to socioeconomic status (SES), specific ethnic minorities and environmental exposures of children residing within Leicester. Furthermore, improvements in modelling performances gained through the incorporation of spatial elements indicate the likely presence of non-stationary processes across Leicester, reconfirming the inappropriateness of traditional non-spatial statistics. While TPM₁₀ emissions appeared to be a substantial driving force behind a child's poor respiratory health in global spatial models incorporating all of Leicester's LLSOA communities, its effects appeared to severely diminish upon the removal of an outlying community. At the same time respiratory cases attributed to residency levels of 'Afro-Caribbean' children and deprivation appeared to increase, which would suggest that multiple burdens are likely interrelated at a local level.

In conducting local spatial regression procedures, one applies location specific weighting schemes instead of a single (OLS model) or generalised spatial (multilevel model) schemes that provide average citywide responses. Favourably this enables one to identify and measure potentially unique relations for each observation within the study area of interest. Although the multilevel models produced here provide a global summary of the influential effects of socio-environmental variables in a manner that addresses generalised spatial structures, one should recall the ESDA outputs which revealed homogeneous and heterogeneous pockets to occur across relatively minor distances of $\leq 455\text{m}$ (first order neighbours). While nesting LLSOA's based upon common levels of disease prevalence accounts for the spatial dependency of respiratory responses, such categorisations are too broad in nature. These classifications are fine when LLSOA's share common non-stationary processes. However in certain instances an upper level collection of LLSOA's may experience many specific non-stationary processes, some of which will inevitably be smoothed away. Logic would therefore dictate that the localised variation associated with the complex relationships between health, social and environmental factors should be explored in further detail using a technique known as Geographical Weighted Regression.

4.4. LOCAL REGRESSION: GEOGRAPHICALLY WEIGHTED REGRESSION (GWR)

The preceding sections of this chapter have established that the relationship between a child's respiratory health and likely socio-environmental influences (spatial predictors) appears to vary as a function of a communities geographical positioning within the City of Leicester. A summary of such effects has been provided via global regression strategies accounting for a range of broad spatial structures solely concerning the geographical relationship of respiratory outcomes.

In a broad sense multilevel models offer a structurally appropriate solution, particularly in the capturing of poor respiratory health across inner city locales experiencing elevated levels of deprivation, TPM_{10} emissions and occupancy from certain ethnic groups. Yet in other circumstances these associations appear less pronounced. For instance, Univariate Local Moran's statistics demonstrate looser spatial ties amongst cold-spots of health and the aforementioned socio-environmental influences of interest. Furthermore, bivariate correlations of community respiratory hospitalisations and socio-environmental measures across first order neighbours occasionally deviate from their typically well-defined trend of influence (Figure 4.4: see outlying locales). Here, areas that are fringing the inner-city respiratory hot-spot record reduced J00-99 admission rates despite TPM_{10} emission and deprivation levels remaining unanimously high across adjacent communities. In terms of Indian residency, Bivariate Local Moran's correlations generally agree with this social group reduces community respiratory hospitalisation incidences, yet situations arise where high levels of this population are associated with locales with high visitation rates. Likewise, bivariate plots of Afro-Caribbean residency are shown in rare circumstances to be associated with reduced respiratory hospitalisation rates; thus highlighting the importance of local variations in relationships (non-stationarity) experienced within particular spaces, which are predetermined by spatially autocorrelated socio-physical aspects of the environment.

Local regression techniques, such as GWR provide a means for integrating and exploring multiple non-stationary relationships within a traditional regression model, therefore allowing for a realistic calculation of parameters across space. In certain cases the presence of non-stationarity may produce a mosaic like pattern, whereby the distribution is reflective of the magnitude of spatial deviation between predictor and response components. Yet, circumstances may arise to cause these mosaics to portray scale dependent patterns, which Openshaw (1984) labels as the Modifiable Areal Unit Problem (MAUP). Under its initial premise, MAUP details how the aggregation of raw areal data alters inferences, which may consequently provide a different set of conclusions, thereby raising questions of modelling

uncertainty. With GWR, one may ask the same questions, not at the Leicester datasets spatial resolution as this contains a relatively comprehensive sample of intra-urban communities, but rather placing focus upon the resolution and therefore detail of the spatial interactions that that one would wish to capture.

In some aspects, the flexible nature of GWR may be seen as beneficial, with models constructed from decreasing kernel bandwidths allowing for an increasingly local analysis, which reveal additional geographical details like some sort of spatial microscope (Fotheringham et al 2002). Therefore, larger bandwidths will offer solutions closer to that of global OLS models, whereas small bandwidths will characterise parameter estimates that are increasingly depend on observations of close proximity, but as a result have increased levels of variance. The problem is therefore how to select an appropriate bandwidth to address this bias-variance trade-off. Fortunately, several GWR failsafe (or model critique) measures exist to help select an appropriate window of bandwidths, which accurately capture the occurrences of spatial processes unique to each dataset.

Validation of appropriate GWR spatial weighting structures was primarily achieved through conducting three ANOVA based generalised degrees of freedom F-tests, using the 'spgwr 0.6-14' [R] package. Outputs from the three generalized degree of freedom ANOVA F-tests (Fotheringham et al 2002, Leung et al 2000) collectively identified GWR models constructed from a 40-80NN weighting scheme as significantly improving upon the RSS of OLS models (Table 4.6) in respect of increased model complexity ($P \leq 0.05$). Akaike Information Criterion (AIC) scores describing the relative goodness-of-fit in relation to the degrees of freedom, following a correction for observation sample size (AICc), would appear broadly in agreement with the previously designated lower range of appropriate weighting schemes. For GWR models placing weight upon their 60-180NN AICc scores remaining relatively stable, 0.99-1.30% below their OLS counterpart value (Figure 4.5, Table 4.6). AICc scores for 50NN and 40NN schemes mildly deviated from the OLS test score by -0.14% and +2.24% respectively, marking the point before trade-offs in model complexity and performance become problematic.

As previously discussed, it is possible for GWR models to provide a near-perfect model fit when placing an increased dependency on observations of close proximity; as observed by a R^2 value of 1.00 when conducting a GWR model with a 10NN scheme. Yet such traditional goodness-of-fit coefficients are inappropriate as standalone measures of performance because of their inability to account for bias-variance trade-offs associated with spatial modelling approaches. A more appropriate application of traditional goodness-of-fit approaches was

accomplished through a cross-validation (CV) of GWR models with omitted children's respiratory admission rates from 2004-05. Under these circumstances CV-R² values were observed to meet critical values of ≥ 0.50 , for weighting schemes containing fewer than 70NN's; which once again broadly complement the upper threshold weighting schemes identified via the three F-tests (Figure 4.5, Table 4.6). CV-R² values also indicate that a sufficient level of model robustness exists for schemes inclusive of and containing more than 40NN's, with such coefficients diverging from their modelled R² by $< 30\%$.

Once again one should recall that the presence of spatial autocorrelation within the residuals is considered a violation of one of the fundamental expectations of traditional modelling strategies, which assume observations to be independent of one-another. Global Moran's Z-Scores observe significant clustering ($P \leq 0.05$) amongst residuals from the OLS model and GWR models containing schemes placing weigh on more than 160NN's, which offer solutions close to that of the traditional global model. While weighting schemes placing emphasis upon 160-70NN contain no signs of spatial patterning deeming them eligible for use, one may notice that schemes inclusive of and below 60NN contain significantly dispersed residuals ($P \leq 0.05$); therefore implying that such models optimally define detailed localised spatial structures present within Leicester UA (Figure 4.5, Table 4.6). Finally, in constructing a ratio of AICc and CV-R² scores, it becomes possible to combine measurements of model complexity vs. accuracy with external dataset validation values. Such a ratio score portrays GWR model performances to gradually increase as greater emphasis is placed upon proximal localities, with optimal weighting schemes ranging from 30-60NN (Figure 4.5). Meanwhile GWR schemes including fewer than 30NN appeared to reveal characteristics of over-fitting.

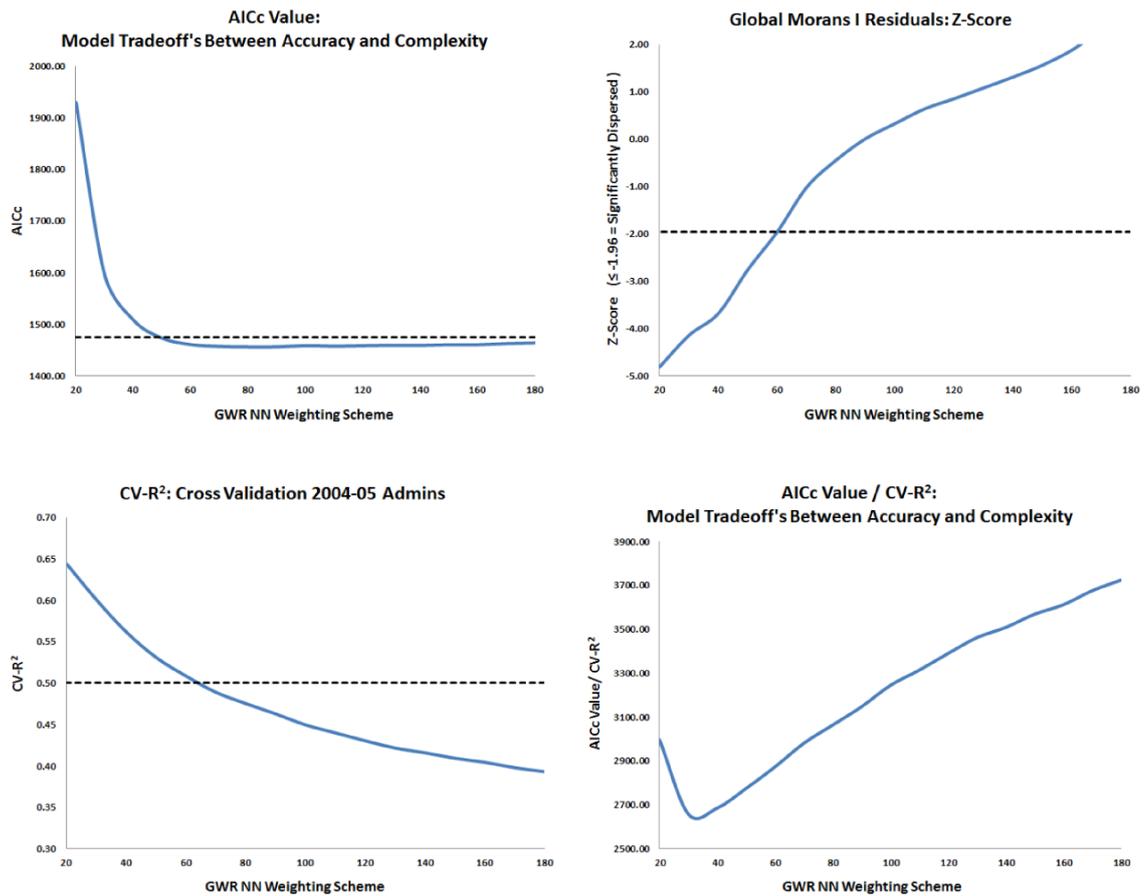


FIGURE 4.5: Statistical evaluation of the GWR spatial weighting schemes

Based on satisfying the three GWR F-tests, minimising the AICc score and avoiding the spatial autocorrelation of residuals it was decided that GWR weighting schemes entailing 80-40NN would offer valid solutions to the spatial processes operating within Leicester UA. From this window of plausible local weighting structures, it was decided that GWR models placing spatial emphasis upon their 80NN, 60NN and 40NN would be examined in further detail in-order to accurately ascertain the impact of the predetermined socio-environmental influences on a child's respiratory health. Furthermore, in examining a range of spatially suitable models, it becomes possible to determine whether scale dependent interactions exist and, if so, to extent do such additional geographical details influence a models conclusions.

Regression Model: Weighting Structure	Goodness-Of-Fit Measures			Relative Goodness-Of-Fit: Accuracy Vs. Complexity		Global Moran's I: Residual Patterning		F-Test: Relative Improvement GWR Vs. OLS		
	R ²	CV-R ²	RSS	AIC	AICc	Moran's I	Z-Score	FBC-F	LMZ-F1	LMZ-F2
OLS Model: 187	0.40	0.35	26086.13	1474.10	1475.35	0.15	3.76			
GWR 180	0.48	0.39	22763.49	1443.65	1463.90	0.10	2.53	1.15	0.92	2.37*
GWR 170	0.49	0.40	22146.64	1440.09	1462.59	0.09	2.30	1.18	0.91	2.30*
GWR 160	0.51	0.40	21478.56	1435.86	1460.55	0.08	1.88	1.21	0.89	2.29*
GWR 150	0.52	0.41	21014.59	1433.39	1460.55	0.07	1.56	1.24	0.88	2.18*
GWR 140	0.53	0.42	20389.00	1429.37	1459.10	0.05	1.31	1.28	0.87	2.15*
GWR 130	0.54	0.42	19942.10	1426.94	1459.45	0.04	1.08	1.31*	0.86	2.06*
GWR 120	0.56	0.43	19282.01	1422.65	1458.54	0.03	0.85	1.35*	0.85	2.01*
GWR 110	0.58	0.44	18508.40	1417.43	1457.61	0.02	0.64	1.41*	0.83	1.97*
GWR 100	0.59	0.45	17753.26	1412.73	1458.67	0.01	0.33	1.47*	0.82	1.87*
GWR 90	0.62	0.46	16644.00	1403.96	1456.41	0.00	0.01	1.57*	0.79	1.86*
GWR 80	0.65	0.48	15411.31	1394.09	1456.24	-0.03	-0.44	1.69*	0.76*	1.81*
GWR 70	0.68	0.49	14046.07	1382.21	1457.24	-0.05	-1.01	1.86*	0.73*	1.74*
GWR 60	0.72	0.51	12353.85	1365.67	1460.79	-0.09	-1.96*	2.11*	0.69*	1.67*
GWR 50	0.76	0.53	10514.55	1345.64	1473.24	-0.13	-2.76*	2.48*	0.65*	1.56*
GWR 40	0.80	0.56	8693.49	1323.86	1508.39	-0.16	-3.68*	3.00*	0.63*	1.42*
GWR 30	0.86	0.60	6192.52	1281.53	1595.88	-0.19	-4.14*	4.21*	0.60*	1.27
GWR 20	0.93	0.64	2962.37	1176.92	1929.46	-0.22	-4.81*	8.81*	0.53*	1.13
GWR 10	1.00	0.63	83.98	564.35	42979.82	-0.12	-2.92*	310.64*	0.49	1.00

Footnote: [A] Moran's I: * P ≤0.05 (Significant Dispersion); [B] GWR F-Tests: * P ≤0.05

TABLE 4.6: Statistical evaluation of the GWR spatial weighting schemes

As previously reported, a single OLS linear regression model was initially applied to examine the concurrent effects of eight explanatory socio-environmental variables on children's respiratory health, the results of which are summarised in Table 4.7. OLS parameter estimates indicate that average output area rates of TPM₁₀ emissions and deprivation are positively associated with children's respiratory hospital admissions. Furthermore, regression estimates for Leicester's primary ethnic minority groups indicate the proportion of Indian residents within an area to be significantly and negatively related to risk of respiratory-based hospital admissions, whereas elevated levels of 'Afro-Caribbean' and 'Other South Asian' residents are significantly and positively associated with respiratory-based hospital admissions. While the ANOVA F-test indicates model significance at the 95% confidence level, reported R² (0.40) and CV-R² (0.35) values for the OLS model indicate only a moderate goodness-of-fit to the dataset (Table 4.7). Furthermore, OLS model residuals were identified to have significant spatial clustering (P<0.05), which suggests that the models assumed uniform relationships between respiratory hospitalisations and individual explanatory variables are invalid, thus reconfirming the presence of local or regional dataset variations.

GWR models with an 80NN, 60NN and 40NN 'Bi-Square Adaptive' weighting scheme were subsequently constructed to quantify the extent to which localised relationship variations influence respiratory outcomes. As previously discussed, adaptive kernels were favoured over a fixed distance spatial kernel scheme in-order to prevent issues of poor model calibration, caused by restricted data point counts in areas where LLSOA's are spatial expansive. Tables 4.7, 4.8 and 4.9 display the summarised numerical results respectively associated with the 80NN, 60NN, 40NN GWR models alongside their Aspatial regression counterparts.

The R-square scores for all three of the investigated GWR models (80NN=0.65, 60NN=0.72, 40NN=0.80) indicate a marked improvement on the OLS (Aspatial) models performance (0.40). This improvement can be further observed by a 40.92%, 52.64% and 66.67% reduction from the OLS mean squared errors value within the 80NN, 60NN and 40NN GWR models, respectively. Yet, in targeting the parameter estimates indicated as significant within the OLS regression model it is possible to observe that the overall relationship between factors of ethnicity, deprivation and TPM₁₀ emissions produce broadly similar outcomes on respiratory hospital admissions. While the global regression model may misrepresent local conditions and yield weaker relationships than the GWR model, such techniques in reality are of a complementary nature; with global models defining significant attributes across a study area, whose interactions and likelihood may then be explored spatially by GWR.

Independent Variable: (Normalised 0-1)	OLS (Aspatial) Linear Regression			Linear 80 Nearest Neighbours Bisquare-Adaptive GWR					
	β Value	Std. Error	P Value	Min. β	Med. β	Max. β	Std. Error	% Census Areas $P \leq 0.05$	
								Detrimental (+)	Beneficial (-)
Intercept	28.81	4.20	0.00*	-8.01	26.37	66.80	1.18	---	---
Carstairs Index 2001	19.03	7.98	0.02*	-25.31	14.65	83.67	1.87	31.55	1.60
TPM ₁₀ Emissions (t/yr.)	20.59	8.14	0.01*	-37.36	27.02	95.09	1.74	54.01	0.00
% Smoking Prevalence (Age 16yrs+)	1.82	6.47	0.78	-23.38	12.92	41.70	1.35	25.13	8.02
% Obesity Prevalence (Age 16yrs+)	2.01	5.35	0.71	-42.43	-2.28	25.91	1.21	12.83	6.42
% 0-15y White Non-British	-6.96	4.81	0.15	-12.10	-1.52	18.49	0.63	0.00	0.00
% 0-15y Indian	-24.07	5.60	0.00*	-62.00	-23.44	2.68	0.97	0.00	56.68
% 0-15y Other South Asian	13.91	6.51	0.03*	-6.25	12.37	95.85	2.04	37.43	0.00
% 0-15y Afro-Caribbean	21.07	9.77	0.03*	-33.99	15.95	68.92	1.74	27.81	0.00
R-Square	0.40			0.65					
Residual Sum Of Squares (RSS)	26086.13			15411.31					
Mean Squared Error (MSE)	139.50			82.41					
AIC	1474.10			1394.09					
AICc	1475.35			1456.24					
F-Test	15.01*			1.69*					
R-Square Cross-Validation: 2004-06 J00-99 Hospital Admissions	0.35			0.48					

* $P \leq 0.05$

TABLE 4.7: 80NN Geographically Weighted Regression (GWR) model of annual average J00-99 hospital admissions per 1,000 Children, 2000-09

Independent Variable: (Normalised 0-1)	OLS (Aspatial) Linear Regression			Linear 60 Nearest Neighbours Bisquare-Adaptive GWR				% Census Areas P≤0.05	
	β Value	Std. Error	P Value	Min. β	Med. β	Max. β	Std. Error	Detrimental (+)	Beneficial (-)
Intercept	28.81	4.20	0.00*	-16.61	28.08	98.62	1.39	---	---
Carstairs Index 2001	19.03	7.98	0.02*	-42.63	15.57	116.11	2.24	32.09	5.35
TPM ₁₀ Emissions (t/yr.)	20.59	8.14	0.01*	-86.18	13.07	116.00	2.19	19.79	6.42
% Smoking Prevalence (Age 16yrs+)	1.82	6.47	0.78	-29.69	12.59	49.61	1.53	25.13	14.44
% Obesity Prevalence (Age 16yrs+)	2.01	5.35	0.71	-84.37	-0.84	29.85	1.53	10.70	5.88
% 0-15y White Non-British	-6.96	4.81	0.15	-15.07	-3.04	23.65	0.76	1.60	0.00
% 0-15y Indian	-24.07	5.60	0.00*	-67.59	-20.11	11.24	1.21	0.00	41.18
% 0-15y Other South Asian	13.91	6.51	0.03*	-18.53	23.20	119.63	2.31	39.04	0.00
% 0-15y Afro-Caribbean	21.07	9.77	0.03*	-48.95	15.99	67.29	2.06	22.46	3.74
R-Square	0.40			0.72					
Residual Sum Of Squares (RSS)	26086.13			12353.85					
Mean Squared Error (MSE)	139.50			66.06					
AIC	1474.10			1365.67					
AICc	1475.35			1460.79					
F-Test	15.01*			2.11*					
R-Square Cross-Validation: 2004-06 J00-99 Hospital Admissions	0.35			0.51					

* P ≤ 0.05

TABLE 4.8: 60NN Geographically Weighted Regression (GWR) model of annual average J00-99 hospital admissions per 1,000 Children, 2000-09

Independent Variable: (Normalised 0-1)	OLS (Aspatial) Linear Regression			Linear 40 Nearest Neighbours Bisquare-Adaptive GWR					
	β Value	Std. Error	P Value	Min. β	Med. β	Max. β	Std. Error	% Census Areas $P \leq 0.05$	
								Detrimental (+)	Beneficial (-)
Intercept	28.81	4.20	0.00*	-33.87	27.28	138.20	1.77	---	---
Carstairs Index 2001	19.03	7.98	0.02*	-62.11	19.63	181.31	2.77	32.09	4.28
TPM ₁₀ Emissions (t/yr.)	20.59	8.14	0.01*	-134.61	5.32	147.04	2.78	5.35	10.16
% Smoking Prevalence (Age 16yrs+)	1.82	6.47	0.78	-39.30	12.05	63.70	1.83	13.90	12.83
% Obesity Prevalence (Age 16yrs+)	2.01	5.35	0.71	-133.05	2.46	41.79	2.14	3.21	3.74
% 0-15y White Non-British	-6.96	4.81	0.15	-21.67	-2.32	32.23	0.92	4.81	0.53
% 0-15y Indian	-24.07	5.60	0.00*	-106.80	-16.06	28.06	1.86	0.00	28.34
% 0-15y Other South Asian	13.91	6.51	0.03*	-34.39	24.69	182.48	2.77	25.13	2.14
% 0-15y Afro-Caribbean	21.07	9.77	0.03*	-71.01	11.27	102.97	2.71	15.51	5.88
R-Square	0.40			0.80					
Residual Sum Of Squares (RSS)	26086.13			8693.50					
Mean Squared Error (MSE)	139.50			46.49					
AIC	1474.10			1323.86					
AICc	1475.35			1508.39					
F-Test	15.01*			3.00*					
R-Square Cross-Validation: 2004-06 J00-99 Hospital Admissions	0.35			0.56					

* $P \leq 0.05$

TABLE 4.9: 40NN Geographically Weighted Regression (GWR) model of annual average J00-99 hospital admissions per 1,000 Children, 2000-09

The spatial distributions of local R-square values generated by the GWR analysis across the three scales of interest are depicted in Figure 4.6. Geographic variations in these values demonstrate how the combined statistical effect of the explanatory variables on respiratory hospitalisations in the 0-15year age group differs across output areas in Leicester. It may be observed that local regression models contained within their corresponding 80NN, 60NN and 40NN weighting schemes provided an improvement upon the R-square value obtained from the global regression model within 90.37%, 89.98% and 92.51% of Leicester’s output areas, respectively. The following section of this chapter will place descriptive focus on the 60NN GWR outputs, based upon the understanding that sufficient model improvements were obtained whilst also retaining a certain degree of smoothing to assist in the description of local variation trends. Where necessary, deviations or omissions in spatial processes between the other explored weighting schemes will also be discussed in further detail.

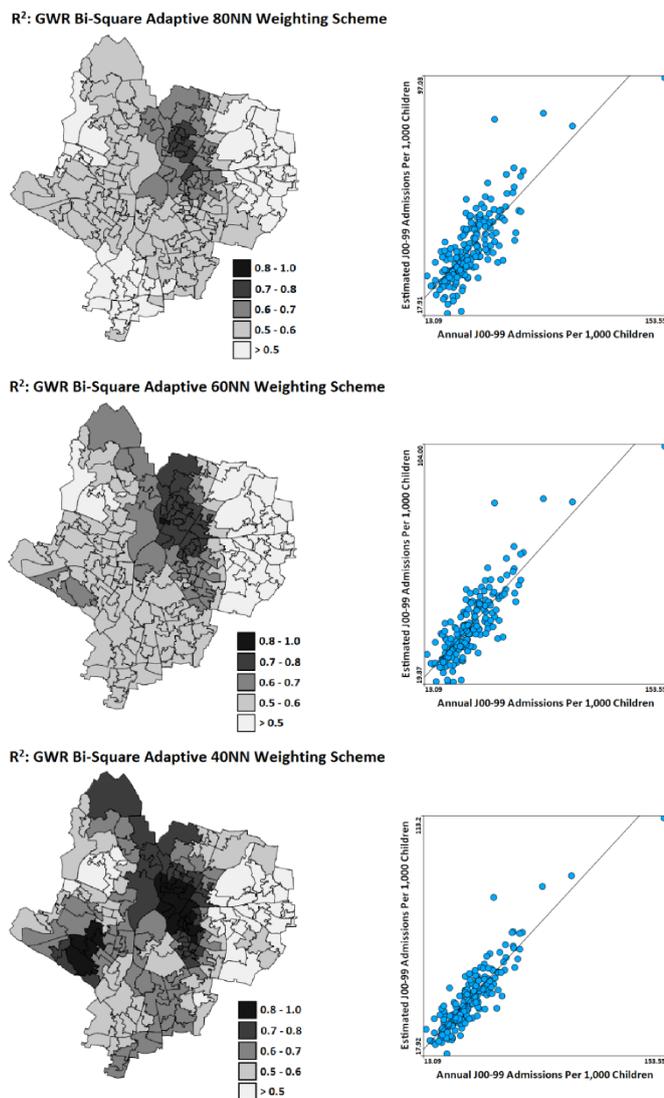


FIGURE 4.6: Local model goodness-of-fit scores contained within each GWR scheme

The strongest model performances for a 60NN scheme are to be found around the north-western section of the city centre, across LLSOA's surrounding and contained within the upper section of high hospital clustering denoted within the LISA analysis (Figures 4.2 and 4.6). In particular LLSOA's contained within the wider wards of Latimer ($R^2=0.78$), Charnwood ($R^2=0.69$) and Spinney Hills ($R^2=0.66$) were observed on average to provide the highest levels of performance. Within the respiratory hot-spot of interest R^2 vales ranged from 0.58-0.73, with a coefficient value of 0.64 for the focal LLSOA of particular interest. However, in other output areas, specifically towards the eastern fringes of Leicester, respiratory hospital admission rates are not explained adequately by our set of explanatory variables. Specifically, LLSOAs contained within the wider wards of Humberstone ($R^2=0.22$), Thurncourt ($R^2=0.18$) and Evington ($R^2=0.43$) were observed on average to exhibit particularly low performance levels.

Interestingly LLSOAs experiencing the highest residual admission rates appear to be located around the incomplete eastern section of Leicester's outer ring road, whose congested traffic flows exist at a higher resolution than that captured within the NAEI modelled road-traffic emission dataset (Figure 4.7). This might imply that the spatial resolution of the emission dataset captures broad trends too ill defined for a select few locales where relatively localised interactions take place. However, on the grander scale of things, the NAEI emission dataset is capable of capture the primary interactions of interest and is readily accessible to the wider population. It is also likely that common epidemiological study issues concerning inter-individual population differences are involved in reduced model performances. Such a statement is made more likely by the relatively low average annual J00-99 admission rates experienced by LLSOAs contained within Humberstone, Thurncourt and Evington (37.33 per 1,000 children), which are 7.35% below average citywide LLSOA levels. These generally high levels of well-being make it highly unlikely that an unknown malicious socio-environmental factor is at play. LLSOAs contained within these three wards also experienced relatively low levels of deprivation, smoking adults, obesity prevalence and ethnic minority groups which were not of Indian ethnicity. Such an amalgamation of 'good' socio-physical community characteristics and relatively few respiratory cases reinforce the likelihood of an individual's actions influencing health to an unmeasurable and minor extent, which only becomes apparent across communities with few health issues.

Upon comparing the performance of localised regressions across the three different weighting schemes of interest it becomes apparent that similar areas consistently model better than others, and that incremental improvements in the fitting of the dataset occur from the additional detail of localised weighting schemes (Figure 4.6). Under a 40NN scheme,

LLSOAs contained within the wider wards of Latimer ($R^2=0.86$), Charnwood ($R^2=0.71$) and Spinney Hills ($R^2=0.77$) were once again observed on average to exhibit a best fit, improving slightly upon the 60NN model. It would appear that the greatest improvements in performance between the 60NN and 40NN models arises along a section of Leicester's western periphery, as observed via average LLSOA R^2 values in Braunstone rising from 0.60 to 0.74. As weighting schemes become increasingly localised, performance generally improves with emphasis placed on capturing the processes occurring across the cities southern and western peripheries, previously skewed from a focus on inner-city locales. However, reduced levels of model performance remain under a 40NN scheme across LLSOAs located within the cities eastern periphery, where relatively few respiratory cases are experienced. Perhaps an individual's rather than community actions are the primary mechanism for poor health in relatively privileged communities, with such noise elements providing a restricted degree of influence elsewhere.

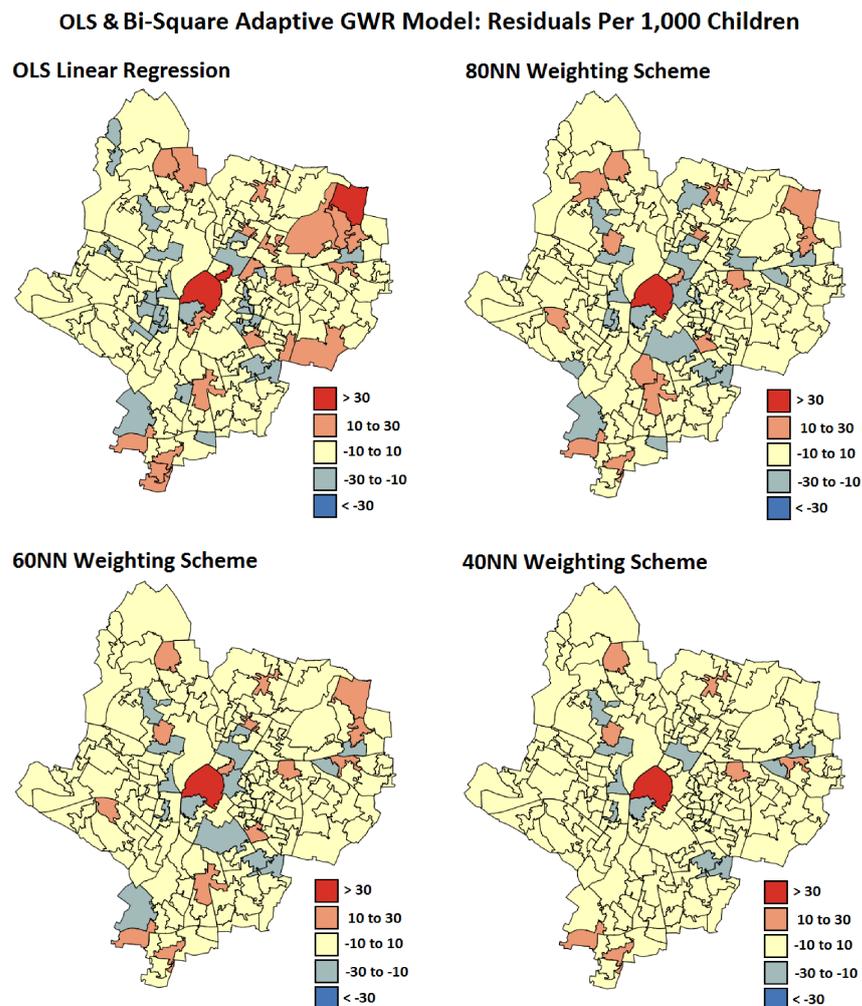


FIGURE 4.7: Spatial distribution of OLS/GWR residual J00-99 hospitalisations per 1,000 children

An assessment of the GWR model residuals (Figure 4.7) confirms this notion of a generally favourable model performance, which cumulatively improves upon the fit of the dataset provided by OLS models with increased localisation. However, it should be noted that high residuals exist within the focal point of the central cluster zone of high respiratory admissions. This is indicative of a reduction in performance of the city centre model (60NN $R^2=0.64$), combined with the area experiencing the highest rate of annual children’s respiratory admissions within Leicester UA (147.83 per 1,000 children). Nevertheless, GWR residuals for the inner-city point of focus were found to reduce drastically from the OLS value (90.61), with respective recordings of 55.92, 48.99 and 39.91 residual admissions per 1,000 for 80NN, 60NN or 40NN models, respectively. This would imply that extremely localised socio-environmental interactions are at play, which likely account for the elevated hospital admissions specifically within this census area. The OLS model also records substantial residual clusters across Leicester’s western periphery within the wards of Humberstone and Thurncourt, which appear to reside under the implementation of localised modelling techniques. Nevertheless, a small band of LLSOA’s within Thurncourt, running from the eastern fringe towards the inner-city consistently under or overestimate GWR modelled respiratory outcomes. Upon recalling the ESDA, such problem locales follow an arterial road feed by Leicester’s Outer Ring road (A563), which terminates at this area of interest, forcing traffic from a dual to a single carriageway towards the city centre. The impact of which becomes apparent for 4 LLSOAs under 60NN and 40NN GWR weighting schemes, but may potentially be alleviated if higher resolution traffic data were to be applied.

Kolmogorov-Smirnov scores for the 80NN, 60NN and 40NN GWR schemes indicated that residuals were distributed in a normal fashion ($P \leq 0.05$), with Global Moran’s I outputs statistically recognising the 80NN, 60NN and 40NN scheme residuals to significantly contain no elements of spatial autocorrelation ($P > 0.05$) (Table 4.10). Such findings reveal a satisfactory model performance, indicating that localised regression techniques are capable of dealing with extreme spatial outliers.

	Kolmogorov-Smirnov (K-S) Statistic:			Global Moran’s I:	
	K-S Value	$P \leq 0.05$	$P \leq 0.01$	Z-Score	Spatial Pattern
OLS	0.11	Reject	Accept	3.76	Clustered
GWR 80NN	0.07	Accept	Accept	-0.44	No Pattern
GWR 60NN	0.08	Accept	Accept	-1.96	Dispersed
GWR 40NN	0.08	Accept	Accept	-3.68	Dispersed

TABLE 4.10: Empirical and spatial assessments of normality in OLS and GWR residuals

In comparing the GWR parameter estimates of the 80NN, 60NN and 40NN GWR models it is possible to ascertain the extent to which spatial interactions are influenced by scale across the statistically selected structural schemes of interest (Table 4.11). By comparing the upper (80NN) and lower (40NN) thresholds of valid GWR schemes it is possible to confirm that overall model estimates are comparable ($R^2=0.90$), and that applying additional geographic detail acts to sharpen such conclusions, akin to a spatial microscope. Furthermore, Pearson's R correlation coefficients indicate strong correlations for parameter estimates relating to deprivation (0.87), TPM_{10} emissions (0.81) and smoking prevalence (0.83). Between the upper and lower thresholds noteworthy differences were only observed between levels of obesity prevalence ($R^2=0.27$, Pearson's R = 0.52) and background admission rates recorded via the Intercept ($R^2=0.36$, Pearson's R = 0.60). One may therefore perceive that the models within the weighting band of interest were already calibrated to a required standard, and that fine-tuning was primarily achieved through these two variables. It is therefore unlikely that scale dependent interactions, if present, will vastly alter inferences derived from the critiqued GWR outputs.

	R²: Model A Coefficient Vs. Model B			Pearson's R Correlation (P≤0.05)		
	80, 60NN	60, 40NN	80, 40NN	80, 60NN	60, 40NN	80, 40NN
Intercept	0.78	0.67	0.36	0.82	0.82	0.60
Carstairs Index	0.93	0.89	0.75	0.94	0.94	0.87
TPM₁₀ Emissions	0.90	0.81	0.65	0.90	0.90	0.81
Smoking Prevalence	0.92	0.84	0.69	0.92	0.92	0.83
Obesity Prevalence	0.71	0.63	0.27	0.80	0.80	0.52
White Non-British	0.91	0.76	0.64	0.87	0.87	0.80
Indian	0.79	0.71	0.39	0.84	0.84	0.63
Other South Asian	0.84	0.73	0.44	0.86	0.86	0.66
Afro-Caribbean	0.83	0.76	0.50	0.87	0.87	0.71
Residuals	0.97	0.92	0.84	0.96	0.96	0.92
Model Estimate	0.98	0.96	0.90	0.98	0.98	0.95

TABLE 4.11: Comparison of GWR coefficient estimates across multiple spatial schemes

Targeting the parameter estimates indicated as significant within the Aspatial regression model it is possible to observe that the overall relationship between factors of ethnicity, deprivation and TPM_{10} road-transport emissions generally produce similar outcomes on respiratory hospital admissions within the GWR models of interest (Table 4.7, 4.8, 4.9). Such observations confirm the complementary nature of traditional models within a local analysis, whereby global regression methods solely identify key influences across a study area, whose interactions and likelihood may then be accurately defined in a spatially detailed fashion via

GWR. A summary of GWR modelled citywide annual children’s respiratory cases resulting in hospitalisation (Table 4.12) reveals levels of deprivation (17.96-21.74%), smoking (3.82-4.27%) and residential levels of ‘Afro-Caribbean’ children (1.32-2.88%) as detrimentally influencing such outcomes in a relatively stable manner. Meanwhile levels of ‘Indian’ (-14.98 to -16.70%), ‘White Non-British’ (-0.55 to -0.72%) residency and obesity (-4.22 to -9.91%) were found to beneficially reduce the number of J00-99 hospitalisation cases across Leicester during 2000-09.

In terms of total respiratory cases only background respiratory levels, recorded via the Intercept (69.21-89.04%), and admissions related to TPM₁₀ emission levels (7.09-21.74%) produced a noteworthy spread between the explored GWR model schemes. The discrepancy between GWR intercept values described in the previous paragraph, relates to the fine-tuning of model weighting schemes focusing on these unique LLSOA background rates. To understand the spread of TPM₁₀, one should recall the performance of localised regression models. Here, as weighting schemes become localised, attention shifted towards capturing processes across the cities southern and western peripheries previously skewed by a modelling focus upon extreme inner-city observations, particularly within the global model. Modelled residuals also provide the impression that extremely localised socio-environmental interactions are at play within the inner-city respiratory hot-spots, which would now appear to predominantly revolve around interactions with TPM₁₀ emissions. However, because of this strong connection to ‘poor’ respiratory health, global and more generalised spatial models would appear to have overestimated the influence of this environmental factor across other districts of the Leicester.

	Int.	Carst. Index	TPM ₁₀	Smoking Prev.	Obesity Prev.	White N-Brit.	Indian	Other S. Asian	Afro-Car.	Resid.
OLS	71.74	22.29	15.10	1.79	3.62	-3.63	-19.89	5.67	4.23	-0.92
80NN	68.21	17.96	21.74	4.34	-4.22	-0.72	-14.98	5.41	2.88	-0.62
60NN	76.18	17.93	15.66	4.37	-6.08	-0.55	-14.98	5.93	2.40	-0.85
40NN	89.04	21.41	7.09	3.82	-9.91	-0.71	-16.70	5.69	1.32	-1.04

TABLE 4.12: Percentage of citywide annual children's J00-99 hospital admissions associated with GWR modelled background and socio-environmental influences

Overall, GWR 60NN model estimates recognise residentially experienced road-traffic emissions (TPM₁₀) after adjustment for social covariates, as accountable for 392 children’s respiratory admissions per annum (15.66% of all children’s respiratory admissions) across Leicester during 2000-09. Median values of the normalised TPM₁₀ parameter estimates were observed to decline from 27.02 (80NN) to 5.32 (40NN), whereas maximum parameter estimates positioned within inner-city locales rose from 95.09 to 147.04 J00-99 hospitalisations per 1,000 children as a greater emphasis was placed on localisation. Cartographic GWR model outputs of TPM₁₀

emissions were produced to specifically explore the spatial influence of environmental pollutants on respiratory health (Figure 4.9). Under a relatively smooth 80NN weighting scheme, TPM_{10} emissions were observed to solely provide a detrimental influence to a communities respiratory health during childhood across the 54.01% of LLSOAs deemed to be of significance ($P \leq 0.05$). The majority of adverse health impacts may be observed to focus upon inner-city residents. In contrast, a relatively detailed 40NN scheme was observed to associate a mixture of detrimental and beneficial health impacts with TPM_{10} emissions, across 5.35% and 10.16% of Leicester's LLSOAs respectively ($P \leq 0.05$).

The 40NN scheme would appear to clarify the inner-city zone of detrimental influence, focusing exclusively upon LLSOAs associated with the J00-99 admission hot-spot. Outputs from a 40NN scheme also significantly highlight a beneficial impact of reduced TPM_{10} emissions across the somewhat rural north-eastern suburbs of Leicester (Rushey Mead and Humberstone). Areas previously associated with low admission rates, but in a non-significant way under wider weighting schemes. Similarly, the southern suburb of Eyres Monsell also appears to link low TPM_{10} emissions with improved respiratory health ($P \leq 0.05$). On a final point, the 40NN model illustrates the emergence of a minor TPM_{10} related respiratory hot-spot following an arterial road into the city centre, which is situated on the fringe and partially within the comparatively affluent south-easterly ward of Knighton (Figure 4.2). One may recall that the car ownership plays a pivotal role in the construction of the Carstairs Index, whereby affluent communities are highly mobile and thus have the potential to create high levels of traffic emissions, some of which will inevitably be emitted at the trip origins. Yet their residentially experienced level of TPM_{10} emissions might only account for a fraction of their total outputs, as observed by the Knighton emission trail which is suggestive of such residents entering the city centre for work or leisure related pursuits. The extent to which such environmental injustices prevail will be explored in a successive chapter focusing upon localised 'Polluter Pays Principles' (PPP).

Across Leicester, GWR 60NN model estimates identify Carstairs Index records of deprivation to annually contributing towards 449 children's respiratory admissions per annum (17.93% of all children's respiratory admissions) during 2000-09. Under all of the explored weighting schemes significant parameter estimates ($P \leq 0.05$) were observed to only drive hospitalisation rates within the direction of deprivation (i.e. affluence was never causally linked to declining respiratory health during childhood). Median values of the normalised deprivation parameter estimates were observed to slightly increase from 14.65 (80NN) to 19.63 (40NN), reflective of the increasing maximum parameter estimates from localised

weighting schemes. The upper parameter estimates were associated with deprived inner-city locales, positioned adjacent to the southern side of the city centre LLSOA. Subsequently, cartographic GWR model outputs of Leicester's LLSOA Carstairs Index deprivation scores were produced to specifically explore the spatial influence of socio-economic status on respiratory health (Figure 4.8).

For either a relatively smooth or detailed spatial weighting scheme, deprivation was observed to detrimentally impact 31.55-32.09% of LLSOAs, consistently placing significant focus upon the central locations positioned within the southern section of the J00-99 respiratory hot-spot ($P \leq 0.05$). However, a hot-spot analysis of children's respiratory admissions associated with deprivation reveals a much wider spread of influence encompassing all of the overall inner-city respiratory hot-spot. These findings clearly indicate that a 'double burden' of environmental exposure and deprivation operates across inner-city communities to collectively impede respiratory health during childhood. Furthermore, reduced levels of deprivation towards the north of the J00-99 inner-city hotspot would appear to significantly reduce communal health burdens, thereby buffering the spatial spread of such collective health burdens.

However, it would appear that this mutual relationship might have an element of exclusivity to inner city locales, as a second deprivation cluster of concern along Leicester's southern periphery (Eyres Monsell) records relatively low TPM_{10} emission levels. Eyres Monsell is a former council estate, therefore one would expect housing stock to be of a relatively reduced standard to the cities other suburban areas. Nevertheless, average Carstairs Index scores for LLSOAs contained within Eyres Monsell (1.09) are not drastically different to the citywide average (0.00) and are substantially better than LLSOAs contained within the J00-99 hot-spot (4.11). This highlights the importance of allowing for non-stationary relationships, as it would appear that deprivation takes precedent in explaining respiratory outcomes along Leicester's southern periphery.

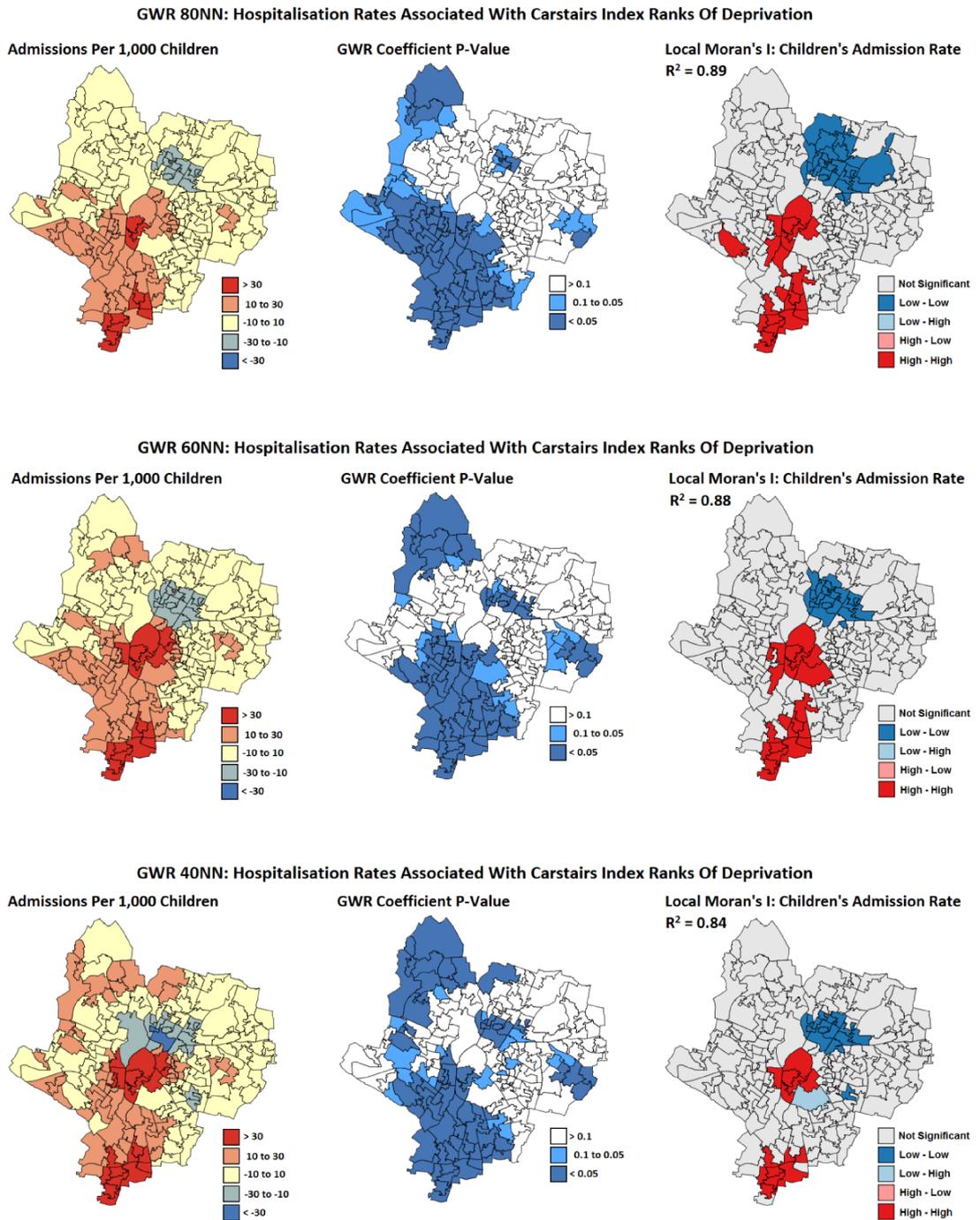


FIGURE 4.8: GWR modelled children's J00-99 hospital admission rates associated with Leicester UA's Carstairs Index scores of deprivation

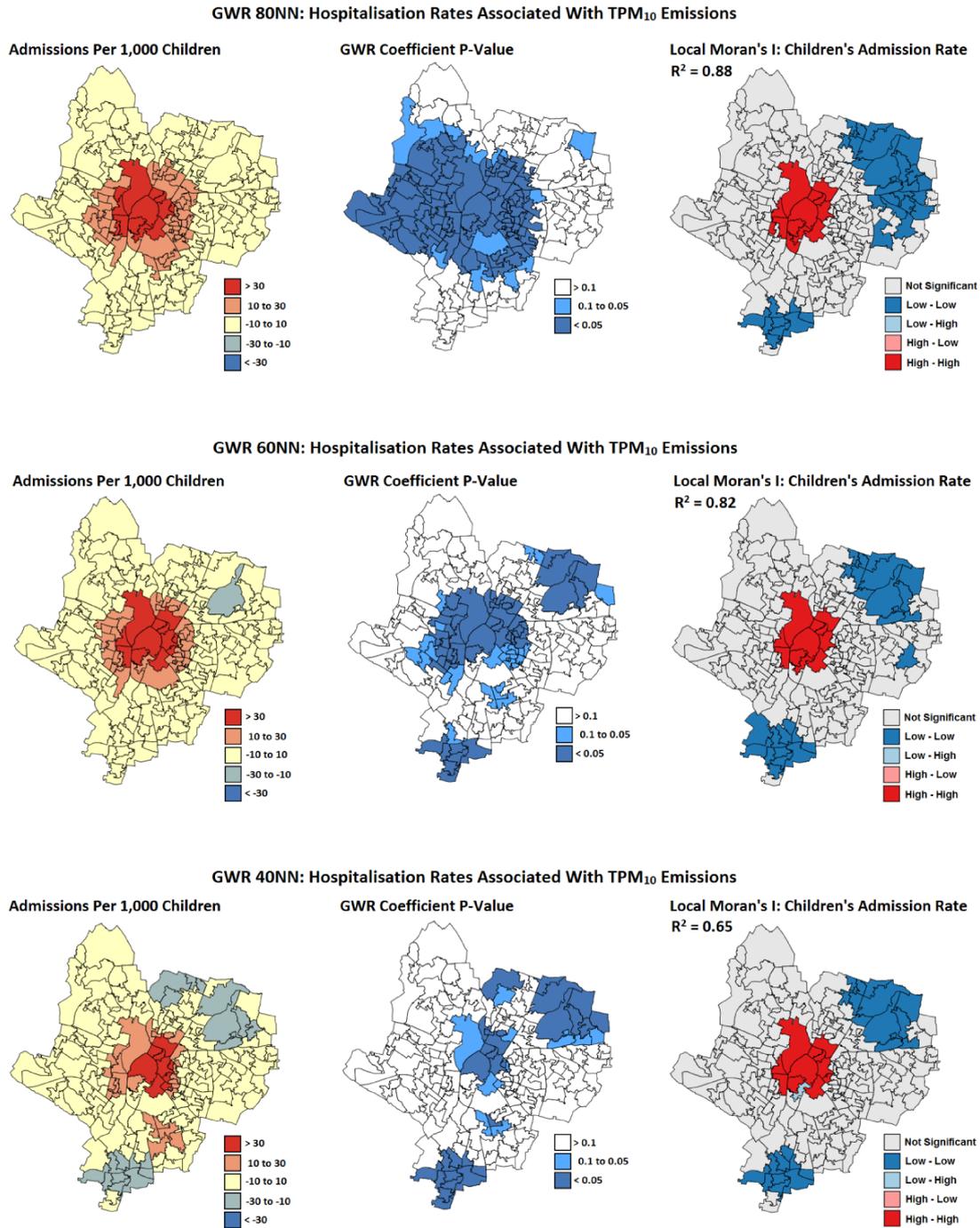


FIGURE 4.9: GWR modelled children's J00-99 hospital admission rates associated with residentially experienced TPM₁₀ emissions

Concerning Leicester's key ethnic minority groups, the lifestyle choices of children of 'White Non-British' Ethnicity seem to have a limited impact on respiratory admissions, whereas elevated levels of 'Afro-Caribbean' and 'Other South' residents are associated with a slight increase in respiratory hospitalisations (Tables 4.7 - 4.9). In-fact the lifestyle choices of 'Afro-Caribbean' residents after adjustment for social-environmental covariates under a 60NN scheme were held accountable for only 60 children's respiratory admissions per annum across Leicester. Such a response is believed to be partially due to low residency levels as median values of Afro-Caribbean normalised parameter estimates range from 15.99 (60NN) to 11.27 (40NN), suggestive of generally detrimental social lifestyle choices. Whilst tending to reside within inner-city LLSOAs surrounding and partially contained within the overall J00-99 hot-spot of concern, it would appear that Afro-Caribbean lifestyle choices here would actually have a low influence on respiratory outcomes.

Local models (Appendix C15) reveal the groups largest detrimental influences to be found around the eastern ward of Charnwood ($\leq 11.14\%$) and south-eastern wards of Freeman ($\leq 4.42\%$) and Knighton ($\leq 4.14\%$) where 'Afro-Caribbean' LLSOA compositions are moderate to low. GWR 60NN normalised parameter estimates for LLSOAs within Charnwood, Freeman and Knighton correspondingly provide average values of 33.40, 40.11 and 31.94 per 1,000 children, whereas average LLSOA estimates within the overall J00-99 hot-spot of concern show that the group provides a beneficial influence (-13.66 per 1,000). This would explain why annual citywide J00-99 admission counts attributed to the ethnic group fall from 4.22% in OLS models to 2.40% under a 60NN GWR scheme. Nevertheless, cartographic plots of admission rates attributed to 'Afro-Caribbean' residence levels clearly show strong associations with increased respiratory admissions (Appendix C15), as indicated by all 27.81% of the significant 80NN GWR modelled outputs acting in a detrimental manner ($P \leq 0.05$).

Whether the lifestyle choices of 'Afro-Caribbean' residents within inner city locales actually mitigate other detrimental influences remains questionable, as all three local models providing insignificant coefficients within such areas. Furthermore, such beneficial outputs fly in the face of the overall citywide trends. Yet, one possibility is that social groups may be forced to positively alter their traditional lifestyle choices, when exposed to an excessive combination of social-environmental burdens that provide recognisable health impacts. Alternatively, the unfavourable lifestyles of 'Afro-Caribbean' residents within inner-city locales may have been overshadowed by the far greater health impacts brought about by the 'double burden' deprivation and air pollutant exposure. In either case, it would appear that this ethnic

minority group unfairly experiences a 'triple jeopardy' of social, health and environmental inequalities.

Lifestyle choices of 'Other South Asian' residents after adjustment for social-environmental covariates under a 60NN scheme were identified to annually influence 149 children's respiratory admissions across Leicester. Like 'Afro-Caribbean' residents, the lifestyle choices of this ethnic minority group are predominantly associated with increased respiratory admissions during childhood, as observed by all 39.04% of the significant 60NN GWR modelled outputs acting in a detrimental manner ($P \leq 0.05$). Under a broader 80NN scheme, significantly modelled burdens for these residents exclusively operate within the western and northern districts of Leicester (Appendix C14), heavily populated by White British residents containing on average only 2.48% of 'Other South Asian' residents. It would therefore appear that respiratory problems prevail in spite of the social mobility that has enabled later generation families of 'Other South Asian' migrants to relocate away from problematic inner-city areas. This may be due to persevered lifestyle choices, which potentially break down to an insufficient understanding of how to access basic public services. Alternatively, if young families have recently relocated away from inner-city areas then their children may experience a health gradient constraint, whereby social mobility allows for one only to moderate existing health issues (Blane et al 1999).

Through examining the 'Other South Asian' respiratory spatial processes in further detail (60NN scheme), it became possible to significantly define a section of the central 'Other South Asian' hot-spot called Stoneygate, where 12.28-35.71% of children are recorded as 'Other South Asian'. Despite the ethnic groups substantial community composition here, it would appear that their lifestyles are only associated with moderate respiratory impacts. Nevertheless under the 40NN scheme, a singular LLSOA at the edge of the overall J00-99 hot-spot attributes 'Other South Asian' residency with improved respiratory health. Perhaps these favourable lifestyle choices are a result of increased knowledge and access of public services, through recognition of the extreme TPM_{10} and financial burdens this community faces.

In stark contrast, 60NN GWR model estimates across Leicester identified the Lifestyle choices of 'Indian' residents to substantially decrease the number of children's respiratory admission by 375 cases per annum. Median values of the normalised 'Indian' ethnic composition parameter estimates were observed to slightly increase from -23.44 (80NN) to -16.06 (40NN), in a manner reflective of the greater emphasis being placed upon the beneficial impacts of specific LLSOAs from localised weighting schemes. Under all of the explored weighting schemes, significant parameter estimates ($P \leq 0.05$) were observed to exclusively

reduce hospitalisation rates. Cartographic plots of GWR outputs consistently indicate that the lifestyle choices of 'Indian' residents actively reduces the number of respiratory hospitalisations within the J00-99 inner-city hot-spot, acting in a manner which mitigates the spatial spread of such symptoms to the wider city centre community (Appendix C13). In particular the ward of Spinney Hills is densely populated by 'Indian' children, of which 3 LLSOAs are encompassed within the 'double burden' zone of deprivation (7.81 points) and TPM₁₀ emissions (2.01t/yr.) experiencing on average 76.67 J00-99 admissions per 1,000 children. Here the beneficial nature of 'Indian' lifestyles is present but obscured by greater burdens. Within Spinney Hills 10 other LLSOAs, average levels of deprivation (3.11 points) and TPM₁₀ emissions (1.30 t/yr.) diminish but still prevail at magnitudes of concern, even so Indian residency remains high resulting in a rate of only 37.60 J00-99 admissions per 1,000 children. This corresponds to 2.69 admissions below Leicester's average LLSOA rate, thus highlighting the importance of social decision making alongside the ease to which public services may be accessed by groups whom have fully integrated into the wider society.

In terms of healthy lifestyle choices, community levels of smoking prevalence previously shown by OLS models to have an unclear insignificant impact on a child's respiratory health, would appear to significantly influence respiratory admissions across 39.57% of LLSOAs under a local model using a 60NN scheme. In total, 60NN GWR model estimates recognised passive smoking during childhood after adjustment for socio-environmental covariates, as accountable for 109 children's respiratory admissions per annum across Leicester during 2000-09. Interestingly, cold-spots of passive smoking hospitalisation rates are positioned throughout southern Leicester and parts of the lower inner-city centre (Appendix C10) where LLSOAs on average record moderate adult smoking rates (35.80%). Furthermore, LLSOAs within the northern and eastern passive smoking hot-spots are also associated with moderate adult smoking rates of 35.80% and 32.88%, respectively. This would indicate that adult smoking does not directly influence the extent to which a child is affected by its associated carcinogens. Rather, it would appear that attitudes to smoking would be of greater importance, including whether the adult is informed or takes action in shielding their children from such risks.

Likewise, OLS models defined adult obesity prevalence as a relatively unclear insignificant impact on children's respiratory outcomes, yet a select number of LLSOA localised models report this lifestyle to mildly impact respiratory admissions, in a significant manner. In fact, 60NN GWR model estimates recognised childhood diets and activities inferred from parent's obesity levels, to influence 152 fewer citywide respiratory cases per annum.

Cartographic plots of the 80NN scheme appear to best depict the generalised respiratory admission patterns related to obesity, with local cluster analysis outputs coinciding with significant GWR coefficients (Appendix C11). Children's obesity hot-spot respiratory admission rates are positioned throughout western Leicester, where LLSOAs on average house a reasonably high proportion of obese adults (26.50%). However, like smoking, LLSOAs within the north-eastern obesity admission cold-spot would appear to house a similar rate of overweight adults (26.10%). Upon closer inspection it would appear that respiratory issues related to obesity would affect ethnic groups in different ways, with LLSOAs within the western hot-spot and eastern cold-spot respectively containing on average 82.48% and 33.38% 'White British' children. It would therefore appear that obesity related respiratory issues during childhood are a concern predominantly related to persons of a 'White British' ethnicity.

While such details are of importance it should be remembered that GWR outputs are founded from localised regression models, each producing individual LLSOA parameter estimates and probability values; therefore, a parameter is unlikely to be of global significance. Subsequently, locations identified to produce significant parameter estimates variables from GWR modelling ($P \leq 0.05$) were selected for the construction of universal stimulus-response models, specific to each independent socio-environmental variable, which account for spatial interference (Table 4.13). The stimulus-response models constructed from 80NN, 60NN and 40NN datasets generally identified each socio-environmental variable to individually influence overall respiratory outcomes via a common function. This would indicate that issues of scale dependent relationships are somewhat trivial when exploring weighting schemes deemed suitable by GWR model selection procedures. Rather, it would appear that such relationships are fine-tuned across the range of suitable weighting schemes. Stimulus-response models constructed from significant GWR parameter estimates ($P \leq 0.05$) were considered to produce suitable relationships for future reference if constructed from a minimum of 25 LLSOA's, which demonstrated an $R^2 \geq 0.5$. In certain circumstances it was also beneficial to produce stimulus-response models constructed from harsher GWR parameter estimates ($P \leq 0.01$), whereby relationships became clouded or needed further validation (Appendix C16).

Independent Variable	GWR Model	Observations (P<0.05)	Optimum Model (P<0.05)	R ²	Constant (B0)	B1	B2	B3
TPM ₁₀ Emissions (t/y)	80NN	101	Quadratic	0.90	0.275	-1.452	11.340	
	60NN	49	Quadratic	0.87	-15.691	10.394	11.223	
	40NN	29	Quadratic	0.93	11.084	-60.585	42.357	
Carstairs Index (Score for Leicester)	80NN	62	Quadratic	0.27	21.123	2.151	-0.226	
	60NN	70	N/A					
	40NN	68	Quadratic	0.10	25.827	-0.087	-1.040	
(%) Smoking Prevalence	80NN	62	N/A					
	60NN	74	Cubic	0.20	69.952	-8.125	0.299	-0.004
	40NN	50	Cubic	0.31	60.272	-7.753	0.318	-0.004
(%) Obesity Prevalence	80NN	36	Quadratic	0.33	1082.217	-86.173	1.699	
	60NN	31	N/A					
	40NN	13	Quadratic	0.82	-153.568	21.354	-0.693	
(%) White Non-British Children	80NN	0	N/A					
	60NN	4	N/A					
	40NN	10	Cubic	0.96	-0.045	-2.379	5.510	-0.844
(%) Indian Children	80NN	106	Cubic	0.87	0.508	-0.539	0.009	-7E-05
	60NN	77	Cubic	0.87	0.508	-0.642	0.014	-1E-04
	40NN	53	Cubic	0.71	1.477	-1.272	0.036	-4E-04
(%) Other South Asian Children	80NN	70	Cubic	0.88	-0.026	2.320	-0.349	0.021
	60NN	73	Cubic	0.88	0.126	1.808	-0.104	0.002
	40NN	51	Cubic	0.69	0.754	1.235	-0.038	0.001
(%) Afro-Caribbean Children	80NN	52	Cubic	0.89	0.072	1.232	0.003	0.002
	60NN	49	Cubic	0.18	0.272	0.501	0.324	-0.049
	40NN	40	N/A					

TABLE 4.13: Stimulus-response models describing the relationship between socio-environmental variables and their specific GWR modelled hospital admissions rates per 1,000 children (If coefficient P≤0.05)

Of noteworthy interest, is the relatively consistent manner in which TPM_{10} emissions and 'Other South Asian' residents appear to detrimentally affect overall respiratory outcomes via their respective quadratic ($R^2 \geq 0.87$) and cubic ($R^2 \geq 0.69$) relationships, obtained from multiple weighting schemes and coefficient significance levels. In contrast, beneficial responses appear associated with high levels of Indian residency, as denoted by cubic functions from several significant weighting schemes ($R^2 \geq 0.71$). Detrimental stimulus-responses attributed to levels of 'Afro-Caribbean' residency were observed to only produce suitable relationships under a 60NN scheme ($P \leq 0.01$) and two 80NN schemes ($P \leq 0.05$, $P \leq 0.01$). Likewise, Carstairs Index deprivation scores of Leicester only produced appropriate relationships when using GWR coefficients with $P \leq 0.01$, under 60NN and 80NN schemes (Appendix C16). The drastic drop in model-fit between $P \leq 0.01$ and $P \leq 0.05$ coefficient values would suggest that deprivation is a complex issue, which is unlikely to be adequately modelled by a single input relationship. Stimulus-response models for levels of smoking and obesity prevalence, and 'White Non-British' residency produced insubstantial and ultimately unclear associations of interest (Table 4.13), and were thus not investigated any further.

The socially adjusted effects of TPM_{10} emissions on respiratory cases can be clearly observed to yield a quadratic relationship, upon modelling significant parameter estimates ($P \leq 0.05$) ascertained from all three spatial schemes of interest (Figure 4.10). Furthermore, the overall quadratic trends would appear to vary little from relationships constructed from restrictive GWR parameter estimate sets ($P \leq 0.01$), thereby suggesting that a stable affiliation has been established. The trend derived from 49 TPM_{10} coefficients ($P \leq 0.05$) under a 60NN scheme ($R^2 = 0.87$) was shown to represent a median admission rate estimate and was subsequently deemed the universal TPM_{10} emission response model of choice (Appendix C17). Within this relationship, LLSOAs experienced TPM_{10} emissions of 1 t/yr. were annually identified to experience an additional 5.92 respiratory hospitalisations per 1,000 children across 2000-09 ($P < 0.05$). Using this trend, LLSOA TPM_{10} emission levels were reported to average 8.79 admissions per 1,000 children, which amounts to a rate 1.29 admissions above the corresponding 60NN GWR model parameter estimates of mixed significance. Overall, TPM_{10} emissions across Leicester would be recognised to influence 475 children's respiratory admissions per annum ($p < 0.05$), which amounts to 18.96% of the total J00-99 respiratory burden; a figure slightly larger than the 60NN GWR model estimate of 15.66%.

This relationship could prove to be an effective measure for quantifying the health benefits of reducing traffic emissions within an intra-urban environment. Within Leicester it is calculated that a 5% reduction in residentially experienced LLSOA TPM_{10} emissions would

amount to 110 fewer children’s respiratory admissions per annum ($P < 0.05$), reducing the total amount of respiratory hospital incidents by 4.39%. Furthermore, it is estimated that a negligible amount of TPM_{10} related hospital admissions could be achieved if LLSOA emission levels could be maintained around 0.81t/yr.

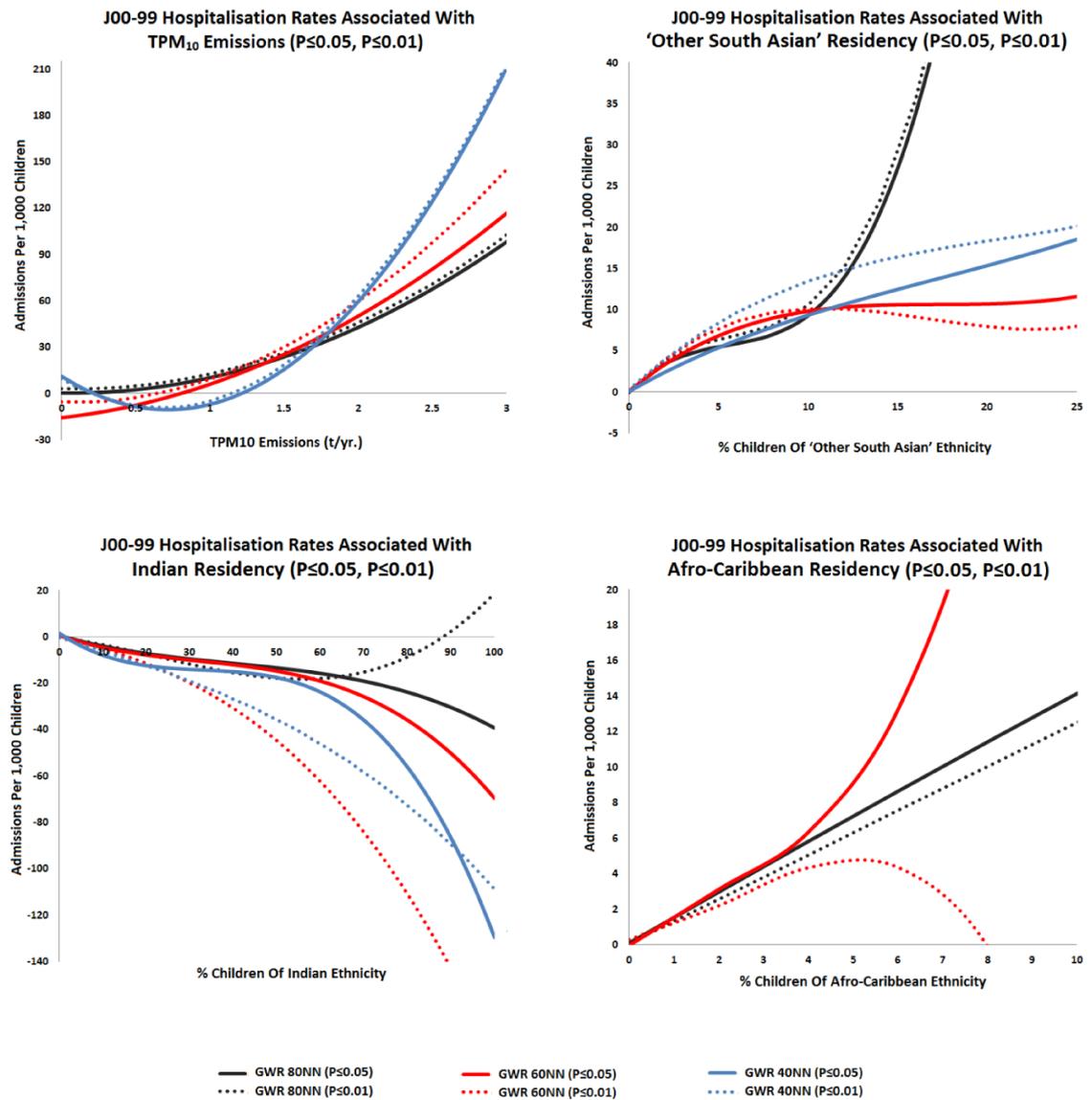


FIGURE 4.10: Universal J00-99 stimulus-responses associated with residentially experienced TPM_{10} emissions, and residential levels of 'Other South Asian', 'Indian' and 'Afro-Caribbean' ethnicities. As constructed from local GWR model outputs of significant ($P \leq 0.05$; $P \leq 0.01$)

The individual influence of 'Other South Asian' residency on respiratory admissions would appear to follow a positive cubic trend, which remains relatively consistent unless >10% of a LLSOA community is constructed from this ethnic group (Figure 4.10). After this point the 80NN scale relationships appear to increase rapidly, yet it would appear that relationships

constructed from 60NN and 40NN coefficients ($P < 0.05$), are constrained within a tight envelope producing similar values. This would suggest that elements of scale dependency are likely associated with this social influence, whereby details are not adequately captured from spatial schemes $> 60NN$. Trends from both 60NN ($R^2 = 0.88$) and 40NN ($R^2 = 0.69$) GWR parameter estimates where $P < 0.05$ equally estimate lifestyles of 'Other South Asian' residency as annually influencing 331 children's respiratory cases across Leicester (Appendix C17). Such figures equate to 13.21% of children's citywide J00-99 burdens, a figure almost twice as high as what was calculated by the complete set of significant and non-significant GWR estimates.

In contrast, the individual influence of 'Indian' residency on respiratory admissions follows a stable negative cubic trend which becomes fine-tuned when including values of $P \leq 0.05$ rather than restricting suitable inputs to significance of $P \leq 0.01$ (Figure 4.10). In-fact the overall cubic trend would appear to scarcely differ from relationships constructed from GWR parameter estimate sets of $P \leq 0.05$ unless a LLSOA community is constructed from $> 80\%$ 'Indian' residents. A trend derived from 77 'Indian' residency coefficients ($P \leq 0.05$) under a 60NN scheme ($R^2 = 0.87$) was shown to represent a central estimate of the envelope in question, and was subsequently deemed the universal response model of choice. Overall, 'Indian' residency across Leicester was associated with the prevention of 699 children's respiratory admissions per annum ($P < 0.05$), reducing the total J00-99 respiratory burden by 27.90%. However, this stimulus-response output is approximately twice the size of the 60NN GWR model citywide estimate of -14.98%.

As previously mentioned, the individual influence of 'Afro-Caribbean' residency on respiratory admissions would appear to follow a positive cubic trend with linear tendencies, which was observed to only act in a significantly consistent nature under the two modelled 80NN scheme trends (Figure 4.10). As with the other ethnic minority groups, it would appear that uncertainty arises in the rare cases whereby an individual minority groups constitute the majority of a LLSOA's population (with exception to levels of Indian residency), due to the limited set of comparable data points for validation. The 52 locations within the 80NN GWR model where $P < 0.05$, estimated that the lifestyle choices of 'Afro-Caribbean' residents would annually influencing 240 children's respiratory cases across Leicester during 2000-09 (Appendix C17). Such figures equate to 9.58% of children's citywide J00-99 burdens, a figure almost three times as high to that calculated by the complete set of significant and non-significant GWR estimates.

In terms of deprivation, the universal stimulus-response models would appear to provide a somewhat obscured relationship, with only the 80NN ($P \leq 0.01$) and 60NN ($P \leq 0.01$)

trends deemed to adequately capture their locational values (Appendix C16). In statistical terms, the best fit was found from an 80NN ($P \leq 0.01$) scheme ($R^2 = 0.80$), which recognised deprivation as contributing to 1714 children’s respiratory admissions per annum. Upon this understanding, deprivation would be associated to influence 68.41% of the total J00-99 respiratory burden, which is a figure substantially larger than the 80NN GWR model estimate of 17.96%. The lack of uniformity between deprivation trends and the wildly differing trend estimates from GWR model outputs would indicate that deprivation is a complex issue, perhaps constructed from a plethora of relationships associated with different elements of social-economic burdens. Upon closer inspection of significant and non-significant 80NN GWR model outputs, it would appear that three different types of deprivation influence respiratory outcomes during childhood (Figure 4.13, Table 4.14).

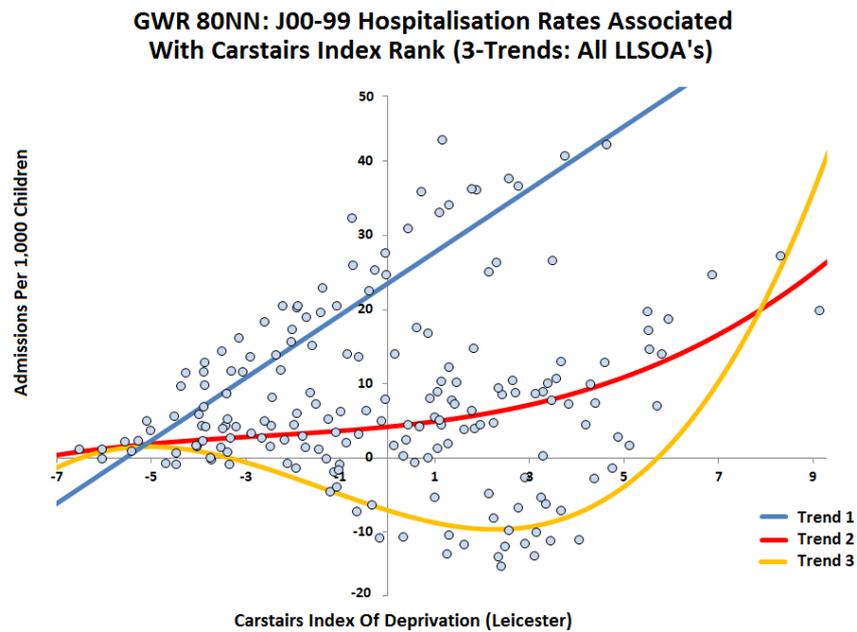


FIGURE 4.11: Universal 3-Trend J00-99 stimulus-responses associated with Leicester UA’s Carstairs Index of deprivation, created from all local GWR model outputs

Carstairs Index	LLSOA	Optimum Model	R ²	Constant	B1	B2	B3
Trend 1 (Upper)	80	Linear	0.80	23.520	4.242		
Trend 2 (Middle)	125	Cubic	0.51	4.187	0.623	0.088	0.011
Trend 3 (Lower)	44	Cubic	0.68	-7.081	-1.967	0.235	0.057

TABLE 4.14: Universal 3-Trend J00-99 stimulus-responses associated with Leicester UA’s Carstairs Index of deprivation, created from all local GWR model outputs

	Carstairs Index Components				
	White British Children (%)	Unemployed Males (%)	No Car/Van (%)	1.0+ Persons Per Room (%)	Social Class D/E (%)
Trend 1 (Upper)	66.77	4.88	33.18	1.83	36.62
Trend 2 (Middle)	53.72	6.48	35.47	4.10	44.69
Trend 3 (Lower)	32.17	5.83	30.86	5.95	44.41

TABLE 4.15: Universal 3-Trend J00-99 stimulus-responses associated with Leicester UA’s Carstairs Index of deprivation, created from all local GWR model outputs

‘Trend 1’ follows a moderate linear relationship, whereby rising deprivation increases respiratory admission rates. ‘Trend 2’ comprises of a relatively flat cubic function, with deprivation only 3-points above the citywide average influencing respiratory outcomes in a notable fashion. Interestingly ‘Trend 3’ portrays moderately deprived communities as experiencing beneficial respiratory responses, which are below responses experienced via relatively affluent communities in respect to this particular type of deprivation. However after a Carstairs Index value of 5 (heavily deprived), children experiencing ‘Trend 3’ type deprivation appear to exhibit characteristics of rapidly diminishing respiratory health.

LLSOAs typical of ‘Trend 1’ would appear to predominantly house ‘White British’ children that experience relatively low levels of overcrowding. Such candidates appear affected by a broad range of deprivation related issues, which as individual components are of only mild-moderate concern (Table 4.15). ‘Trend 2’ is representative of a moderate range of deprivation influenced health impacts, with such communities housing an even split between minority and ‘White British’ residents, whom experience the highest rates of unemployment (Table 4.15). Financial deprivation would appear the main force driving the respiratory issues experienced by this subset. LLSOAs contained within ‘Trend 3’ seem to predominantly house ethnic minorities, who are likely to be from low social class families which experience the highest levels of overcrowding out of the three subsets (Table 4.15). This subset appears chiefly influenced by issues of financial deprivation combined with residing within the city’s poorest housing stock. Yet the increased level of mobility may play some role in evading residentially related aspects of deprivation, possibly offering reason for why moderately deprived communities following this trend have a reduced respiratory burden. After accounting for such observations, it was judged that the appropriate deprivation subset trend should be applied on the following basis:

TREND 1: >50% ‘White British’ Children & <3% Overcrowding
TREND 3: <40% ‘White British’ Children & >5% Overcrowding
TREND 2: All Other LLSOA’s

On this basis the 3-trend universal deprivation-response model (Table 4.14) was found to associate deprivation as annually influence 486 children's respiratory cases across Leicester. This equates to 19.40% of the total J00-99 respiratory burden, a figure that more closely matches its corresponding 80NN GWR model estimate of 17.96%.

4.5. CONCLUSIONS

Geographically detailed population and road-transport emission datasets across the intra-urban arena indicate that children's environmental inequities currently exist within the prototypical British multicultural city of Leicester. In general, children from lower social class households tended to reside within areas experiencing relatively high levels of road-transport emissions, thought to be substantially created by external affluent communities. Such findings appear to be in agreement with Pearce et al's (2010) 'triple jeopardy' of social, health and environmental inequalities.

Traditional global multivariate regression results recorded each tonne of residentially experienced TPM_{10} emissions within Leicester to be accountable for an annual increase of 7.78 J00-99 hospital admissions per 1,000 children over a ten year period ($P < 0.05$), after controlling for socioeconomic characteristics. Community measures of deprivation and ethnic composition were also identified as significant predictors of children's respiratory hospitalisation incidence, indicating that social inequalities do indeed exist. Multilevel models were constructed to address certain dataset issues of spatial nonstationarity through the incorporation of a generalised set of spatial structures (5-quantiles) exploring variations in disease tolerance. Multilevel models similarly identified a 1 t/yr. increment of TPM_{10} to annually account for an additional 8.75 J00-99 hospital admissions per 1,000 children ($P \leq 0.05$). However, after removing an outlying city centre LLSOA of interest, global rates associated with TPM_{10} emissions fell to 2.50 J00-99 admissions per 1,000 children ($P \leq 0.05$). Thus highlighting the spatial dependence of certain socio-environmental burdens, of which inner-city children would appear to experience an overwhelming detrimental impact caused by TPM_{10} emissions. Under the same model, a 10% rise in the levels of Afro-Caribbean residency provided 5.61 J00-99 admissions per 1,000 children ($P \leq 0.05$), a figure consistent with OLS outputs for all 187 LLSOA's. Therefore, one may also infer that social lifestyle choices, including knowledge of and access to public services, has a role to play in preventing relatively severe respiratory outcomes during childhood.

These disparities among rates of respiratory hospital admissions within specific ethnic minority groups may partially be explained by migration patterns within Leicester. During Britain's post-war reconstruction, migrants from the Afro-Caribbean and Asian colonies were actively encouraged under the British Nationality Act 1948 to assist with the reconstruction of the British economy. Within Leicester, a substantial influx of these initial migrants arrived from the South Asian states of India, Pakistan and Bangladesh. Chain migration within these groups continued as vacancies in low skilled factory jobs abandoned by the local population appeared in the post-war boom years (Vidal-Hall 2003). However, the collapse of Leicester's manufacturing industries in the 1970's and 1980's would have significantly impacted the South Asian migrant communities, potentially explaining why the Pakistan and Bangladesh sections of this group continues to reside within deprived areas. In contrast, Leicester's Indian migrants largely comprise of 'twice migrants', whose families previous emigrated from India to East Africa, where they had occupied positions as businessmen and entrepreneurs. Although expelled under 'Africanisation' policies in the 1970's, this wave of Indian migrants were professionally skilled and had prior knowledge of successful integration, potentially bypassing many of the socio-economic complexities migrants tend to experience (Bonney & Le Goff 2007). Thus providing reasoning for why traditional and spatial models of both a global and local nature consistently identify the lifestyle of Indian residents as exclusively influencing a community's respiratory health in a beneficial manner.

Similar discrepancies in respiratory admissions across UK ethnic minorities were reported by a postal questionnaire of 6,080 Leicestershire children aged 1-4 years, which identified odds ratios for cases of multiple wheeze and viral wheeze to be 2.21 and 1.43 times greater respectively in children of South Asian ethnicity (Kuehni et al 2007). A 2004-2007 study of 56,616 infant admissions to Paediatric Intensive Care Units within England and Wales identified acute respiratory failure risk-adjusted mortality to be 1.76 times greater in South Asian infants compared with the rest of the population (O'Donnell et al 2010). Furthermore, a 1990-1991 study exploring the influence of ethnic group on asthma treatment of 5,494 English and Scottish primary school children, identified children of Afro-Caribbean and Indian subcontinent origin to less likely to receive β 2 agonists (Duran-Tauleria et al 1996). This indicates that a limited knowledge of and access to public services, by certain ethnic groups, may be of importance in mitigating adverse health outcomes.

This chapter has also shown the importance of local regression techniques for the integration and exploration of multiple previously unidentified non-stationary relationships, when exploring datasets with a spatially dependency. In contrast to the conventional

regression analysis, GWR indicated that many of the observed statistical associations between respiratory admissions and specific explanatory variables are not uniform across Leicester. Despite the differences in regression methodology, the GWR results consistently indicate the pervasive effect of traffic emissions, deprivation and ethnicity in explaining the geographic distribution of children's respiratory admissions. Local GWR models indicate residentially experienced TPM_{10} emissions after adjustment for social covariates, as accountable for 178-545 children's respiratory admissions per annum (7.09-21.74% of all children's respiratory admissions) across Leicester during 2000-2009. Furthermore, areas of elevated respiratory hospitalisation cases from road-transport emissions appeared to coincide with cases for deprivation, typically across inner city localities housing high levels of ethnic minority groups. Across Leicester UA, deprivation was estimated to account for 449-536 children's respiratory admissions per annum (17.93-21.41% of all children's respiratory admissions).

Kingham et al (2007) reported the occurrence of similar social gradients in exposures to vehicle pollution and variations in exposure among different ethnic groups across Christchurch, New Zealand. Furthermore, the greatest disparities appeared to exist across low-income households (\$0 to \$30,000), with mean vehicle pollution levels differing by approximately 1.5–2.0 times in quintiles at opposing ends of this spectrum (Kingham et al 2007). In an assessment of the Health Survey dataset for England, Wheeler & Ben-Shlomo (2005) identify low social class and poor air quality to be independently associated with decreased lung function, but not asthma prevalence, after adjustment for a number of potential confounders. Within this study, urban lower social class households were generally prone to be located in areas of reduced air quality, but if anything, the association in rural areas became reversed. Thus highlighting the importance of future EJ studies to adopt statistical techniques, like GWR, which consider the importance of localised interactions.

The GWR model outputs also allowed for the creation of a road-transport emissions dose-response relationship from the localised regression models with significance $P \leq 0.05$. Using the trend captured for a 60NN GWR model, LLSOA TPM_{10} emission levels were reported to average 8.79 admissions per 1,000 children, causing 475 children's respiratory admissions per annum across Leicester (18.96% of the total J00-99 respiratory burden). The threshold for the occurrence of hospitalisation was also identified to occur within areas experiencing annual TPM_{10} emission rates above 0.81 tonnes ($P < 0.05$). Furthermore, it is calculated that a 5% reduction in residentially experienced LLSOA TPM_{10} emissions across Leicester would amount to 110 fewer children's respiratory admissions per annum ($P < 0.05$), reducing the total amount

of respiratory hospital incidents by 4.39%. To the authors knowledge this is the first time a dose-response relationship has been specifically associated with road-transport emissions.

While, GWR modelling produced generally favourable outputs, it should be noted that particularly high residuals were identified within the focal point of elevated inner city respiratory admissions. As described earlier, extremely localised environmental and intra-individual population variables, may account for elevated hospital admissions observed within this census area. It is very likely that localised congestion and their associated increase in traffic emission levels have a prominent role to play in this relationship, as observed by other model residuals tending to exist along road junctions of concern. Another solution may reside within the creation of a multiple environmental deprivation index (Pearce et al 2011), which considers other pathogenic factors such as temperature and density of industrial facilities, in addition to factors supporting human well-being including access to green-space and UV radiation. In exploring the local intra-urban relationships of a post-industrial city, such pathogenic factors are of limited importance; however, salutogenic factors such as access to green-space and UV radiation can measure environmental content and magnitudes of psychosomatic illness. Such factors are also likely proxies of periods spent indoors, thus allowing for the accommodation of indoor exposures in future research.

The study's findings concerning local transport planning indicate that the completion of Leicester's outer ring road would likely reduce the severity of children's respiratory outcomes within a small community near the eastern periphery. Furthermore, Leicester's 2011-2026 Local Transport Plan indicates that the completion of the outer ring road, included in a package of over £2million in road improvements, would likely reduce the number of vehicles presently entering the city centre (Leicester City Council 2011). However, it is unlikely that such policies will be of priority, taking into account the current balancing of Local Authorities budgets and the fact that road building is generally not on the current government's agenda. In addition, a substantial carbon impact would have to be accommodated for during the improvement and construction of such road networks.

Like all spatial epidemiological studies exploring the impacts of air pollutants, this research has limitations, specifically involving exposure assessments. First, by international standards, Leicester is a relatively small city and the absolute levels of air pollution are not particularly high. Nevertheless, this study has identified the existence of significant spatial variations in exposure to traffic emissions associated with social patterning. Secondly, estimates of annual vehicle emissions were obtained through modelled vehicle flows of national and regional vehicle compositions, which although vary spatially, do not account for

the actual dispersion of vehicle pollutants. Nonetheless, it should be remembered that studies based around monitored pollutant concentrations and dispersion modelling often assumed the overall contribution from traffic sources, whereas the application of emission data allows for the direct assessment of the road transport component on health outcomes. Thirdly, it has been assumed that people's exposure to vehicle emissions can be approximated by residentially experienced levels, despite the fact that people often spend substantial quantities of time away from home. Although it is reasonable to claim that children spend a larger proportion of time around home or attending educational facilities within close proximity, thus lending credibility to the application of residential emission levels as an exposure proxy.

THE EFFECT OF SOCIO-ENVIRONMENTAL MECHANISMS ON DETERIORATING RESPIRATORY HEALTH ACROSS URBAN COMMUNITIES DURING CHILDHOOD: LEICESTER UA 2000-09

OVERVIEW

Spatial modelling techniques incorporating the social and physical structures of urban environments, previously established a ‘triple jeopardy’ of social, general respiratory health (ICD-10: J00-99) and environmental inequalities as operating within the multicultural UK City of Leicester. This chapter aims to expanding upon these initial findings, through exploring whether spatial relationships exist between specific relatively minor and severe respiratory conditions, and if so, what is the extent to which socio-environmental mechanisms play in the decline of a child’s respiratory health.

Specific focus was placed upon respiratory infections of the upper (ICD-10: J00-06) and lower (ICD-10: J20-22) respiratory tract, which are recognised as the primary influence of children’s respiratory related complaints (58.52%) and portray a progressive decline in respiratory health. Global and Local Indicators of Spatial Autocorrelation (GISA, LISA) statistically describe and illustrate the spatial nature of socio-environmental influences and average annual hospital admission rates associated with upper and lower respiratory conditions experienced by children residing within Leicester UA from 2000-09. Spatially appropriate modelling procedures, accounting for underlying geographical structures at a common spatial resolution, were then applied to define the extent to which socio-environmental variables of interest individually influenced relatively minor and severe respiratory complaints during childhood via shared pathways. This chapter covers objectives 1, 2, 4 and 5 of this project outlined in Chapter 1.

5.1. INTRODUCTION

Road-transport accounts for a substantial proportion of the air quality objective pollutants experienced within the Post-industrial cityscape, attributed to the movement of labour forces and physical merchandise often within close proximity to residential districts. Across England substantial demographic disparities are reported in relation to PM₁₀ exposure, with 20.3% of the most deprived decile residing within locations experiencing the highest 10% of PM₁₀ concentrations, compared to only 2.0% of the country's most affluent decile experiencing such burdens (DEFRA 2006). Furthermore, the relationship between deprivation and exposure would appear most prolific across the 0-14y age group, with population-weighted PM₁₀ exposures per child of 29.1µg/m³ and 22.8µg/m³ experience by England's most deprived and affluent demographics respectively (DEFRA 2006).

In the preceding chapter, I explored the influence of several socio-environmental factors on the complete respiratory burden experienced by children, applying spatial modelling techniques to incorporate the social and physical structures present within an urban environment. The findings indicated significant global relationships to exist between children's hospitalisation rates and social-economic-status, ethnic minorities, and PM₁₀ road-transport (TPM₁₀) emissions within Leicester. Furthermore, Local Indicators of Spatial Association (LISA) and Geographically Weighted Regression (GWR) models identified several important localised variations within the dataset, specifically relating to a double-burden of residentially experienced road-transport emissions and deprivation increasing inner-city children's respiratory cases. Such findings are summarised within a research article published in *Science of The Total Environment* (Jephcote & Chen 2012). Prior to this, 'Environmental Justice' studies had rarely tackled the adverse health implications of exposures from mobile sources (Chakraborty 2009), or had applied statistical techniques appropriate for spatial health datasets (Gilbert & Chakraborty 2011).

Nevertheless the description of geographic phenomenon, often involves a somewhat naive and subjective selection of weighting structures, potentially constructing models that are unable to capture the underlying spatial interactions in an appropriate form. "The problem is that, unlike the simple notion of a time series lag, the spatial lag is a very fluid and complex entity open to multiple definitions within a single study" (Arbia & Fingleton 2008). Yet, LeSage & Pace's (2010) recent in-depth exploration of how to account for spatial structures dispels such universally held beliefs, through demonstrating that the sensitivity of estimates and inferences over a moderate range of spatial weighting structures provides a negligible impact on modelled outcomes.

For a generated dataset of 1,000 observations, levels of correlation between first order row-stochastic weighting schemes were observed to range from 0.37-0.72, yet effects estimates continued to exhibit high levels of correlation (0.92-0.96) across schemes containing 5-30 nearest neighbours (LeSage & Pace 2010). Furthermore, estimates and inferences of both Spatial Auto-Regression (SAR) and Spatial Durbin Model's (SDM) demonstrated a level of collectively stability in analysing influences on voter turnout across 3,107 counties US counties, across weighting schemes constructed from several neighbour choices and distant decay variables. In conclusion, LeSage & Pace (2010) dismiss the necessity of fine-tuning spatial weight scheme, placing greater emphasis on a well-specified spatial model. Such conclusions would appear to be confirmed within Chapter 4, by GWR models examining the influence of spatially dependent parameters on respiratory admissions. Whereby effect estimates generally appeared of a stable nature within the statistically critiqued threshold of appropriate weighting schemes.

Wall (2004) identified potential topics of further interest to this subject matter, in her extensive exploration of the covariance structures used to define elements of spatial dependence, within SAR and Conditional Autoregressive (CAR) models. Whilst implied correlation between a pair of neighbouring areas was negatively associated to the number of neighbours, this relationship appeared complex with much variability left unexplained, existing in a non-uniform manner. For these global spatial data models, Wall (2004) concluded that the implied spatial correlation between different sites using the SAR and CAR models did not seem to follow an intuitive or practical scheme; with such issues to be addressed through direct geostatistical (rather than artificial) modelling of the underlying spatial structures. Once more, such findings favourably lend themselves towards a continued application of GWR modelling practices, constructing an optimum local weighting structure for each individual observation.

It is intended that this chapter will expand upon ones initial discussion of the disparities in children's overall respiratory cases (J00-99), through exploring the extent to which socio-environmental influences sway the development of specific respiratory conditions. This would appear to be a timely question, considering that residential exposures to traffic pollutants (≤ 500 m from freeways) have been recorded to impede lung development, reducing a child's Forced Vital Capacity (FVC) by -63 ml over an 8-year period (Gauderman et al 2007). Furthermore a UK study of 3,911 women aged 60-79 years, revealed a range of social and material influence experienced during childhood to negatively impact adult FEV₁ rates, after adjusting for lifestyle choice (Lawlor et al 2004). In particular this chapter will explore whether a spatial relationship exists between relatively minor and relatively severe respiratory conditions, and if so, then to what extent do socio-environmental mechanisms play in the

decline of children’s respiratory health within Leicester. Spatially appropriate modelling procedures, accounting for underlying geographical structures at a common spatial resolution, will define the extent to which socio-environmental variables of interest individually influenced relatively minor and severe respiratory complaints during childhood via shared pathways.

Classical correlation procedures were initially conducted across the subset conditions accounting for >10% of children’s respiratory hospitalisations (ICD-10:J00-99) across Leicester UA, in order to determine whether communities face a multitude of respiratory issues affecting their overall quality of life. Table 5.1, identifies a tendency for different respiratory conditions to have poor spatial associations, with the exception of strong linear connection between the respiratory subsets J00-06 and J20-22 (Pearson’s R = 0.80). The compositions of these two subsets are of notable importance, with J00-06 recording acute Upper Respiratory Tract Infections (URTI) [i.e. common cold, acute sinusitis and acute tonsillitis etc.] and J20-22 defining the acute Lower Respiratory Tract Infections (LRTI) of bronchitis and bronchiolitis. The strong spatial ties between these respiratory issues likely infer a common causality to exist between relatively severe and mild respiratory complaints (Appendix D1). Therefore, the focus of this chapter is to examine to extent to which socio-environmental mechanisms play in the decline of respiratory health, as denoted by the respiratory subsets J00-06 and J20-22.

	Test	ICD10:	ICD10:	ICD10:	ICD10:
ICD10: J00-06	Pearson Correlation				
	Spearman's Rho				
ICD10: J20-22	Pearson Correlation	0.80**			
	Spearman's Rho	0.66**			
ICD10: J30-39	Pearson Correlation	0.20**	0.22**		
	Spearman's Rho	0.40**	0.33**		
ICD10: J40-47	Pearson Correlation	0.24**	0.30**	0.25**	
	Spearman's Rho	0.39**	0.41**	0.33**	

*P ≤0.05, **P ≤0.01

TABLE 5.1: Correlation of ICD-10 respiratory subset conditions accounting for >10% of children’s respiratory hospitalisations, across Leicester UA: 2000-09

5.2. EXPLORATORY SPATIAL DATA ANALYSIS (ESDA)

5.2.1 SPATIAL AUTOCORRELATION OF CHILDHOOD RESPIRATORY INFECTIONS

Global Moran’s I coefficients of spatial autocorrelation (Moran 1948), were initially applied to examine the existence of spatial autocorrelation within the J00-06 and J20-22 respiratory subsets at a citywide scale (Appendix D2). As within Chapter 4, global autocorrelation was

explored across several row standardised contiguity weighting schemes, the first of which placed weighting solely on first order neighbours (1 Queens Ring), with later tests placing weights up to and including sixth order observations (6 Queens Rings).

Global Moran's I values of 0.40 ($P < 0.01$) and 0.35 ($P < 0.01$) under a first order weighting scheme, correspondingly indicate that moderate levels of positive spatial correlation exist for children's J00-06 and J20-22 respiratory admissions. For both respiratory subsets a rapid decrease in Moran's I coefficient values may be observed across larger contiguity weighting schemes, with I values flat-lining around 0.00 in tests accounting for fourth order neighbours onwards (Appendix D2). This would imply that predominantly localised clusters of hospital admissions likely exist across Leicester UA, spreading across observations separated by a distance band of up to 3-Queens Rings (approximately 1365m). Similar observations were identified for the Bivariate Global Moran's I test, depicting the overall spatial strength and direction of the relationship between the two respiratory subsets.

However, Moran's I values alone can only infer a possibility of spatial autocorrelation to exist. A more accurate measurement can be obtained via Monte Carlo simulations, to produce a comparable reference distribution, from which a pseudo significance level is computed. For a significance level of 0.05, the derived Z-Score would have to be less than -1.96 (dispersed) or greater than 1.96 (clustered). Z-Score's from the Univariate and Bivariate Global Moran's I tests indicate significant spatial clustering to exist, when placing weighting on observations separated by a third order distance or lower (Appendix D2). Uniquely for the J20-22 subset, significant clustering albeit at a far weaker magnitude appeared to resurface in tests placing weighting on distant localities. While multi-distant spatial clustering is present, it would appear that this secondary cluster effect has a relatively low influence on the spatial determination of this phenomenon, as portrayed by the relatively insignificant Moran's I values of distant weighting schemes. A plausible explanation for such distant clustering effects, is offered and discussed later upon exploring localised elements of spatial autocorrelation.

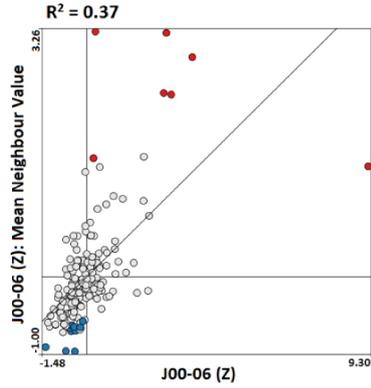
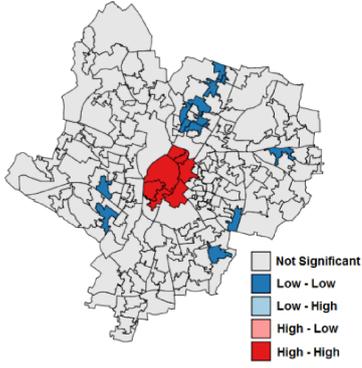
From these combined global measures, it would appear that relatively mild and severe cases of children's respiratory infections are location specific within Leicester UA, with the radius of spatial dependence following a rapid exponentially decaying relationship across a distance of approximately $<1365\text{m}$. Although it should be stated that noteworthy levels of spatial dependency were only detected in relation to first order neighbouring LLSOAs (450m), as indicated by respective second order J00-06 and J20-22 Global Moran's I values of 0.17 ($P < 0.01$) and 0.12 ($P < 0.01$). As previously discussed in Chapter 4, all of the primary socio-environmental factors of interest would appear to share a strong sense of location dependency with proximal LLSOA communities. However their range of influence would

typically be expressed over a wider scale as illustrated by moderate levels of spatial correlation (0.3 to 0.6) remaining amongst third order observations for measures of obesity prevalence and deprivation (1350m). Extending upon such distances, moderate levels of correlation were observed to persist within fourth order observations for levels of smoking prevalence, TPM₁₀ emissions and 'White British' Children (1820m).

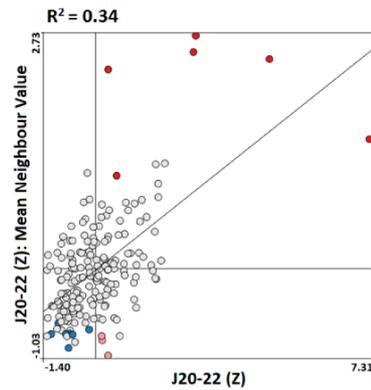
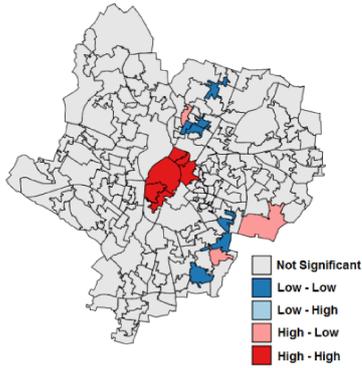
Concerning Leicester's key ethnic minority groups, residential levels of 'Indian' and 'Other South Asian' children decline in a linear fashion with distance, maintaining a moderate degree of correlation with observations separated by distances of up to four orders (radius $\geq 1820\text{m}$). Nevertheless, while such lifestyle, social and environmental influences appear to smoothly share common characteristics across wide areas of Leicester UA, it should be recognised that a considerably greater magnitude of correlation occurs across directly adjacent LLSOA communities. It is this first order correlation that is of particular importance, when investigating the presence of an association between children's respiratory infection rates, which exclusively operate across localised bands of distance. Meanwhile children of 'Afro-Caribbean' ethnicity would appear to favour residing within very specific locales of the city ($< 450\text{m}$), with residency levels plummeting in a manner comparable to the exponentially decaying relationships of children's respiratory infection rates.

While as a composite index, the Global Moran I coefficient is an informative measurement of the overall spatial clustering of the respiratory subsets, its assumption of spatial homogeneity and an inability to determine whether parameters dissipate in magnitude across locales of a mutual positioning, mean that it fails as an analytical tool of local variation. In dissecting the global coefficient, the Local Moran's I statistic (Anselin 1995) allows for a comprehensive location specific analysis of the dataset which complements its global counterpart, yet allows for one to unearth elemental pockets of collective interest. A Local Moran's I analysis of the J00-06 and J20-22 subsets, with weighting placed solely upon first order neighbours, identified mutual clustering of elevated hospital admission rates across inner city districts (Figure 5.1), akin to what was observed with the overall respiratory burden (J00-99). LLSOAs contained within the J00-06 and J20-22 hot-spots respectively experienced annual average admission rates of 42.43 and 18.25 cases per 1,000 children. In contrast, LLSOAs outside of the inner city centre hotspots experience hospitalisation rates 1.7 and 1.9 times lower for the J00-06 (15.82 per 1,000 children) and J20-22 (6.30 per 1,000 children) respiratory subsets.

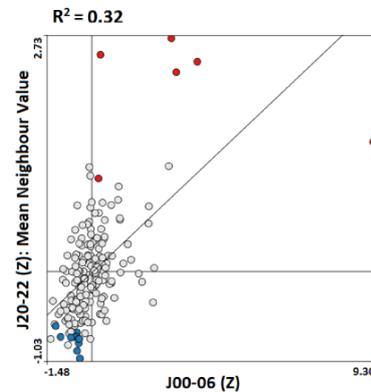
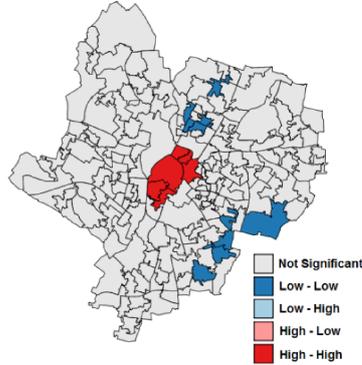
Univariate Local Moran's I:
J00-06 Admissions Per 1,000 Children



Univariate Local Moran's I:
J20-22 Admissions Per 1,000 Children



Bivariate Local Moran's I:
(i) J00-06 (j) J20-22 Admissions Per 1,000 Children



Bivariate Local Moran's I:
(i) J20-22 (j) J00-06 Admissions Per 1,000 Children

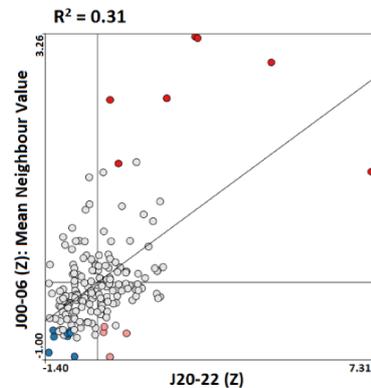
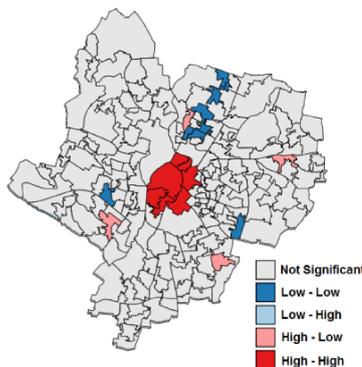


FIGURE 5.1: Local Moran's I cluster and outlier analysis of 2000-09 annual average children's URT (ICD-10: J00-06) and LRT (ICD-10: J20-22) infection hospital admissions

For the J20-22 subset, three High-Low (H-L) outliers are to be observed (Figure 5.1). These outliers are thought to be responsible for the minor clustering effect observed between distant localities (Appendix D2), due to these peripheral LLSOAs classifying inner-city localities found within the J20-22 hot-spot, as neighbours for weighting schemes >5 Queens Rings. The northern H-L outlier, situated within Belgrave, was identified to experience 7.22 admissions per 1,000 children, a rate 0.9 times higher than the mean of its first order neighbours. Within this LLSOA, a Carstairs Index score of 2.92 and residentially experienced TPM₁₀ levels of 1.61t/yr. appear marginally above their respective approximations of 2.42 and 1.03t/yr., recorded at first order neighbours.

Interestingly this is the same northern outlier identified in relation to the overall respiratory burden (J00-99), thought to be caused by a bottleneck in the flow of traffic along a primary radial corridor (A47: Melton Road) during peak hours (Appendix C2). Leicester's Local Transport Plan for 2011-2026 also recognises this particular corridor as a key problem area, frequently experiencing delays via buses and general traffic. Such localised areas of congestion and their associated increase in traffic emission levels appear to have gone undetected by the 1x1km resolution modelled traffic flows of the NAEI PM₁₀ road-transport emission dataset. Furthermore, only 16.76% of children within the community are of White British ethnicity, compared to levels of 23.91% in adjacent neighbourhoods. In line with the previous chapter, it is likely that the lifestyle choices of certain ethnic minorities are partially responsible for avoiding such respiratory outcomes.

A second H-L outlier appears within the district of Knighton, situated towards the South-eastern periphery of Leicester. Here an admission rate of 7.88 per 1,000 children is approximately 1.9 times greater than that of its adjacent neighbours. In terms of LLSOA characteristics, 68.60% of the children are of 'White British' ethnicity, with residents experiencing Carstairs Index scores of -6.03 and TPM₁₀ levels of 0.78t/yr. These socio-environmental factors are closely mimicked by neighbouring localities, which contain only 3.67% more children of 'White British' ethnicity, record mean Carstairs Index values of -6.37, and experience only 0.02t/yr. less TPM₁₀. Furthermore, the structures of ethnic minority groups and overall health of communities also appear comparable, and offer no solution to areas respiratory concerns. Such issues of non-stationarity likely exist because of community specific contextual factors causing spatially differing responses to the same stimuli. Community specific factors may also be responsible for the third H-L cluster located in Evington towards the cities eastern periphery, which akin to its neighbours experiences relatively low Carstairs Index (-3.47) and TPM₁₀ levels (0.52t/yr.). Notable social differences compared to neighbouring locations, include a decrease in children of 'Indian' (-16.44%) and

'Other South Asian' (-5.97%) ethnicities, and an increase in children of White British ethnicity (+6.47%). One possible explanation is that the lifestyle choices of some ethnic minority groups may be associated with good respiratory health. Once again, community specific contextual factors are also likely at play.

Bivariate Local Moran's I analysis of the J00-06 and J20-22 subsets, with weighting placed solely upon first order neighbours, would appear to confirm the existence of a strong spatial tie between children's infections of the upper and lower respiratory tracts. When the URTI subset is held at ego locations (J00-06), one may observe that inner-city locales are exclusively encased by communities whom also experience elevated levels of childhood LRTI's (J20-22). Furthermore, LLSOAs experiencing low levels of URTI's would also appear to be surrounded by communities experiencing reduced LRTI rates along the affluent south-eastern peripheral suburban wards of Knighton and Evington (Figure 4.2), and within the northern wards of Latimer and Belgrave. Such northern wards are positioned just outside of the Melton Road's (A47) culturally diverse business district, in locales appearing to favour the housing of 'Indian' residents (Figure 4.6), whose lifestyle choices were previously recognised to reduce the effect of unfavourable health burdens.

In holding the LRTI subset at ego locations (J20-22), one may conclusively observe that the same inner-city locales also experience elevated levels of childhood LRTI's (J20-22). As reported in Chapter 4, such neighbourhoods would appear to predominantly house certain ethnic minorities whom are forced to experience a 'double burden' of deprivation and pollution emitted from mobile sources. Once more LLSOAs experiencing low levels of LRTI's characteristically appear bordered by communities experiencing reduced URTI rates, across the northern wards of Latimer and Belgrave. However, one exception to this rule exists, as denoted by the J20-22 hot-spot which harbours a potential bottleneck restricting the flow of traffic along the Melton Road (A47). Children housed within this LLSOA would appear vulnerable to LRTI's, yet unusually bear an elevated level of resistance to experiencing URTI's. However, in both cases children's J00-06 and J20-22 admissions were respectively identified to be 0.26 and 0.43 times lower than the citywide average LLSOA rate, which would suggest that this communities respiratory health is of a relatively high standard. Interestingly the eastern outlier of overall respiratory burdens (J00-99), positioned around a terminal link of the outer ring-road which funnels traffic towards Leicester's central districts, is also deemed to house children whom are vulnerable to LRTI's yet are somewhat resistance to URTI's. Within this particular outlier a J00-06 rate of 15.53 and J20-22 rate of 73.01 admissions per 1,000 children, were respectively identified to only differ from citywide average LLSOA rates by -1.29 and +0.62 admissions.

The two other LLSOA outliers recording high LRTI's whilst positioned adjacent to communities experiencing reduced URTI rates, would also appear to be situated alongside key arterial roads linking the cities inner and outer ring-roads. The eastern outlier contained within the ward of Knighton, experiences a J00-06 rate of 13.28 and J20-22 rate of 7.88 admissions per 1,000 children, which are respectively identified to be 0.21 times lower and 0.18 times higher than the citywide average LLSOA rate. It should be noted that this particular LLSOA contain the University of Leicester's campus, therefore one might expect that localised congestion to be partially responsible for such elevated LRTI's. Meanwhile the western outlier contained within the ward of Braunstone, was observed to contain a plethora of educational facilities including Ellesmere College, Fullhurst Community College and St Marys Fields Infant School. Here, J00-06 and J20-22 rates of 14.87 and 9.59 admissions per 1,000 children, are correspondingly recorded as 0.12 times lower and 0.43 times higher than their citywide averages. Once more, it is highly plausible for elements of localised congestion to uniquely occur within this LLSOA across peak hours of the network. One may conclude, that whilst elevated cases of both LRTI and URTI appear to coincide with polluted zones of the city, it has become apparent that LRTI's share a heightened sensitivity to extremely localised spatial-temporal periods in which elevated TPM_{10} emission exposures are residentially experienced. In contrast, URTI's appear to thrive in locales where moderate-high socio-environmental influences remain constant.

5.2.2. NUMERICAL COMPARISON OF SPATIAL EXTREMES

A direct comparison of hot-spots and cold-spots contained within the J00-06 and J20-22 Univariate Local Moran's I outputs, indicate some potentially interesting socio-environmental factors which are likely to reciprocally influence the onset of URT and LRT infections in children (Appendix's D3-D6).

J00-06 LLSOA hot-spots are on average recorded to experience 42.43 J00-06 admissions and 17.60 J20-22 admissions per 1,000 children, which respectively correspond to such areas experiencing 2.50 and 2.72 times more cases than their cold-spot counterparts (Appendix D3). Several socio-environmental factors were observed in hot-spot locations to be noticeably above their cold spot counterparts, including residentially experienced TPM_{10} (+1.24t/yr.), Carstairs Index scores (+4.47), smoking prevalence (+14.76%) and levels of obesity (-4.60%). In exploring the distribution of Leicester's major ethnic minority groups, hot-spot communities were recorded as housing substantially more children of 'Afro-Caribbean' (+11.42%) and 'Other South Asian' ethnicities (+6.00%), and markedly fewer children if 'Indian'

ethnicity (-25.82%). In regards to magnitude, such values portray J00-06 hot-spots as housing 11.53 times more 'Afro-Caribbean' residents, 1.08 times more 'Other South Asian' residents, and 1.03 times fewer 'Indian' residents. This would reinforce the earlier notion, which stated that the lifestyle of certain ethnic minority groups has a beneficial impact on adverse respiratory outcomes. It should be also noted that whilst levels of 'White British' children appeared to decline across cold-spots (-4.46%), this ethnic group is generally associated with relatively average-good levels of respiratory health; as denoted by areas of no significant spatial patterning predominantly housing such children (56.19%). Rather it would appear that as levels of 'Indian' residency rises, then levels of 'White British' residents decline (Figure 4.6).

Likewise, J20-22 LLSOA hot-spots are on average experienced 43.13 J00-06 admissions and 18.25 J20-22 admissions per 1,000 children, which respectively correspond to 3.08 and 3.86 times more cases than what was expected within their cold-spot counterparts (Appendix D4). Whilst no substantial differences in obesity appeared to exist between such extremes, levels of TPM₁₀ (+1.26t/yr.), Carstairs Index scores (+5.76) and smoking prevalence (+17.71%) once again appeared elevated in hot-spot communities. Such incremental increases in these levels of detrimental socio-environmental influences upon what was recorded by J00-06 hot-spot locales, likely explain why respiratory infections in J20-22 hotspots diverge further from their cold-spot locales.

As before, J20-22 hot-spots were observed to predominantly house ethnic minorities, of which there are 12.59 times more 'Afro-Caribbean' residents, 0.57 times more 'Other South Asian' residents, and 1.08 times fewer 'Indian' residents. Interestingly, Univariate J20-22 outlier locations exhibit comparative J00-06 rates to those recorded at their cold-spot locations, whereas J20-22 are recorded as 0.99 times higher at a rate of 7.46 cases per 1,000 children. Yet, inhabitants of these J20-22 outliers experience levels of smoking prevalence, TPM₁₀ emissions and deprivation below what is recorded in cold-spot LLSOAs. Furthermore, such areas house 11.07 times fewer 'Afro-Caribbean' residents, and 0.42 times fewer 'Indian' residents. As previously discussed, it is most likely that LRTI's share a heightened sensitivity to rare localised spatial-temporal episodes of elevated TPM₁₀ exposures, which have remain undetected from the 1x1km resolution NAEI emission database.

5.2.3. BIVARIATE CORRELATION OF SOCIO-ENVIRONMENTAL INFLUENCES & HEALTH

Traditional dataset correlation tests were conducted to statistically determine whether relationships between individual socio-environmental influences and cases of children's URT

(J00-06) and LRT (J20-22) infection hospitalisations, exist within Leicester's LLSOA communities (Table 5.2).

For the J00-06 subset, Pearson's R-values identified several important linear correlations to exist between children's hospitalisations and deprivation ($R=0.34$), TPM_{10} emission ($R=0.47$) and residents of 'Afro-Caribbean' ethnicity ($R=0.39$), with significance at the 99% confidence level. Meanwhile moderate levels of non-linear correlation were also observed to occur between J00-06 admissions and levels of 'Indian' children ($Rho=0.31$) and smoking prevalence ($Rho=0.43$), as denoted by Spearman's Rho values recorded at the 99% confidence level. In all cases, similar relationships were reported when exploring the overall respiratory burden experienced by children (J00-99). However it would appear that URTI infections have a weaker associations with deprivation (-0.06), 'Indian' residency (-0.07) and levels of smoking prevalence (-0.04), while having a stronger dependency on TPM_{10} emissions ($+0.10$) than what was accounted for by the complete respiratory set.

For the J20-22 subset, Pearson's R-values observed weaker but still important linear correlations to exist in relation to TPM_{10} emissions ($R=0.38$) and residents of 'Afro-Caribbean' ethnicity ($R=0.34$), while stronger ties were reported in relation to deprivation ($R=0.38$), all at the 99% confidence level. Meanwhile moderate levels of non-linear correlation were significantly ($P \leq 0.01$) observed to occur between J20-22 admissions, 'Indian' residency ($Rho=0.46$) and levels of smoking prevalence ($Rho=0.56$). Furthermore, it would appear that such non-linear influences have a stronger association to LRTI's than what was reported for URTI's.

As Chapter 4 previously reported, Carstairs Index values of deprivation across Leicester were observed to solely yield significant linear correlations of interest with TPM_{10} emissions ($R=0.40$), obesity rates ($R=0.51$), and community levels of 'Afro-Caribbean' children ($R=0.30$). Additional correlations were observed in a linear format between levels of TPM_{10} emissions and residents of 'Afro-Caribbean' ($R=0.42$) and 'Other South Asian' ($R=0.43$) ethnicities. Meanwhile children of 'Other South Asian' origins shared a strong non-linearly correlation with 'Indian' children ($Rho=0.73$), yet followed a linear correlation with levels of 'Afro-Caribbean' residency ($R=0.35$). Such correlation statistics would appear to broadly compliment the aforementioned outputs of the Local Moran's I statistics.

	Correlation Statistic	J00-06	J20-22	Carstairs Index	TPM ₁₀	Smoking Prevalence	Obesity Prevalence	White Non-British	Indian	Other South Asian
J00-06 Per 1,000 Children	Pearson R									
	Spearman's Rho									
J20-22 Per 1,000 Children	Pearson R	0.80**								
	Spearman's Rho	0.66**								
Carstairs Index (Leicester)	Pearson R	0.34**	0.38**							
	Spearman's Rho	0.33**	0.36**							
TPM₁₀ Emissions	Pearson R	0.47**	0.38**	0.40**						
	Spearman's Rho	0.18*	0.11	0.37**						
Smoking Prevalence	Pearson R	0.27**	0.45**	0.33**	0.04					
	Spearman's Rho	0.43**	0.56**	0.31**	0.00					
Obesity Prevalence	Pearson R	-0.02	0.06	0.51**	-0.27**	0.35**				
	Spearman's Rho	0.21**	0.25**	0.51**	-0.20**	0.46**				
White Non-British Children	Pearson R	0.07	0.15*	-0.12	0.11	0.06	-0.27**			
	Spearman's Rho	0.08	0.17*	-0.15*	0.05	0.06	-0.18*			
Indian Children	Pearson R	-0.21**	-0.36**	0.17*	0.17*	-0.68**	0.01	-0.24**		
	Spearman's Rho	-0.31**	-0.46**	0.06	0.22**	-0.75**	-0.24**	-0.16*		
Other South Asian Children	Pearson R	0.24**	0.02	0.29**	0.43**	-0.41**	-0.14	-0.08	0.52**	
	Spearman's Rho	0.07	-0.14*	0.22**	0.36**	-0.50**	-0.25**	-0.08	0.73**	
Afro-Caribbean Children	Pearson R	0.39**	0.34**	0.30**	0.42**	-0.04	-0.03	0.25**	-0.01	0.35**
	Spearman's Rho	0.26**	0.15*	0.16*	0.28**	0.01	-0.14	0.12	0.13	0.31**

Significance Levels: *P ≤0.05, **P ≤0.01

TABLE 5.2: Traditional linear (Pearson's R) and non-linear (Spearman's Rho) dataset correlations of children's respiratory infections and socio-environmental influences, experienced by residents of Leicester UA: 2000-09

Bivariate Local Moran's I statistics were initially conducted in a manner, which held children's J00-06 admissions at ego locations (i) and placed individual influences of interest at neighbouring LLSOAs (j). Test outputs therefore display the spatial relationship between respiratory cases and surrounding social-environmental influences, recorded across first-order locations. In viewing the global summaries of the Bivariate Local Moran's I statistics (Table 5.3), one may observe two definitive relationships of particular spatial interest, appear to involve levels of TPM₁₀ emissions (R²=0.22) and 'Afro-Caribbean' residency (R²=0.32). To a lesser extent deprivation would also appear to influence children's URT hospitalisations, as indicated by a global coefficient I-value of 0.21. Nevertheless such spatial ties with socio-environmental influences appear to generally occur on a substantially weaker level to what was observed when examining the complete respiratory hospital burden (Table 4.2); with the exception of the TPM₁₀ emission bivariate model whose global coefficient was found to rise from 0.28 (J00-99) to 0.37 (J00-06). Unlike the local bivariate J00-99 models, community levels of smoking prevalence did not appear to share spatial ties to J00-06 admission rates.

	Bivariate Moran's I: (i) J00-06; (j) ...			Bivariate Moran's I: (i) J20-22; (j) ...		
	R ²	I Value	P-Value	R ²	I Value	P-Value
Carstairs Index (Leicester)	0.09	0.21	0.00	0.06	0.19	0.00
TPM₁₀ Emissions (t/yr.)	0.22	0.37	0.00	0.12	0.29	0.00
% Smoking Prevalence 16yrs+	0.05	0.17	0.00	0.13	0.29	0.00
% Obesity Prevalence 16yrs+	-0.00	-0.01	0.47	0.00	0.04	0.16
% 0-15y White Non-British	0.00	0.02	0.31	0.01	0.05	0.10
% 0-15y Indian	-0.01	-0.11	0.00	-0.05	-0.23	0.00
% 0-15y Other South Asian	0.05	0.18	0.00	0.00	0.00	0.53
% 0-15y Afro-Caribbean	0.32	0.26	0.00	0.15	0.18	0.00

TABLE 5.3: Summary of the Bivariate Local Moran's I analysis, revealing spatial associations between children's J00-06 or J20-22 admissions and surrounding socio-environmental influences

It would appear, as previously discovered, that elevated levels of deprivation encompass and inhabit LLSOAs within focal point of Leicester's inner-city J00-06 hot-spot (Figure 5.2). Raised levels of deprivation continue to prevail east of the inner-city hot-spot, yet such areas would appear to only resemble problem zones where interactions with environmental hazards occur. Meanwhile, relatively affluent areas towards the cities and eastern and periphery's, particularly around the Knighton district, would appear spatially associated with reduced levels of J00-06 respiratory symptoms. Likewise, substantial positive correlations between respiratory symptoms and TPM₁₀ emission levels appear to inhabit inner city LLSOAs, encompassed and adjacent to the inner city ring road and its northern arterial roads. However one may observe that TPM₁₀ emissions broadly encompass Leicester's central district, with

severe outbreaks of J00-06 admissions (Figure 5.2) occurring only in locales experience a 'double burden', which entails issues of deprivation.

Interestingly, LLSOAs recording moderate J00-06 admission rates within the eastern peripheral ward of Humberstone are deemed to be of a highly outlying nature, caused by their decidedly affluent portrayal and considerably low exposures to TPM_{10} under the provided emission inventory. It would appear that these outlying communities are situated along the north-easterly section of Leicester's outer-ring road prior to and inclusive of its terminal junction. During peak hours in the network, the terminal outer ring-road junction experiences periods of localised congestion (Appendix C2), which from this evidence is likely to influence traffic movements across the preceding outer ring-road exits. As previously discussed, such localise flows have evaded the 1x1km TPM_{10} emission grids, yet these excess emissions which have been smoothed out would appear to contribute to only a minor portion of the cities respiratory burden. Reduced J00-06 hospitalisation cases accompany insignificant TPM_{10} levels along Leicester's eastern periphery, are marked via a missing section of the outer ring-road which subsequently forces traffic to enter the heart of the city. In one aspect, the peripheral wards of Evington and Thurncourt may therefore be deemed to have unfairly shifted their allocated respiratory burdens onto central districts of the city. The same findings would appear to be individually observed for LRTI's (J20-22) and across the entire respiratory set (J00-99).

In terms of 'Afro-Caribbean' residency, one may observe that children of this ethnic group are associated in a spatial manner to inner-city communities experiencing severe URTI's. However, one exception to this rule exists in a north-easterly inner city LLSOA on the fringe of the 'double-burden' zone, which experiences reduced J00-06 admissions yet remains spatially connected to 'Afro-Caribbean' residencies. Fascinatingly, this is one of the communities on the inner-cities fringe, where the positive influence of 'Indian' lifestyles appears to provide a recognisable force in mitigating respiratory impacts. As discussed in Chapter 4, there is a possibility that other minority groups residing within the same community have partially adopted these beneficial social lifestyle choices. One may also observe that the southern peripheral ward of Eyres Monsell experiences moderate J00-06 admission rates, yet is strongly disassociated to 'Afro-Caribbean' residency levels. It would appear that this ward comprises of a 'White British' majority, and that raised URTI's are associated with issues of obesity and smoking, which appear to uniquely affect 'White British' children.

Bivariate Local Moran's I:
 (i) J00-06 Admissions Per 1,000 Children; (j) ...

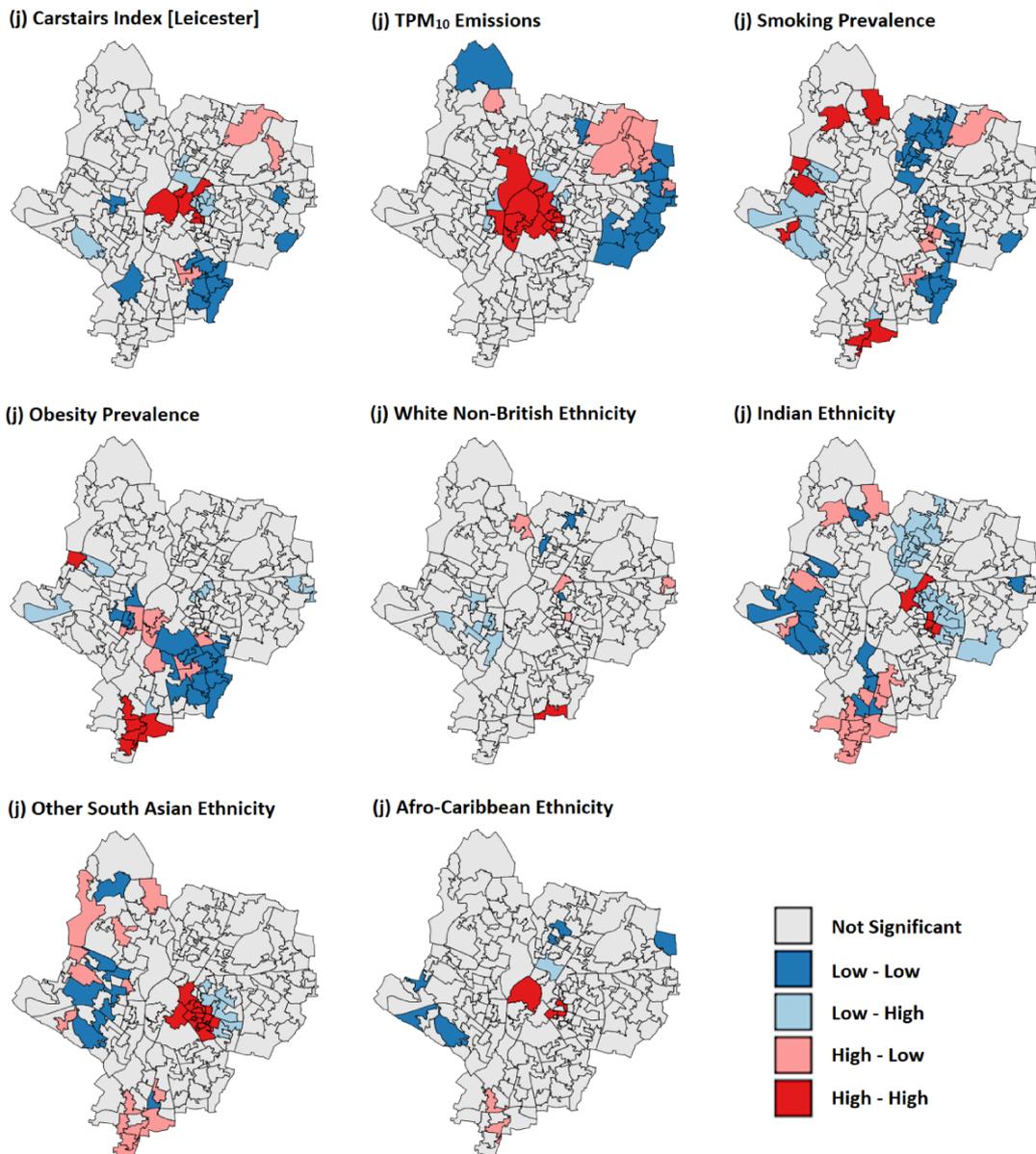


FIGURE 5.2: Bivariate Local Moran's I cluster and outlier analysis of 2000-09 annual average children's URTI admissions (ICD-10: J00-06), and neighbouring socio-environmental influences of interest

Bivariate Local Moran's I statistics were subsequently conducted in a manner, which held children's J20-22 admissions at ego locations (i) and placed individual influences of interest at neighbouring LLSOAs (j). In viewing the global summaries of the J20-22 Bivariate Local Moran's I statistics (Table 5.3), one may observe R^2 values as denoting substantially weaker relationships to what was observed by the J00-06 bivariate outputs. Upon viewing the actual tests summary coefficient of global autocorrelation, one may detect minor-moderate positive J20-22 connections occur in relation to deprivation (0.19), TPM_{10} emissions (0.29), smoking prevalence (0.29) and 'Afro-Caribbean' (0.18) residency. Minor-moderate negative associations

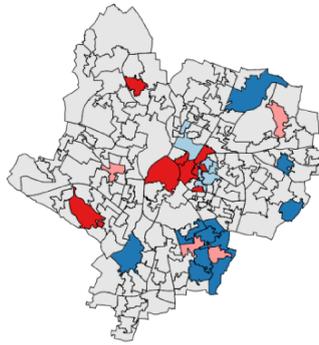
were found with 'Indian' (-0.23) residency levels. In all circumstances, the global summary coefficients were recorded to have weaker associations than what was recorded in the J00-06 bivariate tests, with the exception of variables recording 'Indian' residency and smoking prevalence.

Upon exploring the spatial distributions (Figure 5.3), it would once again appear that when elevated levels of deprivation and TPM_{10} emissions combine, they produce J20-22 burdens of a far greater magnitude than what would be produced by such components if they were to be assessed individually. Furthermore, upon comparing the spatial interactions of socio-environmental factors of interest independently on J00-06 and J20-22 outcomes, one may observe that localised pockets and trends of interest are broadly of a similar nature; despite the reduced global associations with J20-22 cases. Such observations would imply that certain socio-environmental mechanisms are perhaps culpable for a gradual decline in children's respiratory health, as denoted by the onset of URT and LRT infections across the City of Leicester.

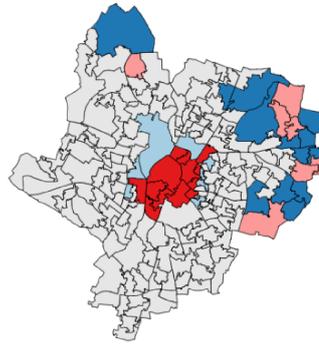
Nevertheless, minor differences between the positioning of URT and LRT infections in relation to deprivation are detected within a select few LLSOAs positioned across the cities western district. Previously under the J00-06 bivariate model, two of these communities were considered low health outliers, whereas in the J20-22 model such areas were classified to be deprived and experience comparatively higher admissions to other nearby locales. In addition, a Knighton community at the heart of the south-easterly J00-06 healthy-affluent pocket was found to experience J20-22 admission 0.18 times above the average LLSOA rates. This LLSOA is thought to represent localised congestion from the university campus at peak periods, and thus highlights the heightened susceptibility of LRTI's to air pollutants. Through comparing the models, one may confirm that 'Indian' social lifestyle choices provided a greater beneficial impact on J20-22 outcomes, as denoted by the replacement of J00-06 'Indian' residency cold-spots with J20-22 outliers along the cities western periphery. Such J20-22 outliers would also appear to explain the weakened association between 'Afro-Caribbean' residency levels, to what was previously recorded in relation to J00-06 incidents. It would also appear that healthy lifestyle choices including passive smoking and obesity specifically amongst 'White British' children have a greater say in LRT rather than URT infections.

Bivariate Local Moran's I:
(i) J20-22 Admissions Per 1,000 Children; (j) ...

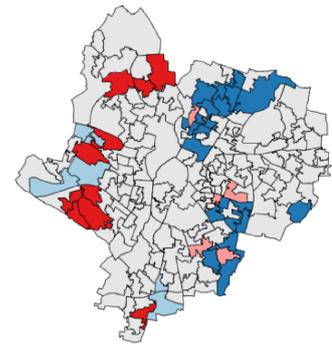
(j) Carstairs Index [Leicester]



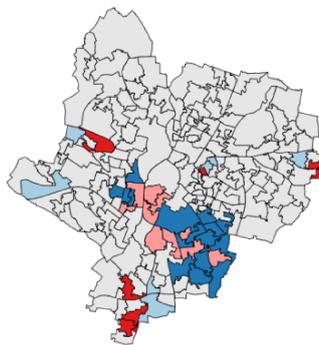
(j) TPM₁₀ Emissions



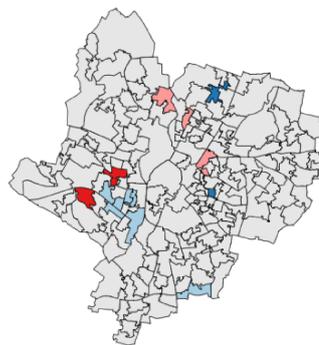
(j) Smoking Prevalence



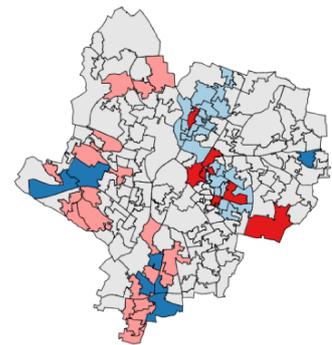
(j) Obesity Prevalence



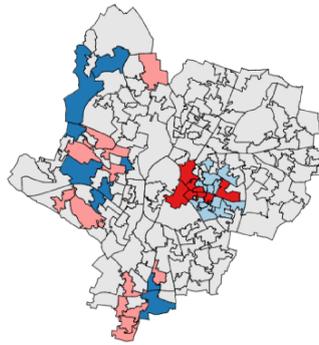
(j) White Non-British Ethnicity



(j) Indian Ethnicity



(j) Other South Asian Ethnicity



(j) Afro-Caribbean Ethnicity

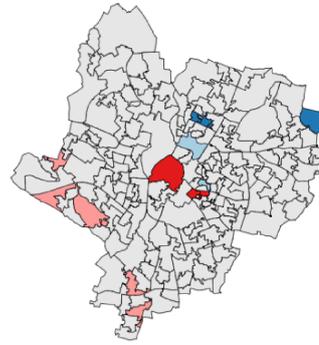


FIGURE 5.3: Bivariate Local Moran's I cluster and outlier analysis of 2000-09 annual average children's LRTI admissions (ICD-10: J20-22), and neighbouring socio-environmental influences of interest

5.3. SPATIAL MODELLING: GEOGRAPHICALLY WEIGHTED REGRESSION (GWR)

5.3.1. GWR MODEL VALIDATION

Ordinary Least Squares (OLS) regression models were initially applied to examine the concurrent effects of eight explanatory socio-environmental variables on children's URT and LRT infections across the period of 2000-09. Despite the global models misrepresentation of local conditions, such a procedure was nevertheless conducted in-order to summarise the relative importance of each attribute, whose interactions and likelihood, where of interest, may then be explored in a spatially detailed manner via GWR. While ANOVA F-tests indicated model significance at the 95% confidence level, the R-Squared values for the J00-06 (0.37) and J20-22 (0.42) OLS models portrayed only a moderate goodness-of-fit to the data. The J00-06 OLS model residuals were identified to have significant spatial clustering ($P \leq 0.05$), whereas the J20-22 OLS model residuals were neither significantly dispersed nor clustered. This lack of dispersion amongst regression model residuals in conjunction with earlier Local Moran's plots indicating a clustering of dependent variables within inner city localities, reinforce the application of GWR models to account for the spatial nature of the dataset. Furthermore, the presence of spatial autocorrelation within the residuals is considered a violation of one of the fundamental assumptions of OLS models (Longley & Tobon 2004, Ibeas et al 2012).

As previously discussed, GWR models constructed with a sharp cut-off bandwidth scheme, placing weight on a few proximal observations, may offer a near perfect fit but at a cost of increased model complexity. In practice, the simplest model is preferred if the later offers little improvement, as a result there must be always be a trade-off between bias and variance when selecting an appropriate GWR weighting scheme. In addressing such issues, I propose a four point scheme to assist future researchers in selecting an optimally weighted GWR model:

- I. Three generalized degree of freedom ANOVA F-tests (Fotherington et al 2002, Leung et al 2000) should collectively identify GWR models as significantly improving upon the Residual Sum-of-Squares (RSS) produced from their corresponding OLS model. To establishing whether improved model performance has occurred in the presence of increased model complexity.
- II. AIC and AICc tests describing the relative goodness-of-fit in relation to the degrees of freedom, when acceptable should yield GWR test values preferably of a lower, or similar nature to their OLS model counterpart.
- III. Residuals should be spatially dispersed in a significant manner, as indicated by the Global Moran's I Z-Score. Thus indicating that the study areas spatial processes have been adequately captured.

- IV. The cross-validation R-Square value ($CV-R^2$) of predicted model outputs vs. 2004-06 admission rates must not substantially differ from the GWR R^2 value, in order to certify overall model robustness.

Basic GWR model performance tests were applied to weighting schemes ranging from 180NN to 20NN, and are summarised in the graphical format of Figure 5.4. Global Moran's I tests examining the spatial distribution of residuals, identify all of the explored GWR scheme residuals as displaying no significant elements of geographical clustering ($Z\text{-Score} \leq 1.96$). However, significantly dispersed residuals were observed for both respiratory infection subsets when applying weighting schemes, which explored fewer than 60NN's ($P \leq 0.05$). Such test outputs would suggest that a satisfactory calibration of the datasets spatial components has been achieved. AICc tests describing the relative goodness-of-fit in relation to the degrees of freedom after correcting for sample size, identify the arrival of model complexity issues at a 50NN weighting scheme, with such issues becoming a major concern for weighting schemes less than 30NN.

A Cross-Validation (CV) of GWR model predictions with 2004-06 respiratory admission rates, detect a satisfactory level of performance when a weighting of 70NN or fewer is used to investigate the J00-06 respiratory subset ($R^2 \geq 0.5$). For the J20-22 subset the optimum weighting scheme appears to be around 50-40NN as indicated by no further improvements in $CV-R^2$ values. Ratio scores combining the AICc and $CV-R^2$ values, indicate optimum model performance at 50NN for the J20-22 subset, whereas an optimum weighting window of 30-50NN was observed for the J00-06 subset. Further analysis of candidate GWR weighting schemes, was subsequently focused around the 50NN mark.

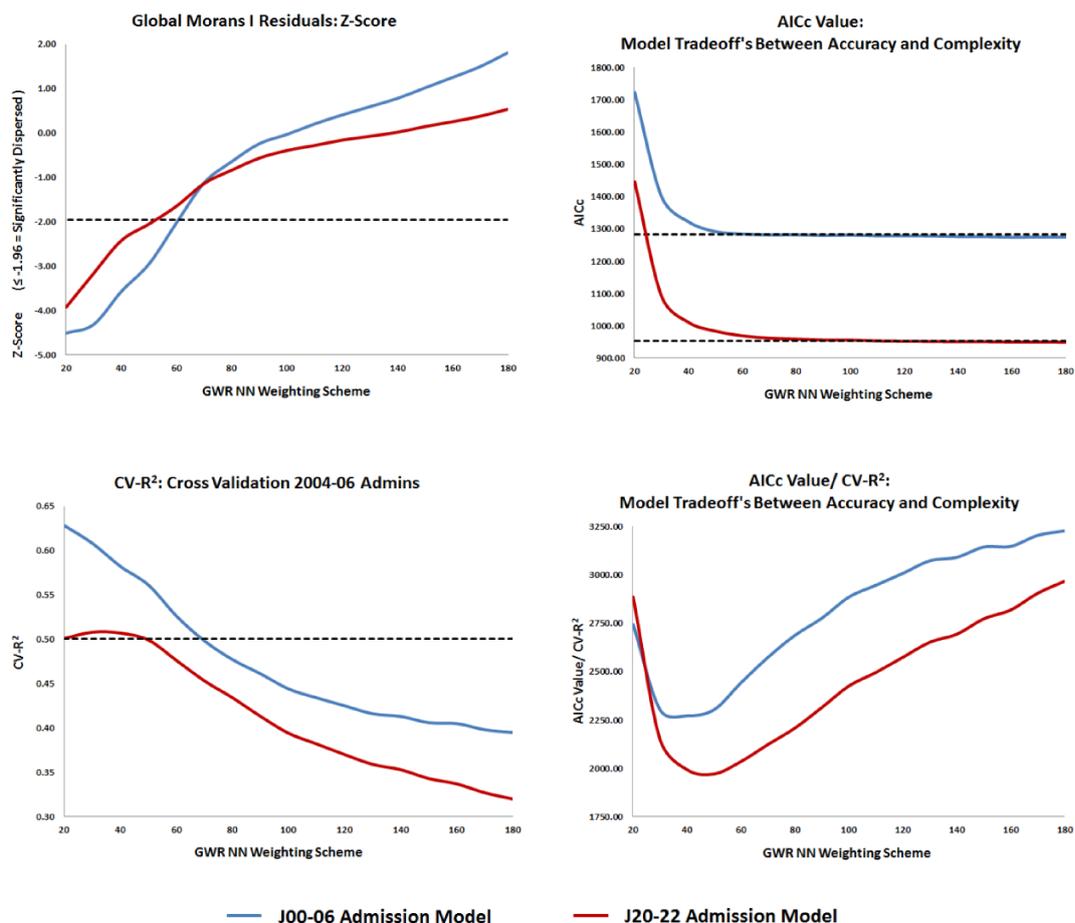


FIGURE 5.4: Statistical evaluation of the J00-06 and J20-22 GWR spatial schemes

For the respiratory subset J00-06, GWR models with weighting schemes placed upon the 60 Nearest Neighbours (NN), 50NN and 40NN were identified to pass the three GWR F-tests, indicating improved model performance over OLS models in relation to modelling complexity (Appendix D7). For the respiratory subset J20-22, only the 50NN and 40NN GWR models produced significant GWR F-test values. To effectively cross-examine the extent to which socio-environmental mechanisms play in the decline of respiratory health, J00-06 and J20-22 GWR models should share weighting schemes in-order to directly compare the influence of independent variables across a common spatial resolution. Under this premise, the 50NN and 40NN weighting schemes were explored in greater detail to confirm the most suitable option.

The AIC scores corrected for observation sample size (AICc), identify small percentage dissimilarity's between J00-06 GWR and OLS models on weighting schemes placed up to 50NN (+0.71%). For the J20-22 subset, dissimilarities between GWR and OLS models are seen to rapidly increase for models with fewer weighted observations; as observed under the 50NN (+3.09%), 40NN (+5.92%) and 30NN (14.41%) schemes. Cross-validation of J00-06 and J20-22 GWR models with 2004-06 admission rates met critical CV-R² values of ≥0.50, for both the

50NN and 40NN weighting schemes. For the J20-22 subset GWR discrepancies (%) between GWR model R^2 and CV- R^2 values appeared to exist at a stable level from 80NN (29.09%) up until the 50NN (30.15%) weighting scheme, after which a sharp decline can be noticed indicate potential model over-fitting. On this basis and under the constraints of minimising the AICc score, a weighting scheme of 50NN was deemed most appropriate for exploring the influence of socio-environmental influences on the J00-06 and J20-22 subsets.

On a final validatory point, it has been noted that spatial modelling techniques have previously paid only a limited amount of attention to standard diagnostic techniques, especially relating to issues of multicollinearity. In a paper addressing these concerns, Wheeler & Tiefelsdorf (2005) identified a potential for local regression coefficients to yield collinear relationships, even when variables in the data generating process appeared uncorrelated. In numerous cases, coefficient estimate movements in one direction were demonstrated to force at least one coefficient in the other direction, thus invalidating any interpretation of individual GWR parameter estimates. Issues of multicollinearity for the J00-06 and J20-22 Bi-square Adaptive 50NN GWR models were explored through the Variance Inflation Factor (VIF) diagnostic tool within SPSS 20.0. VIF values <5 were deemed to indicate no collinearity issues, VIF's between 5 and 10 indicated moderate collinearity, and VIF's 10 or greater indicated a serious issues (Schuenemeyer & Drew 2011). For both respiratory subsets no issues of multicollinearity were detected amongst GWR coefficients, with VIF values respectively averaging 1.93 and 2.43 for the J00-06 and J20-22 models.

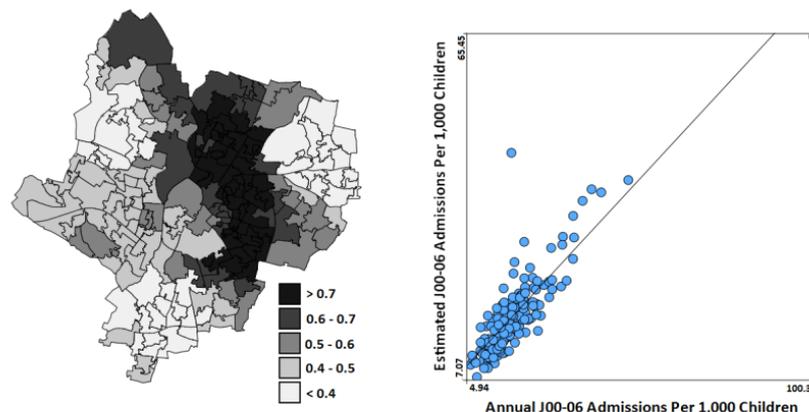
5.3.2. GWR MODEL PERFORMANCE

GWR models with a 50NN 'Bi-Square Adaptive' weighting scheme were subsequently constructed, to quantify and compare, the extent to which socio-environmental variables of interest locally influenced relatively minor and severe respiratory complaints during childhood. As previously discussed, adaptive kernels were favoured over a fixed distance spatial kernel scheme in-order to prevent issues of poor model calibration, caused by restricted data point counts in areas where LLSOAs are spatial expansive. The R-square scores for both the 50NN J00-06 (0.73) and J20-22 (0.72) subset GWR models symbolise a marked improvement upon their respective OLS regression models performances of 0.37 and 0.42. This improvement can be further observed through the corresponding 57.21% and 52.29% reductions on OLS mean squared errors values produced by the J00-06 and J20-22 subset GWR models.

The spatial distributions of local R-square values generated by the GWR analysis of the J00-06 and J20-22 subsets of interest are depicted in Figure 5.5. Geographic variations in these

values demonstrate how the combined statistical effect of the explanatory variables on upper and lower respiratory hospitalisations in the 0-15 year age group differs across output areas in Leicester. It may be observed that local regression models contained within the J00-06 and J20-22 GWR models, recorded respective improvements upon their specific global regression model R-square value across 82.89% and 70.59% of Leicester's output areas.

R²: J00-06 GWR 50NN Bi-Square Adaptive Scheme



R²: J20-22 GWR 50NN Bi-Square Adaptive Scheme

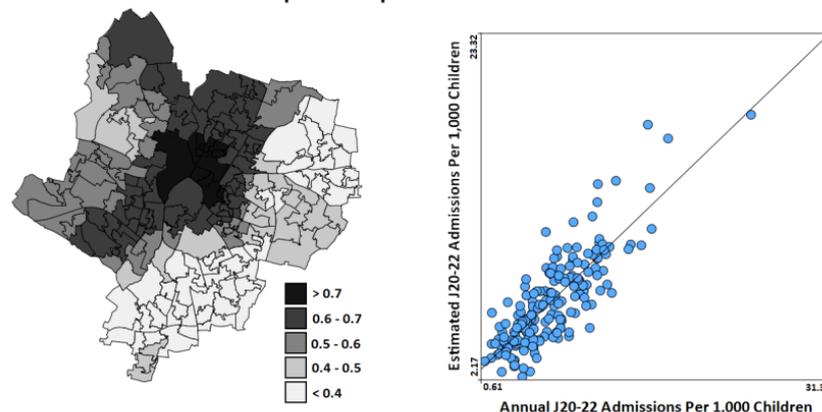


FIGURE 5.5: Local model goodness-of-fit scores contained within each GWR scheme

The strongest model performances for the J00-06 subset are to be found around the western portion of the city centre, across LLSOAs housing elevated levels of ethnic minority groups, fringing the URTI hot-spot of concern identified within the LISA analysis. In particular LLSOAs contained within the wider wards of Charnwood ($R^2=0.79$), Latimer ($R^2=0.73$), Coleman ($R^2=0.72$), Spinney Hills ($R^2=0.72$) and Stoneygate ($R^2=0.72$) were observed on average to provide optimum levels of performance. Within the J00-06 respiratory hot-spot of interest R-square values ranged from 0.46-0.74, with a coefficient value of 0.52 for the focal LLSOA of particular interest. Yet, towards the eastern and to a lesser extent the southern fringes of Leicester, respiratory hospital admission rates appear inadequately explained by the selected

explanatory variables. Specifically, LLSOAs contained within the wider wards of Humberstone ($R^2=0.21$), Thurncourt ($R^2=0.22$) and Aylestone ($R^2=0.30$) were observed to provide particularly low performance levels.

As with the wider respiratory condition set, LLSOAs providing the poorest levels of model performance were typically located around the incomplete eastern section of Leicester's outer ring road, where congested traffic flows exist at a higher resolution than what could be captured through the NAEI modelled road-traffic emission dataset. It is quite likely that these micro-scale peak flows during certain times of the day are solely responsible for such respiratory burdens, considering that Humberstone and Thurncourt typically display relatively low LLSOA J00-06 admission rates (16.52 per 1,000 children), 6.77% below average citywide levels. Such generally high levels of well-being make it highly unlikely that an unknown malicious socio-environmental factor is at play. Alternatively, inter-individual population differences may provide some reasoning for reduced model performance, whereby an individual's actions influence health to an unmeasurable and minor extent, which only becomes apparent across communities rarely troubled by health burdens.

In contrast, the southern ward of Aylestone shows no signs of experiencing such unrecorded high-resolution interactions with any of the examined socio-environmental influences, in this or the preceding chapter. One should note that urban sprawl has only recently encroached upon the semi-rural ward of Aylestone, which remains characterised by an expanse of water-meadows running alongside a corridor of the River Soar and Grand Union Canal. Due to the wards position upon the fringes of the urban heat in combination to containing several bodies of open, one might expect the cooler damper outdoor environment to perhaps be responsible for URTI's experienced here. Nevertheless, one should understand that typical LLSOA J00-06 admission rates of 17.67 per 1,000 children within Aylestone remain 0.05 cases beneath the citywide LLSOA average figures, indicating that these uncharted rural respiratory issues are only of minor concern. On the other hand, such outputs would show that GWR techniques should be examined in a more critical manner in future study areas, where drastic spatial shifts in environmental phenotypes consistently occur.

The strongest model performances for the J20-22 subset are to be found around the north-western portion of the city centre, across LLSOAs contained within the wider wards of Latimer ($R^2=0.74$), Belgrave ($R^2=0.67$) and Spinney Hills ($R^2=0.64$) and Stoneygate ($R^2=0.72$). It would appear that the J20-22 GWR model offers a more spatially even distribution of model performance, in contrast to the J00-06 GWR model, which tended to provide either a very strong or an acceptable local level of fit. Within the J20-22 respiratory hot-spot of interest R-square values ranged from 0.60-0.80, with a coefficient value of 0.69 for the focal LLSOA of

particular interest. As before, respiratory hospital admission rates towards the eastern fringes of Leicester are inadequately described by the selected explanatory variables, as recorded via poor LLSOA performance levels within the wider wards of Thurncourt ($R^2=0.23$) and Humberstone ($R^2=0.24$).

Specifically relating to the J20-22 subset, one may observe that reduced levels of modelling performance have expanded across the southern periphery, as indicated by local model fits across the wards of Aylestone ($R^2=0.32$) and Knighton ($R^2=0.32$). Akin to URT conditions, J20-22 admission rates per 1,000 children in the wards of Thurncourt (6.39), Humberstone (6.02), Aylestone (6.81) and Knighton (4.64), were respectively recorded to exist at a level 7.25%, 1262%, 1.16% and 32.65% below expected citywide LLSOA values. It is believed that the newly identified poor performance ward of Knighton, relates to localised traffic congestion occurring around the University of Leicester. As previously detailed within the ESDA, it would appear that LRTI's share a heightened sensitivity to spatiotemporal pollutant episodes, whereas URTI's appear dependent on prevailing pollutant levels of moderate intensity.

An assessment of the J00-06 and J20-22 GWR model residuals (Figure 5.6) confirms this notion of a generally favourable model performance, which with increased localisation, cumulatively improves upon the dataset fit provided through their respective OLS models. For the J00-06 subset it should be noted that high residuals remained within the focal point of the central cluster zone of high respiratory admissions, which is indicative of a reduction in model performance (60NN $R^2=0.52$), combined with the area experiencing the highest rate of annual children's URTI admissions within Leicester UA (95.65 per 1,000 children). Nonetheless, J00-06 GWR residuals for the inner-city point of focus (34.50) were found to reduce drastically from the OLS value (65.02), coinciding with the concept of their being extremely localised socio-environmental interactions specifically operating inside this zone. Furthermore, it would appear that the OLS model contains substantial issues of residual clustering across Leicester's western periphery within the wards of Humberstone and Thurncourt, which reside under the implementation of localised modelling techniques. Nevertheless, two LLSOAs within Thurncourt would appear too consistently under or overestimate GWR modelled respiratory outcomes. If one harkens back to the ESDA, such problem locales would appear to mark the junction upon which Leicester's Outer Ring road (A563) terminates, forcing traffic on a single carriageway heading towards the city centre.

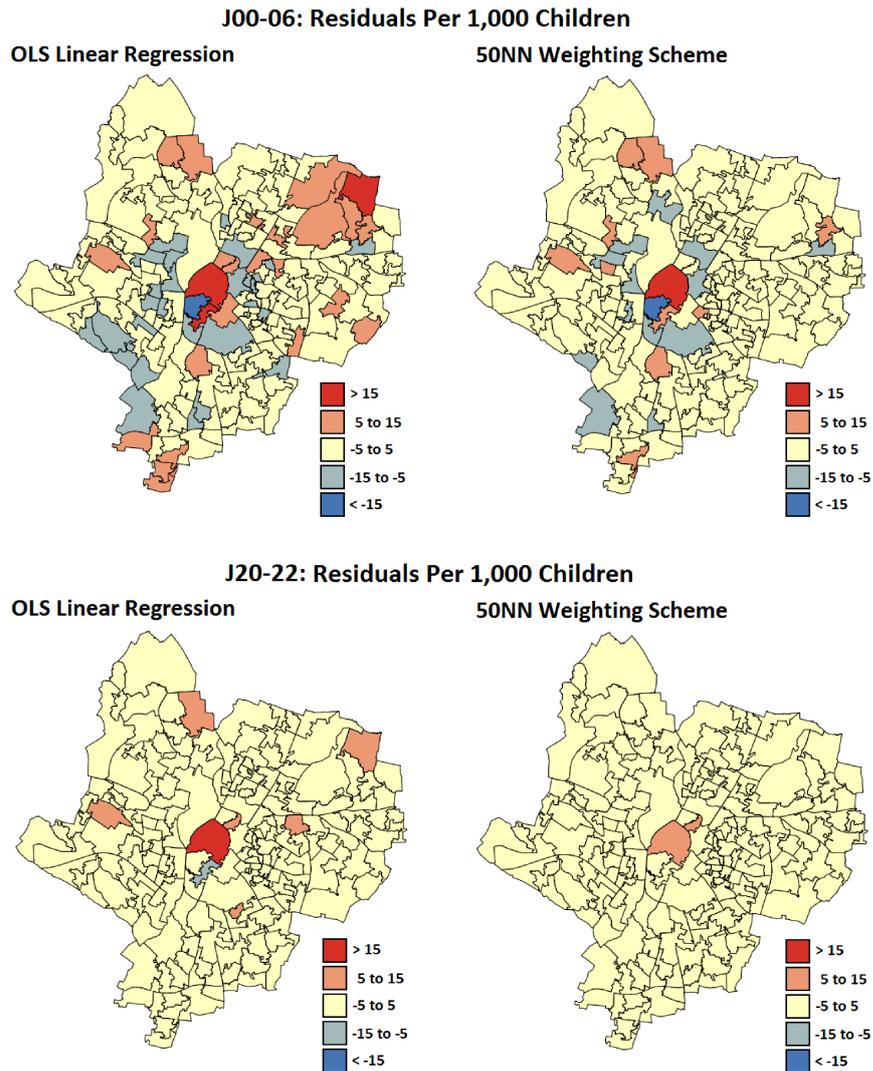


FIGURE 5.6: Spatial distribution of OLS and GWR residual J00-06 and J20-22 hospital admissions per 1,000 children

	Kolmogorov-Smirnov (K-S) Statistic: Residuals Normal Distribution			Global Moran's I: Residuals	
	K-S Value	P ≤ 0.05	P ≤ 0.01	Z-Score	Spatial Pattern
J00-06: OLS	0.13	Reject	Reject	3.29	Clustered
J00-06: 50NN	0.11	Reject	Accept	-2.95	Dispersed
J20-22: OLS	0.08	Accept	Accept	1.33	No Pattern
J20-22: 50NN	0.08	Accept	Accept	-2.05	Dispersed

TABLE 5.4: Spatial distribution of OLS and GWR residual J00-06 and J20-22 hospital admissions per 1,000 children

Likewise, for the J20-22 subset, residuals would appear to remain high under spatial modelling techniques within the focal point of the inner-city hot-spot, however this is more of a case of an exceedingly high LRTI admission rate (34.78 per 1,000 children) rather than reduced model performance (60NN $R^2=0.69$). As illustrated in Figure 5.6, J20-22 GWR residuals for the inner-

city point of focus (7.49) were found to reduce drastically from the OLS value (18.42), once again suggestive of there being extremely localised interactions enforced across inner-city locales. Spatial modelling of the J20-22 subset would also appear to mitigate issues of under-prediction around Western Park, and over-prediction within Humberstone. Kolmogorov-Smirnov scores of the both the J00-06 and J20-22 GWR models identified the residuals to be distributed in a normal fashion ($P \leq 0.01$), with Global Moran's I outputs statistically recognising such residuals to significantly contain no elements of spatial autocorrelation ($P > 0.05$) (Table 5.4). Such findings reveal a satisfactory model performance, indicating that localised regression techniques are capable of dealing with extreme spatial outliers.

5.3.3. SUMMARY OF MODELLED OUTPUTS

Global regression modelling of the J00-06 respiratory subset identified relative rises in residentially experienced TPM_{10} emissions to have the most prolific health impact on children, out of all explored socio-environmental influences (Table 5.5). In exploring the role Leicester's major ethnic minority group's play of community construction, one may observe that increased proportions of 'Indian' residents are associated with substantially lower levels of children's respiratory hospitalisations. Potentially the social attitudes and or response to stimuli within 'Indian' residents may have a role in offsetting major detrimental socio-environmental community burdens. In contrast, elevated levels of 'Other South Asian' residents were significantly and positively associated with J00-06 respiratory hospital admissions.

These findings are in agreement with the J00-06 Univariate Local Moran's I outputs, which indicate there to be 1.1 times more 'Other South Asian' children, and 1.0 times fewer 'Indian' children, residing within inner-city hot-spots (Appendix D3). What's more, Bivariate Local Moran's I outputs revealed J00-06 inner-city hot-spots situated within the immediate vicinity of J20-22 problem areas, to house 1.7 times more 'Other South Asian' children and 1.9 times fewer 'Indian' children (Appendix D4). Such increased levels of association between communities experiencing both URT and LRT infections, present the likely case for such ethnic minority groups as having a noteworthy role in shaping a child's respiratory functions. Children of 'Afro-Caribbean' backgrounds were accredited to the greatest relative risk of experiencing a J00-06 hospital admission out of all of the ethnic minorities investigated, although one was unable to credibly confirm such findings at a global scale ($P = 0.09$).

J00-06 50NN GWR model outputs appear to broadly compliment the OLS regression outputs, identifying TPM_{10} emissions and community levels of 'Indian' children estimates as

the two most spatially significant factors (Table 5.5). Highly positive median TPM₁₀ parameter estimates portray a distribution favouring a broadly detrimental impact on J00-06 respiratory health citywide. The strong beneficial influences of 'Indian' residents on community health may also be observed through (a) strong negative median parameter estimate, (b) all 25.13% locations deemed to provide significant GWR outputs ($P \leq 0.05$) contain negative coefficients. A tendency for J00-06 risks to prevail amongst 'Other South Asian' children was also observed, reconfirming OLS outputs. Modelling differences may be observed through examining the role of deprivation and 'Afro-Caribbean' residents, which now appear significantly detrimental on J00-06 outcomes within certain localities.

Cartographic plots of GWR modelled J00-06 admission rates directly associated with TPM₁₀ emissions (Figure 5.7), almost exclusively identify children residing within Leicester's central locations to be impacted by such a burden, with the severest outcomes affecting inner city children housed within the previously identified J00-06 hot-spot. Specifically within the focal LLSOA of interest, encased within the inner-city ring-road, TPM₁₀ related hospitalisations were recorded to peak at a rate of 64.27 admissions per 1,000 children ($P \leq 0.05$). After which, TPM₁₀ admission rates were observed to rapidly decline across first order neighbouring LLSOAs to an average rate of 22.89 admissions per 1,000 children, thus reconfirming the highly localised influence of environmental afflictions within Leicester. While elevated TPM₁₀ URTI admission rates exist across the overall J00-06 hot-spot of interest, one should note that significant GWR parameter estimates are only derived within the areas central and wider range of nearby communities to the north.

Interestingly, issues of deprivation would also appear to provide the greatest detrimental impacts upon inner-city communities contained within the overall J00-06 hot-spot (Figure 5.7). However, while a common burden is felt across this area, it would appear that such concerns are most prominent within the pockets southern LLSOAs. Average levels of deprivation within these four LLSOAs are record to create an additional 34.90 J00-06 admissions per 1,000 children, and are coincidentally the only LLSOAs within the inner-city zone to provide significant GWR estimates ($P \leq 0.05$). Within the focal LLSOA of interest, deprivation related hospitalisations were at a rate of 29.99 admissions per 1,000 children ($P = 0.12$). While, socio-environmental factors are inclined to affect J00-06 outcomes in one direction, these relationships do not remain spatially uniform, with such affects appearing more pronounced in some areas and non-existent in others. In particular, one should note that a beneficial URTI response surprisingly occurs in relation to increased levels of deprivation across LLSOAs located alongside the north-eastern border of the J00-06 hot-spot, within the wards of Latimer and Charnwood ($P \leq 0.05$).

Such observations would indicate that a rather complex set of interactions govern the nature and extent to which a specific division of socio-economic status influences upper respiratory health during childhood. In-fact, J00-06 outcomes accredited to social positioning, were determined to only operate in the direction of a communities respective socio-economic status across 52.69% of the cities LLSOAs (i.e. deprivation increases, whereas affluence decreases risk likelihoods). Meanwhile 11.30% of deprived areas found within the wards of Latimer and Charnwood were accredited with a beneficial response, while in 36.01% of the cities LLSOAs affluence was considered to detrimentally influence URT outcomes. Yet it should be noted that this section of relatively affluent communities only marginally influence a child's upper respiratory health in an undesirable fashion, with such communities principally occupying western radial segments categorised to experiencing only '-5 to 5' J00-06 cases per 1,000 children (Figure 5.7).

Cartographic plots of GWR outputs illustrates that the Lifestyle choices of 'Indian' residents actively reduces the number of respiratory hospitalisations within the J00-06 inner-city hot-spot, acting in a manner which mitigates the spatial spread of such symptoms to the wider city centre community (Figure 5.7). In particular LLSOAs within the ward of Spinney Hills are densely populated by 'Indian' children, whose social lifestyle is observed to on average prevent 18.56 J00-06 cases per annum, which are likely to occur from their residentially raised deprivation (3.87) and TPM₁₀ emission (1.50t/yr.) levels. Therefore, one should not discount the importance of social decision making alongside the ease to which groups whom have fully integrated into the wider society may access public services.

Independent Variable: (Normalised 0-1)	Aspatial Linear Regression (OLS)			Linear 50 Nearest Neighbours Bisquare-Adaptive GWR				% Census Areas P≤0.05	
	β Value	Std. Error	P Value	Min. β	Med. β	Max. β	Std. Error	Detrimental (+)	Beneficial (-)
Intercept	11.16	2.51	0.00*	-9.09	9.11	51.18	0.80	---	---
Carstairs Index 2001	6.58	4.76	0.17	-25.04	8.12	95.92	1.18	17.65	3.21
TPM ₁₀ Emissions (t/yr.)	17.98	4.86	0.00*	-39.35	10.24	64.27	1.31	23.53	3.21
% Smoking Prevalence (Age 16yrs+)	1.72	3.86	0.66	-16.10	4.34	28.53	0.61	8.02	0.00
% Obesity Prevalence (Age 16yrs+)	-0.03	3.20	0.99	-45.57	1.00	34.89	0.97	6.95	4.81
% 0-15y White Non-British	-4.22	2.88	0.14	-18.48	-0.66	17.26	0.44	4.81	1.60
% 0-15y Indian	-11.73	3.35	0.00*	-46.04	-7.36	9.83	0.72	0.00	25.13
% 0-15y Other South Asian	8.60	3.89	0.03*	-9.22	9.05	72.77	1.03	18.72	0.00
% 0-15y Afro-Caribbean	10.04	5.83	0.09	-30.76	5.84	50.63	1.25	23.53	0.00
R-Square	0.37			0.73					
Residual Sum Of Squares (RSS)	9302.40			3980.84					
Mean Squared Error (MSE)	49.75			21.29					
AIC	1279.28			1164.01					
AICc	1282.53			1291.61					
F-Test	13.24*			2.34*					
R-Square Cross-Validation: 2004-06 J00-06 Hospital Admissions	0.34			0.56					

* P ≤ 0.05

TABLE 5.5: Linear Aspatial and Geographically Weighted Regression (GWR) models of annual average J00-06 hospital admissions per 1,000 persons aged 0-15yrs within Leicester UA: 2000-09

J00-06 Hospital Admissions Per 1,000 Children

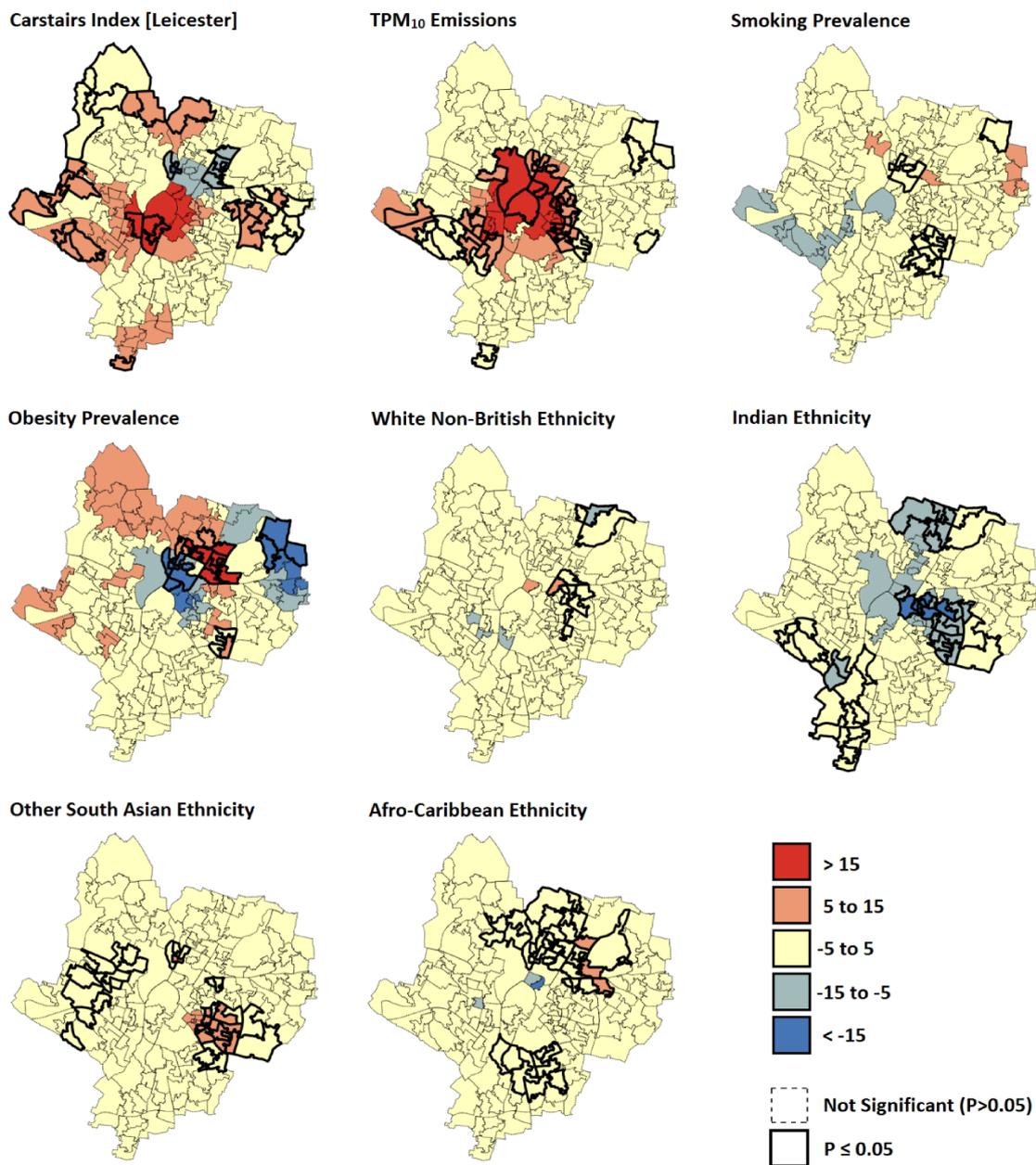


FIGURE 5.7: GWR modelled children's J00-06 hospital admission rates instigated through interactions with an individual socio-environmental influence of interest

In total, both global and local model estimates mutually recognised background respiratory statuses (or base rate), recorded via the Intercept, to be accountable for a similar and sizable portion (64.52-69.54%) of the expected 1,079 annual children's URTI hospital admissions, experienced across Leicester UA (Table 5.6). Noteworthy differences between the two modelling techniques were only to be observed in relation to cases determined by TPM₁₀ emissions and 'Afro-Caribbean' residency levels, whereby GWR outputs respectively depicted such factors as influencing 6.98% and 2.94% fewer URTI cases (Table 5.6). Once more, these

findings are indicative of their highly localised natures, suggesting that a majority of Leicester’s communities are disassociated with such social and environmental burdens. Out of the explored socio-environmental influences, levels of TPM₁₀ emissions (+23.62%), deprivation (+19.2%) and ‘Indian’ residency (-23.27%) were observed to influence the greatest quantity of children’s citywide J00-06 cases, under a local modelling scheme. Meanwhile the lifestyle choices of ‘Afro-Caribbean’ (+1.74%) and ‘Other South Asian’ (+7.42) residents, were associated to barely influence citywide J00-06 health burdens, primarily due to their relatively low levels of residency across Leicester UA.

	Int.	Carst. Index	TPM ₁₀	Smoke Prev.	Obese Prev.	White N-Brit.	Indian	Other S. Asian	Afro-Car.	Resid.
J00-06 OLS	64.52	17.89	30.60	3.94	-0.12	-5.11	-22.50	8.14	4.68	-2.04
J00-06 GWR	69.54	19.20	23.62	3.37	1.05	-0.51	-23.27	7.42	1.74	-2.17
J20-22 OLS	55.63	35.03	22.23	14.32	-11.60	0.76	-21.64	0.69	5.82	-1.23
J20-22 GWR	79.61	42.77	4.79	17.73	-35.69	4.02	-14.40	0.90	2.44	-2.18

TABLE 5.6: Percentage of citywide annual children's J00-06 and J20-22 hospital admissions associated with global and locally modelled background and socio-environmental influences

Akin to the J00-06 outputs, global regression modelling of the J20-22 subset recognised the significantly detrimental impact of TPM₁₀ emissions and beneficial influence of ‘Indian’ residents on a community’s respiratory health (Table 5.7, Figure 5.8). Yet unique to the J20-22 subset, levels of Afro-Caribbean residents and deprivation were shown to significantly increase children’s respiratory risks at the citywide level. Once again, parameter estimates from GWR modelling appear to broadly compliment OLS modelling outputs. However, it should be noted that GWR modelling has acted to drastically reduce the influence of TPM₁₀ on J20-22 health outcomes, as denoted by a negative median parameter estimate (Table 5.7).

In-fact, TPM₁₀ effects would appear particularly pronounced within inner city localities where children experiencing the greatest health burdens (Figure 5.8), yet such impacts appear hardly evident in other areas. Normalised increases in Carstairs Index deprivation levels were identified to have the most prolific impact on children’s J20-22 admissions rates, out of all explored influences across a global and localised scale. The overwhelming influence of deprivation may be further observed by all 32.62% of areas deemed to produce significant GWR deprivation coefficients ($P \leq 0.05$) resulting in a detrimental health impact, in-line with their respective socio-economic status (i.e. deprivation increases risk likelihood). This would suggest that deprivation impacts LRT outcomes in a far more simplistic manner, yet one may observe that deprivation typically burdens communities in a fashion which mutually elevates children’s cases of both URT and LRT infections.

Independent Variable: (Normalised 0-1)	Aspatial Linear Regression (OLS)			Linear 50 Nearest Neighbours Bisquare-Adaptive GWR				% Census Areas P≤0.05	
	β Value	Std. Error	P Value	Min. β	Med. β	Max. β	Std. Error	Detrimental (+)	Beneficial (-)
	Intercept	3.79	1.04	0.00*	-9.10	4.49	21.27	0.31	---
Carstairs Index 2001	5.08	1.98	0.01*	-8.44	6.43	30.26	0.40	32.62	0.00
TPM ₁₀ Emissions (t/yr.)	5.15	2.02	0.01*	-29.03	-1.09	31.63	0.58	5.35	6.42
% Smoking Prevalence (Age 16yrs+)	2.47	1.61	0.13	-8.42	4.25	11.89	0.30	17.65	1.07
% Obesity Prevalence (Age 16yrs+)	-1.10	1.33	0.41	-18.67	-2.64	4.27	0.29	0.00	8.02
% 0-15y White Non-British	0.25	1.20	0.84	-4.44	2.69	9.41	0.27	13.37	10.16
% 0-15y Indian	-4.45	1.39	0.00*	-19.28	-4.01	2.96	0.29	0.00	15.51
% 0-15y Other South Asian	0.29	1.61	0.86	-7.36	0.89	23.47	0.65	19.25	0.00
% 0-15y Afro-Caribbean	4.92	2.42	0.04*	-15.87	3.48	18.57	0.55	15.51	4.28
R-Square	0.42			0.72					
Residual Sum Of Squares (RSS)	1605.70			766.13					
Mean Squared Error (MSE)	8.59			4.10					
AIC	950.77			855.85					
AICc	954.02			983.46					
F-Test	16.10*			2.10*					
R-Square Cross-Validation: 2004-06 J20-22 Hospital Admissions	0.27			0.50					

* P ≤ 0.05

TABLE 5.7: Linear Aspatial and Geographically Weighted Regression (GWR) models of annual average J20-22 hospital admissions per 1,000 persons aged 0-15yrs within Leicester UA: 2000-09

J20-22 Hospital Admissions Per 1,000 Children

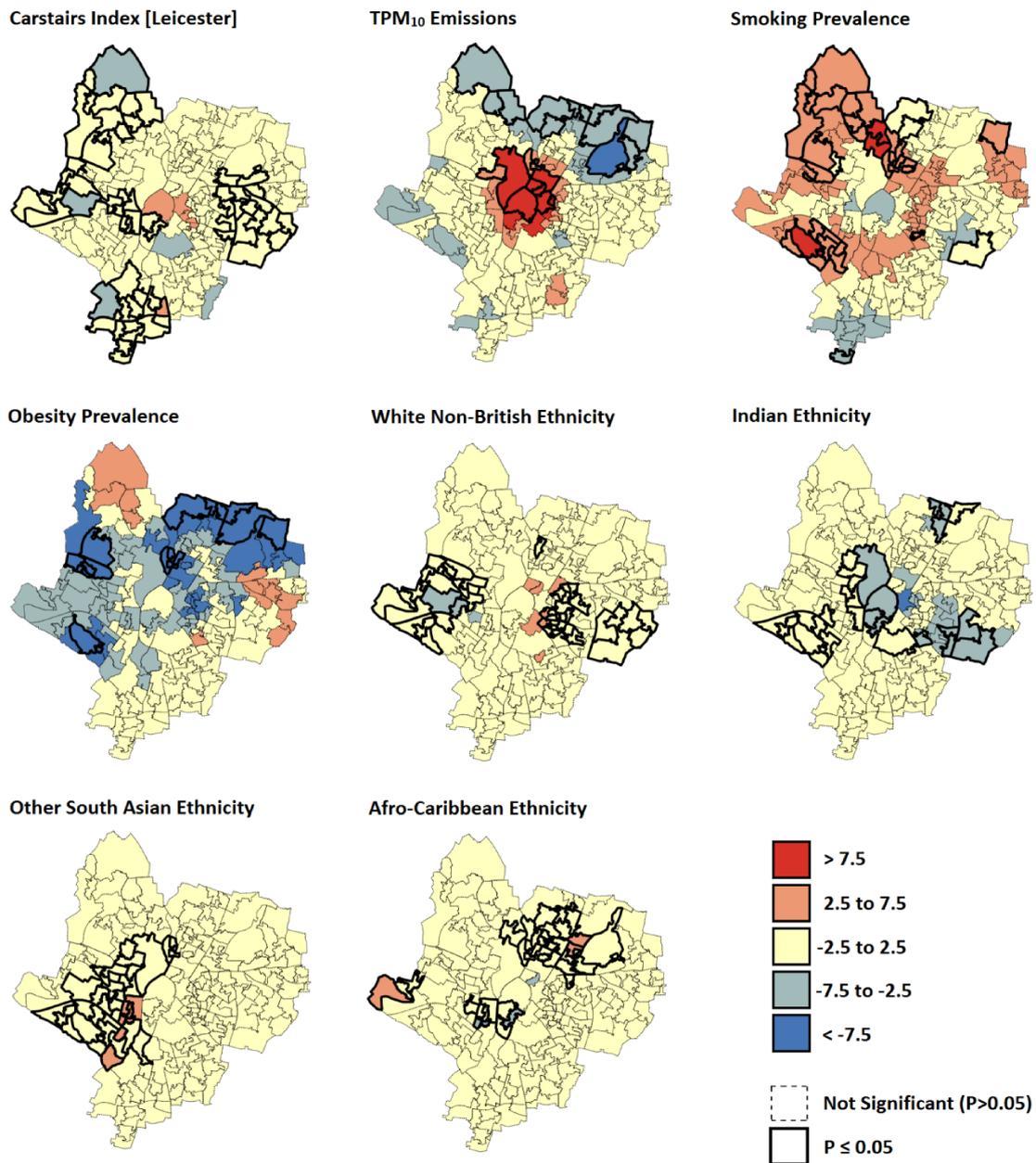


FIGURE 5.8: GWR modelled children’s J20-22 hospital admission rates instigated through interactions with an individual socio-environmental influence of interest

In total, both global and local model estimates mutually recognised background respiratory statuses (or base rate), recorded via the Intercept, to be liable for a sizable portion of the expected 425 annual children’s J20-22 hospital admissions, experienced across Leicester UA (Table 5.6). However, unlike J00-06 modelling, localised techniques appeared to place a far greater accountability on J20-22 background respiratory levels (79.61%), when compared to their global counterpart (55.63%). Consequently, several sizeable changes were observed between the two modelling techniques, particularly in relation to the number of cases caused

by levels of TPM₁₀ emissions and obesity prevalence, which respectively induced 17.44% and 24.09% fewer J20-22 hospitalisations under localised models (Table 5.6). As a result of such changes, childhood activity and dietary intake construed from GWR model estimates of parent's obesity levels, appeared to now provide the primary force in mitigating citywide J20-22 outcomes. Out of the explored socio-environmental influences, levels of deprivation (+42.77%) and obesity prevalence (-35.69%) were observed to influence the greatest quantity of children's citywide J20-22 cases, under a local modelling scheme. Meanwhile, residentially experienced TPM₁₀ emissions (+4.79) and the lifestyle choices of 'Afro-Caribbean' (+2.44%) residents, due to their restricted nature of elevation, provided a minimal influence upon citywide J20-22 health burdens.

Cartographic plots of the relative J20-22 admission rates confirm these findings, identifying smoking and obesity prevalence as the main explanatory factors for LRTI's outcomes in children residing outside of the inner-city hot-spot of interest (Figure 5.8). In LSOA's where dietary intake and activity were believed to instigate >2.5 (high) and <-2.5 (low) J20-22 admissions per 1,000 children, levels of adult obesity prevalence were recorded at the respectively comparable rates of 25.14% and 25.21%. While this may at first appear confusing, additional exploration of the raw data reveals beneficial obesity responses (<-2.5 admissions) to typically occur in communities where 50.99% of children are of 'White British' ethnicity, whom experiencing an indifferent Carstairs Index score of 0.58. In contrast, detrimental obesity responses (>2.5 admissions) were generally observed across somewhat affluent areas (Carstairs Index -2.02), housing similar levels of 'White British' children (57.03%).

Cartographic plots subsequently indicate both obesity responses to be somewhat exclusively experienced by Leicester's 'White British' inhabitants, therefore explaining why this lifestyle indicator governs a considerable proportion of citywide J20-22 responses. Furthermore, these observed trends in the native populace are to a certain degree what one would hope for, with the typical family household appearing to take a proactive stance if their child shows LRT symptoms. However, one may observe that smoking prevalence, depicted as the second leading explanatory factor outside of the J20-22 inner city hot-spot, would generally appear to offset the overwhelming beneficial influences recorded via levels of obesity prevalence. For instance, in locales where smoking prevalence were displayed to be of detriment (Figure 5.8), community levels of obesity and smoking prevalence were typically recorded to respectively prevent 5.66 or stimulate 4.16 annual J20-22 admissions per 1,000 children. If taken together, the overall beneficial response recorded via obesity prevalence (-35.69%) appears visibly moderated, presenting a 17.96% reduction in the number of potential citywide J20-22 cases.

Meanwhile, the impact of TPM_{10} emissions on LRTI's would appear more localised than what was previously recorded in relation to URTI's, restrictively operate around the core of the inner city hot-spot and immediate north-eastern communities (Figure 5.8). One should note that these LLSOAs are positioned in-line with prevailing winds from the inner-city ring-road, in addition to accommodation several key arterial road connections. Specifically within the focal LLSOA of interest, encased within the inner-city ring-road, TPM_{10} related hospitalisations were recorded to peak at a rate of 36.19 admissions per 1,000 children ($P \leq 0.05$), under a residential exposure of 2.65t/yr. After which, TPM_{10} admission rates were observed to rapidly decline across first order neighbouring LLSOAs, averaging 11.98 admissions per 1,000 children under a 2.01t/yr. exposure, thus reconfirming the highly localised influence of environmental afflictions within Leicester. This reduced zone of impact would suggest that long-term (annual) exposure is a less effective means of initiating LRTI's unless a critical value is reached, and that perhaps LRTI's are driven in a more episodic fashion (i.e. determined via seasonal weather conditions and or localised traffic flows throughout the day). This heightened sensitivity of J20-22 cases to daily episodes, perhaps alludes to a previously identified outlying southern LLSOA community encompassing the University of Leicester campus, which experiences traffic flow issues only during peak hours of the day. Yet significant convergence of such outputs here and at the three other J20-22 outliers has failed, likely due to the previously described resolution limitations of the 1x1km NAEI dataset.

Compared to the aforementioned J20-22 socio-environmental influences, deprivation would appear to operate in a relatively smooth universal manner, with beneficial responses only observed within the cities 16 LLSOAs (8.56%) deemed to exhibit Carstairs Index scores below 0. This consistent nature explains how socio-economic positioning is deemed to encourage 42.77% of children's J20-22 cases within Leicester UA. Nevertheless, spatially elevated rates would appear to once again occur across the inner city zone of interest, with admissions per 1,000 children peaking at a rate of 5.34 towards the most western part of the J20-22 hotspot ($P=0.13$). However, deprivation may only defined to significantly burden a child's LRT health across two LLSOAs contained within the J20-22 hot-spot, averaging a modest 1.71 admissions per 1,000 children ($P \leq 0.05$).

Akin to the J00-06 models, levels of 'Indian' residency were once again observed to provide a consistently beneficial influence towards children's J20-22 outcomes. In-fact, only 30 LLSOAs (16.04%) recorded levels of 'Indian' residency as providing a detrimental influence, and even then such an influence on average only resulting in 0.42 additional admissions per 1,000 children. Once again, the full force of 'Indian' residency may be observed across inner-city locales, with 4 particular LLSOAs of the J20-22 hot-spot, on average describing their 27.29% of

Indian residents as actively preventing an additional 4.03 admissions per 1,000 children ($P \leq 0.05$). However one should also note that the lifestyle choices of 'Indian' residents, is not an advantageous phenomenon restricted to inner city locales, as observed by low 'Indian' residency levels within the LLSOAs of Braunstone (4.09%) preventing on average 0.47 J20-22 admissions per 1,000 children ($P \leq 0.05$).

5.3.4. SOCIO-ENVIRONMENTAL MECHANISMS OF DETERIORATING RESPIRATORY HEALTH

Under the premise of examining the extent to which socio-environmental mechanisms play in the decline of respiratory health, GWR coefficients were used to derive specific admission rates for independent variables, for both the J00-06 and J20-22 subsets. Two possible scenarios have been imagined: Socio-environmental factors (a) have no common mechanism in the decline of respiratory health; (b) weaken the upper respiratory system initially causing minor upper respiratory tract complaints (J00-06), with prolonged exposure(s) leaving the lower respiratory tract vulnerable to conditions (J20-22) which may prevail throughout childhood.

Simple X-Y plots of J00-06 vs. J20-22 admission rates for each individual socio-environmental factor (Table 5.8), indicated levels of TPM_{10} and Carstairs Index deprivation as likely causalities for a decline in children's respiratory health ($R^2 \geq 0.5$). In terms of community compositions, levels of White Non-British and Indian ethnic minority groups were detected to operate in a spatially nature which influenced both children's J00-06 and J20-22 admission rates in a mutual manner ($R^2 \geq 0.5$). Pearson's R Correlation statistics confirm such findings, with admission rates for the aforementioned socio-environmental factors scoring from +0.7 to +1.0, indicating the existence of strong positive global associations. Spearman's Rho values were observed at a lower level than their Pearson's R counterparts for the four factors of interest, which would imply that their individual influences on hospital admissions for relatively severe and mild respiratory complaints are linearly dependant in nature.

With the Moran's I global index reflecting a spatially weighted form of Pearson's correlation coefficient, it was deemed appropriate to apply a derivative of the global index known as the Local Bivariate Moran's I, for the exploration of spatial patterning. The spatial influence of 'White Non-British' residents was not investigated further, due to the group's relatively low associations towards respiratory risk combined with their being only a limited number of areas possessing significant GWR coefficients. Bivariate Local Moran's I R^2 plots indicative of global spatial associations between an ego location (i) and directly neighbouring localities (j), display a good amount of spatial correlation between J00-06 and J20-22

admission rates associated with TPM₁₀ levels, when either subset was held as the ego (Table 5.8). Citywide J20-22 admission rates attributed to deprivation also appeared to have good correlations with deprivation related J00-06 admissions in neighbouring localities. Only a moderate goodness-of-fit was observed relating to the global benefits on respiratory health brought by 'Indian' residents.

	R ²	Spearman's Rho P≤0.05	Pearson's R P≤0.05	Bivariate Local Moran's: R ²	
				I=J00-06; J= ...	I=J20-22; J= ...
TPM₁₀ Emissions	0.72	0.60	0.85	0.58	0.54
Carstairs Index	0.58	0.67	0.76	0.43	0.52
Smoking Prevalence	0.01	0.27	0.11	0.00	0.00
Obesity Prevalence	0.11	0.10	0.33	0.04	0.05
White Non-British	0.50	0.54	0.71	0.22	0.22
Indian	0.52	0.58	0.72	0.43	0.33
Other South Asian	0.05	0.29	0.22	0.00	0.00
Afro-Caribbean	0.48	0.63	0.69	0.24	0.33

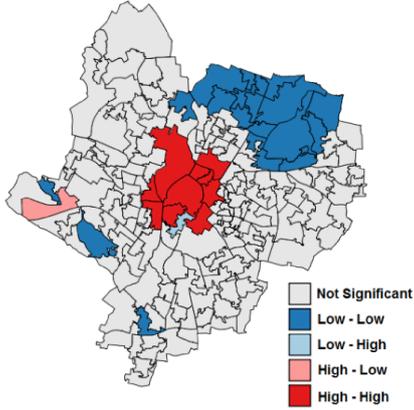
TABLE 5.8: Global correlation statistic outputs of J00-06 vs. J20-22 GWR modelled hospital admissions per 1,000 children attributed to specific socio-environmental factors

One should note that GWR models exploring the respiratory conditions of interest were created within SpaceStat 3.5.6, which produces identical outputs to alternative statistical packages, while offering a more stringent check for significance. Traditionally GWR regression parameter standard errors are calculated using the global error variance, defined as the Residual Sum-of-Squares (RSS) at each of the target points (Fotheringham et al 2002). However, SpaceStat uses the local variance defined as the RSS from the regression calculation at the source points to calculate local rather than global standard errors, and hence local p-values for the regression parameters. It is believed that this approach should more accurately reflect the degree of non-stationarity encapsulated in the geographically weighted calculation.

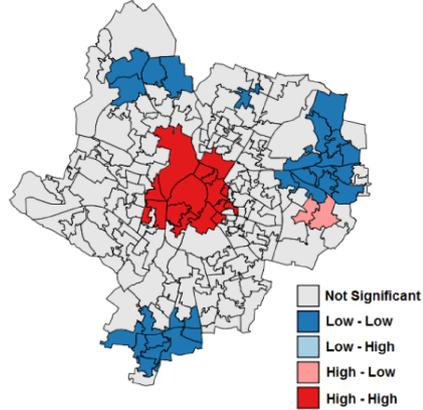
Cartographic plots of Bivariate Local Moran's I outputs (Figure 5.9), identify TPM₁₀ as a significantly responsible factor for both J00-06 and J20-22 admissions within the city centre hot-spots previously identified for both condition sets (P≤0.05). Hospitalisations attributed to deprivation levels also appeared high for both conditions within inner city localities, however GWR deprivation coefficients were only deemed of mutual significance by locally modelled p-values (SpaceStat 3.5.6), within a small southern portion of these inner city neighbourhoods (Figure 5.10). In contrast the wider inner-city hot-spot of modelled deprivation admissions was noted to be of significance, when interpreting the less strict Fotheringham et al (2002) GWR coefficient p-values, constructed from the global error variance.

GWR Modelled TPM₁₀ Admissions Per 1,000 Children

Bivariate Local Moran's I:
J00-06 (i), J20-22 (j)

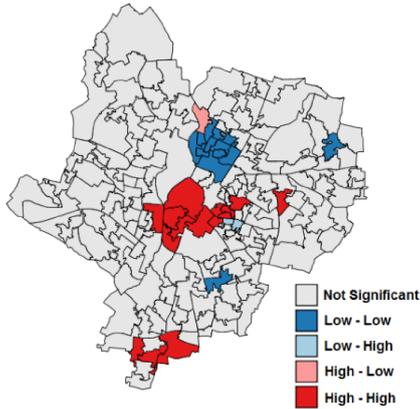


Bivariate Local Moran's I:
J20-22 (i), J00-06 (j)

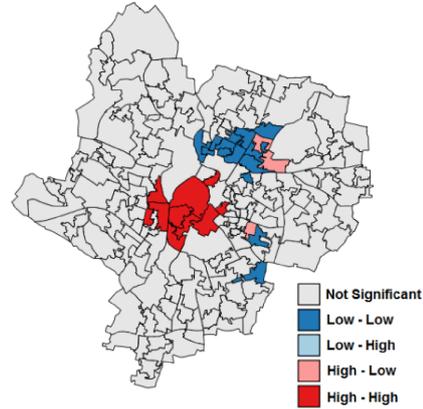


GWR Modelled Carstairs Index Admissions Per 1,000 Children

Bivariate Local Moran's I:
J00-06 (i), J20-22 (j)

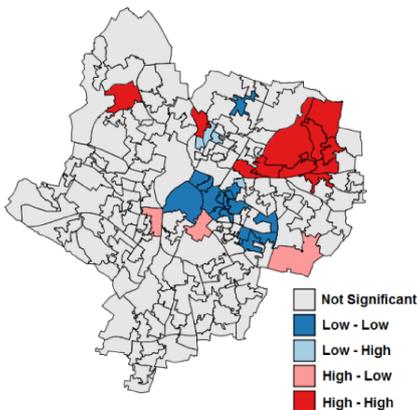


Bivariate Local Moran's I:
J20-22 (i), J00-06 (j)



GWR Modelled Admissions Per 1,000 Children Associated With Levels Of Indian Residents

Bivariate Local Moran's I:
J00-06 (i), J20-22 (j)



Bivariate Local Moran's I:
J20-22 (i), J00-06 (j)

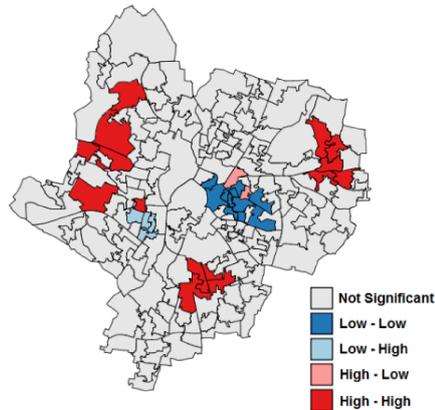
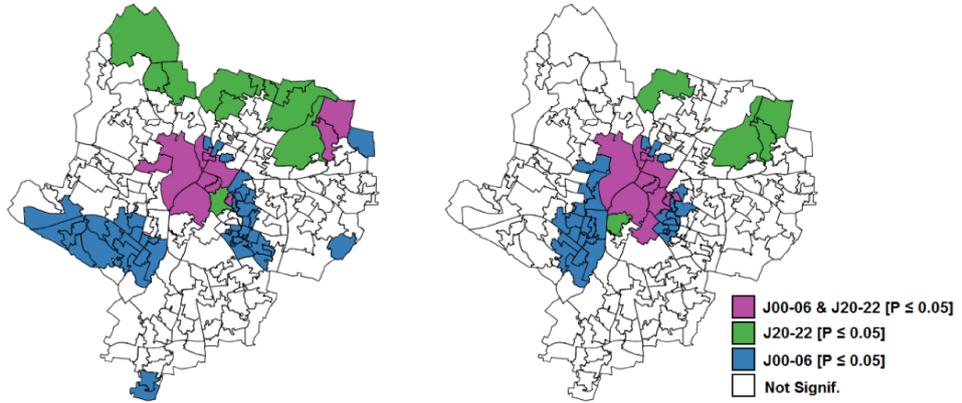


FIGURE 5.9: Bivariate Moran's I cluster analysis of GWR modelled J00-06 and J20-22 admission rates attributed to socio-environmental factors of importance

Significant GWR Coefficients: TPM₁₀ Emissions

Local Models: Spacestat 3.5.6.

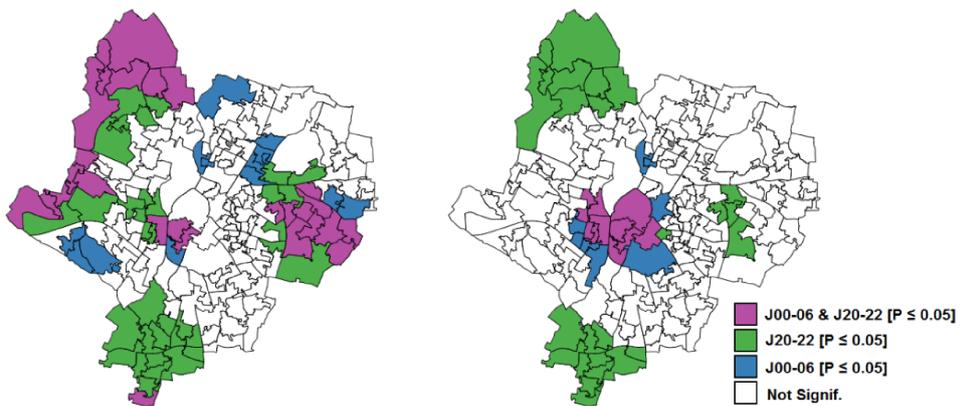
Global Model: Fotheringham et al. 2002



Significant GWR Coefficients: Carstairs Index (Leicester)

Local Models: Spacestat 3.5.6.

Global Model: Fotheringham et al. 2002



Significant GWR Coefficients: Percentage Of Indian Children

Local Models: Spacestat 3.5.6.

Global Model: Fotheringham et al. 2002

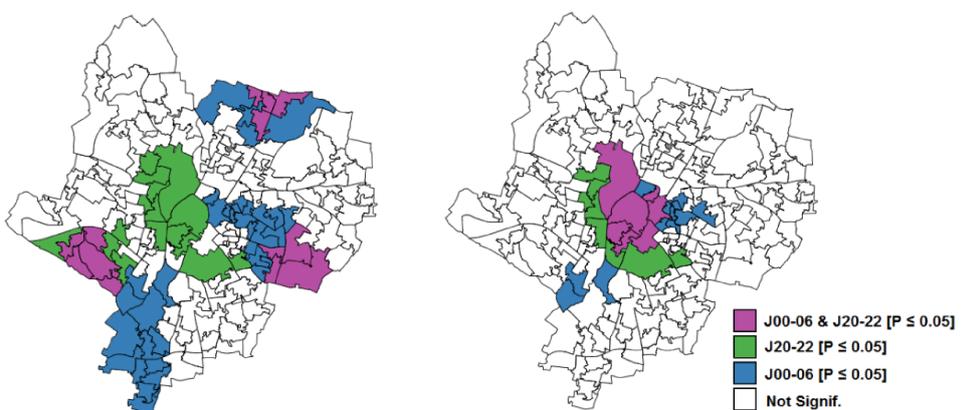


FIGURE 5.10: Local and global significance levels of GWR modelled J00-06 and J20-22 admission rates attributed to socio-environmental factors of importance

Interestingly levels of 'Indian' residents appear to mitigate the influence of detrimental socio-environmental factors, and in part reduce the potential spatial extent of such respiratory issues around other centric localities (Figure 5.10). In particular, Indian residents provide substantially low respiratory admission rates towards the eastern fringes of the previously identified inner city hotspots (Figure 5.1). However, it is only the global GWR coefficient p-values, which record the significance of this social group in influencing overall respiratory mechanisms across common inner-city locations (Figure 5.10). Locally modelled p-values were only able to confirm this social group's beneficial influence on J20-22 cases within inner city localities, which were neighboured by areas experiencing a similar beneficial influence on J00-06 outcomes.

In another point of noteworthy interest, it may be observed that a few pockets exist whereby J00-06 and J20-22 admission rates rise as a consequence of 'Indian' residency. The most prominent of which occurs within Eastern Leicester, when the J00-06 subset is held at the ego location (i). However, under closer inspection these so called hot-spots are only relative to the dataset, with respective average J00-06 and J20-22 annual rates of -0.15 and 0.49 in hot-spots, compared to -17.13 and -3.72 admissions per 1,000 children in cold-spots. In accordance with earlier observations, it is possible to confirm that, 'Indian' residents generally have a substantially beneficial impact on community respiratory health, with the exception of a few locations where their influence on health appears insignificant. Such findings highlight the potential for non-uniform relationships, especially when examining broad social groups, which may share common traits yet still make different lifestyle choices based on the community they reside within.

The findings presented here would indicate that road-traffic emissions and levels of deprivation are likely candidates responsible for a communities deteriorating respiratory health. Nevertheless, it would appear that lifestyle choices, such as those seen by 'Indian' residents, could mitigate the onset of such conditions.

5.3.5. GWR STIMULUS-RESPONSE MODELS

Locations identified to produce significant parameter estimates variables from GWR modelling ($P \leq 0.05$), were selected for the construction of stimulus-response models specific to each independent socio-environmental variable, while accounting for spatial interference (Table 5.9). The stimulus-response models, in general identify each socio-environmental variable to individually influence the different respiratory subsets via a common function. This supports the concept of socio-environmental factors operating under specific mechanisms, potentially

resulting in a range of respiratory conditions, which become more detrimental provided adequate exposure occurs. Of noteworthy interest, is the manner in which TPM₁₀ emissions and ‘Other South Asian’ residents appear to detrimentally affect upper and lower respiratory conditions via their respective quadratic ($R^2=0.95$) and cubic ($R^2 \geq 0.89$) relationships. In contrast, substantially beneficial influences on both upper and lower respiratory health appear to be associated with high community levels of ‘Indian’ residents, as denoted by a cubic function ($R^2 \geq 0.60$).

Independent Variable:	Subset	LLSOAs (P<0.05)	Model Selection (P<0.05)		Parameter Estimates			
			Optimum	R ²	β ₀	β ₁	β ₂	β ₃
TPM ₁₀ (t/yr.)	URTI	50	Quadratic	0.95	-0.86	-4.18	10.63	
	LRTI	22	Quadratic	0.95	-1.57	-4.95	6.56	
Carstairs Index: Leicester	URTI	39	N/A					
	LRTI	61	Cubic	0.43	5.57	0.75	-0.11	-0.01
(% Adult Smoking)	URTI	16	Quadratic	0.83	-7.09	0.80	-0.01	
	LRTI	35	Cubic	0.48	-3.70	0.46	-0.01	0.00
(% Adult Obesity)	URTI	22	N/A					
	LRTI	15	N/A					
(% White Non-British)	URTI	12	Cubic	0.88	-0.31	6.31	-5.24	0.88
	LRTI	44	Cubic	0.31	-0.13	0.97	-0.36	0.02
(% Indian Children)	URTI	47	Cubic	0.60	0.34	-0.45	0.01	0.00
	LRTI	29	Cubic	0.83	0.08	-0.17	0.00	0.00
(% Other South Asian)	URTI	35	Cubic	0.89	0.21	0.80	-0.06	0.00
	LRTI	36	Cubic	0.92	-0.01	0.64	-0.07	0.01
(% Afro-Caribbean)	URTI	45	Linear	0.86	-0.11	0.90		
	LRTI	37	Cubic	0.41	-0.12	0.65	-0.11	0.00

TABLE 5.9: Stimulus-response models describing the relationship between socio-environmental variables and their specific GWR modelled hospital admissions rates per 1,000 children (If P ≤0.05)

In modelling the significant GWR parameter estimates ($P \leq 0.05$), one may observe that the socially adjusted effects of TPM₁₀ emissions affects both upper and lower respiratory health through similar shaped quadratic relationships (Appendix D8). As one might expect the URTI trend is transposed to a higher magnitude of admission rates that what was recorded for LRTI’s, indicative of their increased frequency; in addition to representing that likely relationship between URT conditions increasing the hosts susceptibility to LRT complaints. For the J00-06 GWR derived stimulus-response model, LLSOAs experienced TPM₁₀ emissions of 1.5t/yr. were annually identified to experience an additional 16.79 hospitalisations per 1,000 children across 2000-09 ($P \leq 0.05$). Using the following trend, LLSOA TPM₁₀ emission levels were reported to average 7.76 admissions per 1,000 children, amounting to a rate of 6.40 admissions above the corresponding 50NN GWR model parameter estimates of mixed

significance. Overall, TPM₁₀ emissions across Leicester would be recognised to influence 436 children's J00-06 admissions per annum ($P \leq 0.05$), or 40.40% of the total J00-06 respiratory burden. One should recognise that this figure is approximately double the size of the 50NN GWR model estimate of 23.62%. Across Leicester it is calculated that a 5% reduction in residentially experienced LLSOA TPM₁₀ emissions would amount to 61 fewer children's J00-06 admissions per annum ($P \leq 0.05$), reducing the total amount of respiratory hospital incidents by 5.65%. Furthermore, it is estimated that a negligible amount of TPM₁₀ related hospital admissions could be achieved if LLSOA emission levels could be maintained around 0.55t/yr.

For the J20-22 stimulus-response model, LLSOAs experienced TPM₁₀ emissions of 1.5t/yr. were annually identified to experience an additional 5.76 hospitalisations per 1,000 children across 2000-09 ($P \leq 0.05$). Using the following trend, LLSOA TPM₁₀ emission levels were reported to average 1.29 admissions per 1,000 children, which is of a comparable nature to the figure of 0.63 provided via the complete set of GWR parameter estimates of mixed significance. Overall, TPM₁₀ emissions across Leicester would be recognised to influence 54 children's respiratory admissions per annum ($P \leq 0.05$), which amounts to 12.69% of the total J20-22 respiratory burden; a figure deviating somewhat from the complete 50NN GWR model estimate of 4.79%. In following this proposed trend, it is estimated that a 5% reduction in residentially experienced LLSOA TPM₁₀ emissions would amount to 24 fewer children's J20-22 admissions per annum ($P \leq 0.05$), reducing the total amount of respiratory hospital incidents by 5.64%. Furthermore, it is estimated that a negligible amount of TPM₁₀ related hospital admissions could be achieved if LLSOA emission levels could be maintained around 1.00t/yr.

In-fact if a universal 14% reduction on present LLSOA TPM₁₀ emissions levels would collectively provide the general populace with a 'safe' level of exposure in relation to LRT conditions, potentially stunting the development of URTI's into conditions of increased severity.

In contrast, the individual influence of 'Indian' residency is shown to affect upper and lower respiratory health via two negative cubic functions of a similar trend where the group defines <50% of a LLSOAs children, after which a far grander beneficial response is provided towards URT rather than LRT conditions (Appendix D9). Such findings are unsurprising, considering that this social group's lifestyle was previously recorded to prevent health issues across the entire spectrum of respiratory conditions (J00-99), with one expecting more URTI's to be substantially reduced due to their increased frequency and preventative nature. Nevertheless the J20-22 negative cubic function, would also suggest that 'Indian' residents are capable of inhibiting a child's respiratory health from deteriorating further in cases where URTI's have become established.

Such outcomes likely relate to the social empowerment of this minority group, whom are thought to have the knowledge of and access to the correct medical services. One should also note that a typical URT cases is unlikely to develop into a LRT condition, and that perhaps social lifestyle choices have a far weaker direct influence upon deteriorating respiratory health; as indicated by the limited reduction in J20-22 cases where >50% of a LLSOAs children are of 'Indian' ethnicity. Across Leicester, 'Indian' residency was recognised to prevent 415 children's J00-06 admissions per annum ($P \leq 0.05$), amounting to -38.46% of the cities J00-06 cases experienced by children. In addition, such residency levels were recognised to prevent 145 children's J20-22 admissions per annum ($P \leq 0.05$), or -14.40% of all children's J20-22 admissions. These J00-06 and J20-22 estimates are of a somewhat more advantageous nature that what was provided by GWR estimates of mixed significance, which correspondingly recorded 'Indian' residency as preventing 23.27% and 14.40% of children's citywide cases.

The individual influence of 'Other South Asian' residency on URT and LRT health would appear to follow a positive cubic trend, which exists in somewhat of a uniform manor until >10% of a LLSOAs community is constructed from this ethnic group (Appendix D10). After this point J20-22 admissions are observed to rapidly increase in relation to 'Other South Asian' residency levels, at a magnitude substantially greater than what is recorded for J00-06 rates. If this depiction is considered accurate, then one might hold the opinion that 'Other South Asian' lifestyles contribute to severe, potentially long-lasting respiratory conditions, which periodically exacerbate the host's respiratory status through the instigation of URT episodes. However a detailed exploration of significant and non-significant estimates, signals a considerable level of uncertainty in the tail of the 'Other South Asian' J20-22 response model, primarily due to a lack of usable data points where residency levels >10%; with non-significant locales appearing to flat-line around zero.

In contrast, the J00-06 response model contains significant data points in locales where 25% of the children are of 'Other South Asian' ethnicity. Furthermore, model validity is preserved through the values presented at non-significant locales, which tend to broadly follow the defined J00-06 trend. Upon understanding this information, it is highly likely that 'Other South Asian' lifestyles may initiate URT complaints which if remain unaddressed may develop into LRT complaints, akin to what has been observed within the other socio-environmental influences. J00-06 GWR parameter estimates where $P \leq 0.05$, estimate the lifestyles of 'Other South Asian' residents as annually influencing 185 children's respiratory cases across Leicester during 2000-09, or 17.14% of children's J00-06 admissions. This value is almost twice as high as what was calculated by the complete set of significant and non-significant GWR parameter estimates (7.42%). Meanwhile, J20-22 GWR parameter estimates

where $P \leq 0.05$, recorded such residency levels to annually influence a staggering 416 children's respiratory cases, a figure which wildly deviates from the 4 cases recorded via GWR estimates of mixed significance. Further highlighting the inappropriate nature of the J20-22 trend across LLSOAs where the ethnic group no longer exists as a minority. One would hope to further develop the social group lifestyle stimulus-response models in future research, through expanding the study field to encompass several urban municipalities.

In terms of deprivation, the universal stimulus-response models would appear to once again provide a somewhat clouded relationship, with only the J20-22 subset trend capturing a viable explanatory relationship ($R^2=0.43$). In light of this uncertainty and the previous chapter's findings, it is quite feasible that deprivation once again influences upper and lower respiratory infections via several pathways associated with different elements of experienced social-economic burdens. Such thoughts are confirmed through a closer inspection of the complete set of significant and non-significant J00-06 and J20-22 GWR model outputs, with potential deprivation pathways of influence resembling a common trident structure (Appendix D11, D12), as previously observed when modelling the complete respiratory set (Figure 4.11). One should note that although the respiratory subsets share a mutual form, the scale of the tridents operation for the J00-06 subset occurs across a magnitude twice the size of what is recorded in regard to J20-22 hospitalisation rates. As previously discussed within Chapter 4, appropriate deprivation subset trends were applied on the following basis:

<p>TREND 1: >50% 'White British' Children & <3% Overcrowding TREND 3: <40% 'White British' Children & >5% Overcrowding TREND 2: All Other LLSOAs</p>

On these grounds the J00-06 3-trend universal deprivation-response model (Appendix D13), was found to associate deprivation as annually influence 241 children's respiratory cases across Leicester. This equates to 22.31% of the citywide J00-06 respiratory burden, a figure which effectively captures its corresponding 50NN GWR model estimate of 19.20%. For the J00-22 3-trend universal deprivation-response model (Appendix D14), 168 children's respiratory cases were annually attributed to factors of deprivation across Leicester. This equates to 39.53% of the citywide J20-22 respiratory burden, a figure broadly capturing its corresponding 50NN GWR model estimate of 42.77%.

The general findings from the GWR stimulus-response models indicate that socio-environmental factors influencing the outcome of URT and LRT infections operate through connected pathways, confirming earlier beliefs that prolonged exposures may wear down a child's respiratory system. In particular road-traffic emissions and levels of deprivation are

likely candidates responsible for severely exacerbating a communities deteriorating respiratory health. Nevertheless it would appear that lifestyle choices, such as those seen by Indian residents, can also mitigate the onset of such conditions.

5.4. CONCLUSIONS

Across a 10-year period, acute upper (J00-06) and acute lower (J09-22) respiratory infections were respectively observed to influence 41.65% and 23.66%, of respiratory hospital incidents experienced by children residing within Leicester UA. Such findings are relatively unsurprising, considering that the average person is likely to suffer from 5-7 episodes of acute respiratory infection per annum, with acute upper respiratory infections accounting for 30-35% of all new complaints presented to GP's (Cummings & Semple 1980).

Traditional dataset correlation tests, in the form of the Pearson's R (0.80) and Spearman's Rho (0.66), identified a substantial degree of linear association to exist between relatively severe (J20-22) and mild (J00-06) respiratory infections at the 95% significance level. Only limited associations were observed between the other major subsets affecting children's respiratory health across Leicester's LLSOA communities. Global Moran's I statistics indicated significant spatial autocorrelation to exist within the J00-06 and J20-22 respiratory subsets, up to and including third order LLSOA communities (approximately 1365m). Local Moran's I statistics detected major hot-spots to occur for both respiratory subsets within common inner-city localities, and some common cold-spots towards Leicester's northern and eastern peripheries. Several socio-environmental factors were observed to differ between hot-spots and their cold-spot counterparts, including residentially experienced TPM_{10} (+1.30 t/yr.), Carstairs Index scores (+6.13), smoking prevalence (+18.16%), and levels of Afro-Caribbean (+11.93%) or Indian children (-25.82%).

The significant spatial correlations of community J00-06 and J20-22 incidents presented within this study, likely represent the severest fraction of respiratory conditions (or iceberg of disease) attributed to 'Catarrhal Child Syndrome' (CCS). Such patient's repeatedly experience episodes of coughs and colds, as well as infections of the chest, ear and throat, thought to be caused by an immature immunological system responding to various external stimulants. As many as 80% of children consult general practitioners (GPs) with CCS from 5-6 years of age, with even the most debilitating symptoms (i.e. wheeze) tending to disappear once a sufficient level of natural immunity is established, typically within the first 10 years of life (Fry & Sandler 1993). In spite of the frequency of CCS, its causes have remained uncertain. With cases rarely isolating specific pathogens and only tentative links with allergens existing,

Fry & Sandler (1993) propose that underlying social and genetic factors likely govern the disproportionate burdens experienced in specific families.

Independent J00-06 and J20-22 OLS models, defining the concurrent effect of several socio-environmental variables on children's respiratory health, provided only a moderate goodness-of-fit to the data. Global regression modelling of both subsets recognised the significantly detrimental impact of TPM_{10} emissions and beneficial influence of Indian residents on a community's respiratory health. Yet unique to the J20-22 subset, levels of Afro-Caribbean residents and deprivation were shown to significantly increase children's respiratory risks at the citywide level. OLS models identified relative rises in residentially experienced TPM_{10} emissions to have the most prolific health impact on children, out of all explored socio-environmental influences. Localised regression modelling accounting for the spatially dependent nature of the dataset, substantially improved upon the goodness-of-fit reported by OLS modelling for the J00-06 ($R^2=0.73$, $CV-R^2=0.56$) and J20-22 ($R^2=0.72$, $CV-R^2=0.50$) respiratory subsets. This highlights the importance of contextual issues in producing spatially differing responses in magnitude or direction, to the same stimuli, across an urban environment. Nevertheless, median parameter estimates from GWR modelling appear to broadly compliment OLS outputs, indicating that global models have a complimentary role in summarising the general relationships of spatially dependent datasets.

The next step of this investigation involved the correlation of localised J00-06 and J20-22 admissions influenced by specific socio-environmental factors, as defined via GWR modelling. Pearson's R tests identified a substantial degree of linear association between modelled URTI and LRTI hospitalisations caused by TPM_{10} emissions (0.85), deprivation (0.76), and community levels of Indian (0.72) children. Bivariate Local Moran's I tests identified TPM_{10} emissions and levels of deprivation as likely candidates responsible for a communities deteriorating respiratory health. Exacerbations of J00-06 and J20-20 admissions associated with these two factors appeared to solely affect inner city communities, confirming the previously reported 'triple jeopardy' of social, health and environmental inequalities within Leicester (Jephcote & Chen 2012). Lifestyle choices, such as those seen by Indian residents, were also shown to mitigate the influence of detrimental socio-environmental factors, and in part reduce the potential spatial extent of such respiratory issues around other centric localities. Reasoning for the positive lifestyle choices of Indian residents, and the ability of certain ethnic minority groups to adapt better than others within Leicester, was discussed within Chapter 4.

Particulate induced health effects are understood to predominantly involve the destruction of cellular DNA via oxidative stress, followed by a natural inflammatory response,

which isolates then attacks both the foreign entity and surrounding area affected by its presence (Risom et al 2005). Yet, it has also been shown that exposure to pollutants can alter the hosts defence mechanisms, increasing the likelihood for infections to occur following an exposure episode. This secondary mechanism may be of far greater importance, when considering that a 3-year Australian Cohort of 263 infants detected viruses in 69% of non-hospitalising acute respiratory illnesses (Kusel et al 2006). Of these viral incidents, rhinoviruses were detected in 51.8% URTI's and 40.7% LRTI's, and respiratory syncytial virus (RSV) were detected in 8.6% URTI's and 15.2% LRTI's (Kusel et al 2006). A 2-year Finish Cohort exploring 293 children hospitalised with acute expiratory wheezing, detected a causative viral agents in 88% of cases (Jartti et al 2004). RSV was found in 54% of viral related infant admissions, whereas picornaviruses (including rhinoviruses) were detected in 82% of viral admissions for children age >3 years (Jartti et al 2004). Focusing on the lower respiratory tract illness, Psarras et al (2004) also reported the predominant influence of RSV on LRTI's in children <3 years of age, with the virus accounting for 50-90% bronchiolitis and 10-30% tracheobronchitis hospitalisations. Meanwhile in schoolchildren aged 9-11 years, Johnston et al (1995) detected upper respiratory viral infections in 80-85% of exacerbations of asthma, with picornaviruses (mostly rhinoviruses) accounted for two thirds of the viral infections.

The literature, would therefore suggest that age-specific viruses have a likely role in initiating, prolonging or exacerbating the detrimental effects of certain socio-environmental in establishing a child's respiratory infection. The seasonal decomposition of children's URTI's (J00-06) and LRTI's (J20-22) within Leicester from 2000-09, reveal nearly a 50-50 split for URTI's occurring within the hot and cold seasons (Appendix D15). This seasonal independence would indicate that socio-environmental factors, rather than viral uptake, are more likely to initiate a URTI episode. In contrast, 77.0% of LRTI's (J20-22) appear within the cold season when RSV and picornaviruses are most abundant (Appendix D15). For children residing within Leicester, it is proposed that exposure to detrimental socio-environmental factors may initiate URTI episodes, with prolonging recovery times likely occurring from sustained exposures. If a sufficient level of recovery is not reached in time for the cold season, then the child may become host to a viral infection exacerbating previous respiratory complaints, potentially resulting in lower respiratory tract conditions of greater severity.

Animal toxicology studies have consistently identified the immunosuppressive influence of pollutants in host susceptibility to viral and bacterial infections. Mice challenged with the influenza virus, ensuing a 6 month exposure period to diesel engine emissions ($2\text{mg}/\text{m}^3$), were shown to experience significantly higher levels of lung consolidation (61.5%) than their air-exposed counterparts (33.3%) (Hahon et al 1985). Rats infected with a strain of S.

pneumoniae, exposed to a 5-hour dose of concentrated ambient particulates ($65\text{-}150\mu\text{g}/\text{m}^3$), have also displayed bacteria burdens 300% above air-exposed subjects (Zelikoff et al 2003).

Human in-vitro investigations have indicated alterations in proinflammatory cytokine production of cultured bronchial epithelial cells following diesel exhaust particulate concentrations of $0.077\text{ - }0.33\text{ mg}/\text{mL}$ (up to 39x control value), potentially upsetting the immune homeostasis of the lung (Steerenberg et al 1998). Becker & Soukup (1999) record a 50% decrease in the uptake of RSV in human alveolar macrophages (AM) in the presence of PM_{10} , with PM_{10} exposure in the absence of infection significantly increasing the production of macrophage inflammatory proteins. This would imply that exposure to PM_{10} alters AM-regulated inflammatory responses to viruses, enhancing the spread of infection. Jaspers et al (2005) identify solutions comprising of $25\mu\text{g}/\text{cm}^2$ diesel exhausts to increase viral RNA levels in bronchial epithelial cells 80% above samples that had only been infected with the influenza virus. Yet in contrast to previous studies, levels of inflammatory proteins appeared unchanged, thus suggesting that oxidative stress generated by diesel exhausts acts as the primary mechanism for increases host susceptibility to viral infection. An investigation of 2,604 Washington State infants hospitalised with RSV-Bronchiolitis matched with a case-control cohort dataset, provides evidence for this immunosuppressive influence to occur within areas experiencing low ambient air pollution levels. A $10\mu\text{g}/\text{m}^3$ increase in $\text{PM}_{2.5}$ was associated with a 14% and 4% rise in bronchiolitis hospitalisations in RSV infected and non-infected infants respectively; residing within 150m of a freeway was also identified to increase the likelihood of bronchiole episodes by 7% in non-infected, compared to 17% in RSV infected infants (Karr et al 2009)

Previous investigations have reported childhood deprivation to have a long-lasting influence on adult lung function, thus corroborating with our findings of a causal link for deprivation and declining respiratory health during childhood. In a study of 3,911 women aged 60-79 years, material and social childhood socioeconomic influences on urban families including manual labour(-0.12l), limited car access (-0.10l) and shared bedrooms (-0.03l) were associated with reduced adult FEV_1 rates after adjustment for present lifestyles (Lawlor et al 2004). Spencer et al's (1996) inquiry of Sheffield's 1989/90 RSV outbreak identified 307 children with clinically suspected bronchiolitis. Children from the most deprived electoral wards were reported as 1.5 times more likely to be admitted and 1.4 times more likely to require medical intervention, than those living in other parts of the city.

The consequence of poverty is attributed to an amalgamation of material and social factors, which collectively play a central role towards explaining geographical variations in life expectancy at birth, across England and Wales (Woods et al 2005). Furthermore, it would

appear that inequalities in health have widened between the least and most deprived areas since 1971, through healthier individuals tending to migrate away from deprived areas whereas less healthy individuals are drawn into such areas (Norman et al 2005). It is believed that the housing stock of deprived communities plays a noteworthy role in declining health, with children residing in damp and mouldy dwellings experiencing an elevated prevalence of respiratory symptoms (Hopton & Hunt 1996, Platt et al 1989). However, improvements to living conditions have been shown to only prevent a further deterioration in health rather than bringing about health benefits (Hopton & Hunt 1996). Research exploring the financial aspects of deprivation indicates that social mobility may also only moderate, rather than create or amplify, social class differences in health (Blane et al 1998). This would suggest that if conditions are established, then they might prevail throughout adulthood.

Deprivation would also appear to have an influential role in the accessibility of appropriate healthcare to intervene the establishment of such respiratory issues, despite the fact that the NHS provides care based on need rather than ability to pay. A study of 284 wheezy children residing in London, identified a general lack of treatment for wheezing illness, yet marked differences in the uptake of anti-asthmatic drugs were still prevalent between the most affluent (41%) and deprived (10%) households (Anderson et al 1981). Furthermore, the effect of social class seemed to be explained by its association with the mother's mental health, thus providing an argument for a more 'holistic' approach to family medicine.

'The inverse care law', whereby the availability of good medical care tends to vary inversely with the need for it in the population served, likely explains healthcare issues within deprived communities. In a questionnaire of 3,044 NHS patients in Scotland, Mercer & Watt (2007) identified patients from deprived areas as having more issues to discuss (especially psychosocial), yet clinical encounter lengths were generally shorter. Furthermore, GP stress was higher because of high workloads and patient enablement was lower in encounters dealing with psychosocial problems in the most deprived areas, indicating that an inverse care law continues to operate within the NHS confounding attempts to narrow health inequalities (Mercer & Watt 2007). Patient's views on primary care in deprived areas have also indicated a need for holistic GPs who are competent, genuinely caring and understand the realities of life in such areas, in order to not be viewed as socially distant and emotionally detached (Mercer et al 2007). A report of 689 asthmatic subjects aged 11–59 years residing within Birmingham's deprived inner city wards, revealed the uptake and delivery of preventative health care to be of a poor standard, which worsened with gender and ethnicity (Moudgil & Honeybourne 1998). Only 45.4% persons understood the mechanisms behind the condition, 68.9% reported full drug compliance, and only 16.3% carried out self-management measurements. The lack of

tailored visits and an erosion of a doctor-patient relationship, previously reported offer one plausible explanation for this poor uptake of preventative care. Furthermore, future proposals for a centralisation of NHS critical care services have been shown to disproportionately affect poorer individuals, whereas increased investment in telephone services will predominantly benefit affluent populations, widening inequities in access to emergency care (Shah & Cook 2008).

The significant spatial correlations of community J00-06 and J20-22 incidents presented within this study, likely portray the severest fraction 'Catarrhal Child Syndrome' (CCS) cases, involving the susceptibility of a developing immunological system to respiratory infections. From this investigation, it is suggested that exposure to detrimental socio-environmental factors may initiate URI episodes, with prolonging recovery times likely occurring from sustained exposures. If a sufficient level of recovery is not reached in time for the cold season, then the child may become host to a viral infection exacerbating previous respiratory complaints, potentially resulting in lower respiratory tract conditions of greater severity. The findings presented here indicate that road-traffic emissions and levels of deprivation are likely candidates responsible for a communities deteriorating respiratory health. In contrast lifestyle choices, such as those seen by Indian residents, may potentially mitigate the onset of such conditions.

In spite of the frequency of CCS, its causes have remained uncertain; however it was previously believed that underlying social and genetic factors likely govern the disproportionate burdens experienced by certain families (Fry & Sandler (1993). The findings of this investigation would appear to confirm the existence of a link between extreme cases of CCS and certain socio-environmental influences. Additional research is recommended to confirm such findings.

ANALYSIS OF NATURALLY OCCURRING BOUNDARIES IN CHILDREN'S RESPIRATORY HEALTH AND ASSOCIATED SOCIO-ENVIRONMENTAL INFLUENCES: LEICESTER UA 2000-09

OVERVIEW

Chapters 4 and 5 previously established that certain segments of society uniquely experienced a 'triple jeopardy' of social, environmental and respiratory burdens, throughout childhood. Furthermore, a particular set of socio-environmental variables were identified to individually influence relatively minor and severe respiratory complaints through a shared spatial and arithmetic pathway, indicative of a mechanised decline in health from continued exposures. Upon defining such dose-response relationships, this chapter moves onto the second stage of the research project, which will entail the examination of spatial field's in-order to define the prevalence of critical distance-response connections between respiratory and socio-environmental phenomenon.

Health boundaries are of intrinsic medical interest, in that they reflect the geographic extent and intensity of underlying physical and or social processes, identifying populations whom are most likely to be at risk from a fluctuating health front. Traditionally, the spatial extent of health impacts associated with road-transport pollutants have been explored through the examination of artificially created buffers, defined by subjective distances from specified major road links. Within this chapter an alternative approach is presented using boundary statistics, which describe naturally occurring shifts of magnitude in socio-environmental and health outcomes across the wider urban area. To date, distance-threshold techniques have solely explored the response environmental attributes, without considering the combined influence of additional social burdens. This chapter covers objectives 1, 2 and 6 of this project outlined in Chapter 1.

6.1. INTRODUCTION

Across the urban environment, road-transport constitutes as the predominant source of outdoor air pollution, emitting a concoction of air quality objective pollutants and carcinogenic hydrocarbons within close proximity to residential districts. Simplistic approaches towards distinguishing unique community exposures to traffic pollutants, commonly involve the utilisation of surrogate measures such as residential proximity to major road links, with questionnaires or spirometry tests measuring diminishing respiratory functions in relation to distance (Oosterlee et al 1996, Brunekreef et al 1997). A recent proximity based study inspecting 8-years of lung development within Californian children, identified residents 500m from freeways to experience a decline in Forced Vital Capacity of -63ml, diminishing to -19ml by 1000-1500m (Gauderman et al 2007).

Maantay (2007) investigated the occurrence of asthma hospitalisations in relation to air pollution across the Bronx, NY, through applying established environmental agency proximity standards to construct environmental hazard buffers. Approximately 66% of the Bronx's land mass fell within the buffers, which were predominantly occupied by ethnic minorities (88%) and persons living below the federal poverty level (33%). Children residing inside these pollutant buffers were also 11-17% more likely to be admitted to hospital (Maantay 2007). Within a European context, the Health Survey for England datasets nationally identified respiratory outcomes across 6,015 children aged 7-15 years to become exacerbated across extremely localised distance bands from main roads after adjustment for sex and deprivation. Here, Adjusted Odds Ratios (AOR) using the 120-150m band as a benchmark, identified wheeze, asthma and allergic rhinitis symptoms to progressively increase with proximity, reaching respective AOR's of 1.62, 1.35, and 1.14 for children residing <30m from major roads (Pujades-Rodriguez et al 2009).

Thus, it is well documented that exposure to elevated levels of air pollution causes acute respiratory distress, with such effects tending to become exacerbated within artificially created buffers that are of closest proximity to specified major roadways. However, it remains unclear whether exposure in the every-day environment to naturally occurring zones of rapid change in pollutants, considerably contribute towards the spatial existence of marked boundaries in respiratory health (Jacquez 1995). Health boundaries are of intrinsic medical interest, in that they reflect the geographic extent and intensity of underlying physical and or social processes (i.e. pollutant plumes, social attitudes etc.), identifying populations whom are most at risk of developing new conditions from a fluctuating health front in light of a particular socio-environmental event. This chapter intends to address such issues through the application of geographic boundary analysis techniques, which define the natural occurrence and

magnitude of objects across spatial fields. After which, the degree of overlap between variables of interest is accomplished by null spatial models, which unlike pattern recognition techniques, are able to ascertain whether such thresholds of spatial influence are in some way statistically unusual.

Studies involving the detection and overlap of naturally occurring geographic boundaries have been widely applied within the fields of genetics and ecology (Barbujani et al 1989, Fortin & Drapeau 1995, Hall 2008), for the evaluation of locations portraying an amalgamation of biological, physical and social processes at work. However, few studies have explored the tools application within the wider fields of epidemiology and public health, with applicable studies tending to focus solely on issues concerning the late-stage diagnosis and mortality rates of cancer patients (Jacquez & Greiling 2003).

Boundary analysis methods are favoured over conventional proximity studies because such techniques determine the exact critical distance threshold between substantial gradient shifts in air pollutants and health outcomes without the user testing relationships across arbitrary distances. Furthermore, traditional proximity studies of pollutants on health outcomes have treated supplementary social circumstances purely as confounding measurements, for which populaces may require necessary adjustments. In contrast, boundary analysis methods can be used to define zones of rapid change across multiple socio-environmental variables of interest, which may then be compared to health boundaries. This is of particular importance when considering that specific communities tend to experience a 'triple jeopardy' of social, health and environmental inequalities (Pearce et al 2010). Finally, such techniques should be viewed as a more appropriate form of analysis across urban environments, which contain vast road networks and experience complex transport flows. In some cases, congestion on minor roads close to residential areas perhaps poses a higher respiratory health risk than what would be experienced along fast-flowing major roads. Therefore it would be of greater interest to explore naturally occurring urban pollutant gradients rather than focusing on a specific major roadway.

6.2. UNIVARIATE BOUNDARY DETECTION & VALIDATION

Polygon wombling is interested in the exploration of whether two adjacent census areas, separated by a common border (CBE), portray dramatically dissimilar responses for the individual or set of particular variables in question. In terms of health outcomes, boundaries are of particular importance in understanding whether the presence or disappearance of

extreme values in influential socio-environmental phenomenon(s) may drastically increase levels of risk. Univariate crisp polygon wombling techniques described within Chapter 3 were applied to detect boundaries across children's respiratory hospital admissions, traffic emissions and the other individual social variables within BoundarySeer 1.3.13. Within the Leicester dataset, approximately 25 out of the 508 CBEs were defined as BEs for each respiratory and socio-environmental variable of interest. Upon viewing Figures 6.1 and 6.2, one may see that that BEs for all of the explored socio-environmental and health variables fall well within the right tail of their respective BLV distributions, which would suggest that that the detected boundaries are of an appropriate nature.

For children's J00-99 admission rates, boundary elements were characterised to contain a BLV ranging from 0.06 to 0.58 (Figure 6.1). It should be noted that the raw dataset values recorded at each LLSOA observation are normalised on a scale of 0-1, prior to calculating the Squared Euclidean dissimilarities of the applicable CBE's. As a result, levels of dissimilarity for a univariate boundary analysis may never exceed a value of 1, with such a procedure allowing for a direct comparison of dissimilarity strength across multiple variables. For children's J00-99 admission rates, the six largest levels of dissimilarity were found to encase the southern, eastern and western faces of the focal inner-city LLSOA identified to be of prior interest in Chapters 4 and 5. In-fact these six boundaries were recorded to provide an average BLV of 0.47, with all eight boundaries encircling this particular LLSOA providing a BLV 0.14. One may note that adjacent to the north-eastern face of this community lie two LLSOAs, which may be viewed as an extension of this primary respiratory health front. Within this secondary cluster the six containing boundaries exhibit an average BLV of 0.18, which is well above the criteria threshold, yet somewhat diminished upon the values recorded around the inner-city focal point. This would suggest that a spill-over of the immediate detrimental influences appears focused in a north-westerly direction. One may also notice that a staged transition in deteriorating respiratory health appears to occur towards the south of the inner-city focal point, whereby a second set of three weaker boundary's record an average BLV of 0.06 in marking a transition from moderately high to low symptom outbreaks outside of the city centre.

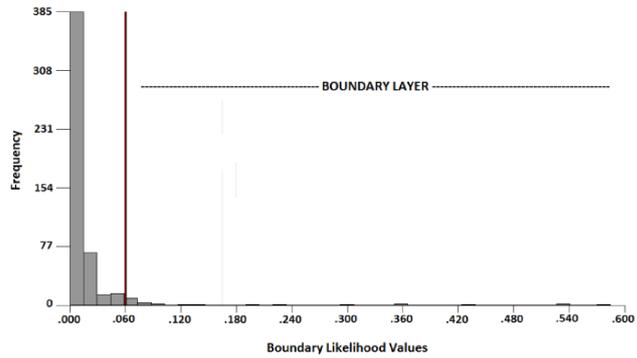
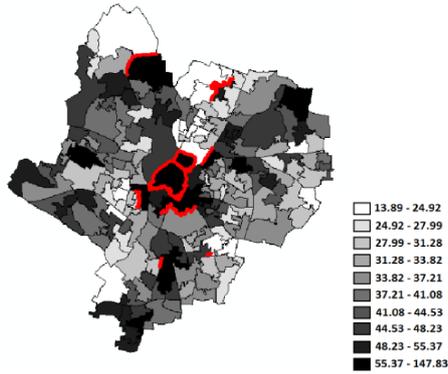
Meanwhile, boundaries in the spatial spread of children's J00-06 admissions were classified through a comparatively weaker threshold (BLV 0.03). Yet at the same time, such boundaries exhibited a range of more extreme dissimilarities, reaching a maximum BLV of 0.73. The largest set of dissimilarities were found to entirely surround the inner-city LLSOA of prior interest, of which BLV values for these eight boundaries were 0.30, providing an average BLV value of 0.52 (Figure 6.1). In contrast, two of the northern boundary faces of this LLSOA

were associated with BLV's as low as 0.23 and 0.14 when exploring the J00-99 spatial field. As before, two LLSOAs adjacent to the north-eastern side of this focal point appeared entirely encased via six boundaries exhibiting an average BLV of 0.17. Likewise, a staged transition in deteriorating URT health appears to occur towards the south of the inner-city focal point, whereby a second set of four boundaries provide a weaker but still substantial average BLV of 0.06. Unique to the J00-06 subset, the outer shell of this step-wise transitional zone also reveals itself along the eastern face of the inner-city, of which four boundaries providing a somewhat weaker average BLV of 0.05. Additional deviations from objects relating to the complete respiratory set, involve the addition of two boundaries running along a LLSOA within the ward of Western Park, which contains several education facilities. Of particular note is the disappearance of three J00-99 boundaries around a single northern LLSOA positioned adjacent to the Melton road, which appears represent a high outlier for J00-99 cases but not for the individual J00-06 or J20-22 subsets.

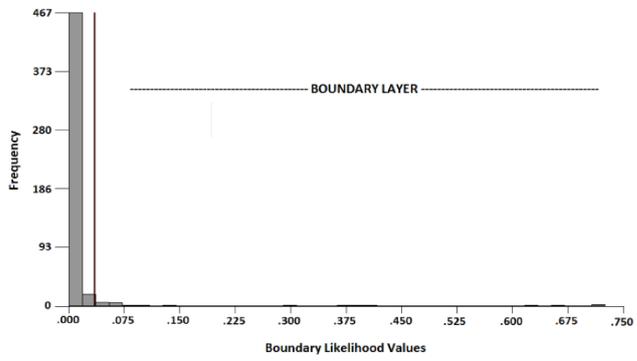
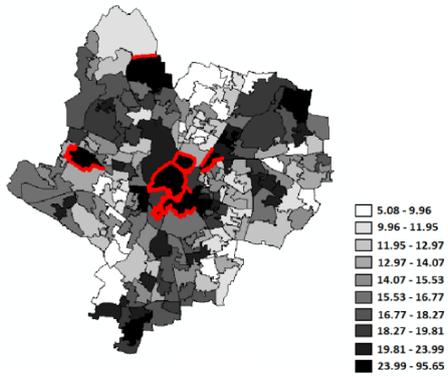
Children's J20-22 admission rate BEs were associated with a BLV's ranging from 0.06 to 0.78, of which the five largest dissimilarities were found to encase the eastern and western faces of the inner-city LLSOA of prior interest (Figure 6.1). BLV values 0.10 were associated with this LLSOA, representative of a weaker rate of change than what was observed for URT conditions. Nevertheless, the entire eight boundaries of this LLSOA produced an average BLV of 0.45, a value marginally below prior health evaluation figures. Two LLSOAs adjacent to the north-eastern side of this focal point are once more of interest, of which the six boundaries encasing the cluster provided BLV's ranging from 0.10-0.38, averaging a value of 0.21.

As with prior respiratory complaints, a staged transition in deteriorating LRT health appears to occur towards the south of the inner-city focal point. However, this time the outer zone encompasses the wider southern portion of the outer city centre, with the five boundaries in question also providing a somewhat grander average BLV of 0.08. In contrast, the J20-22 subset differs from the overall respiratory set through the addition of four boundaries towards the cities eastern periphery, in locales somewhat representative of the terminal and major junctions preceding the missing link of the cities outer ring-road. These boundaries support the findings of Chapter 5, which show that LRT cases are particularly influenced by regular spatiotemporal pollutant episodes that occur at peak times.

J00-99 Admissions Per 1,000 Children



J00-06 Admissions Per 1,000 Children



J20-22 Admissions Per 1,000 Children

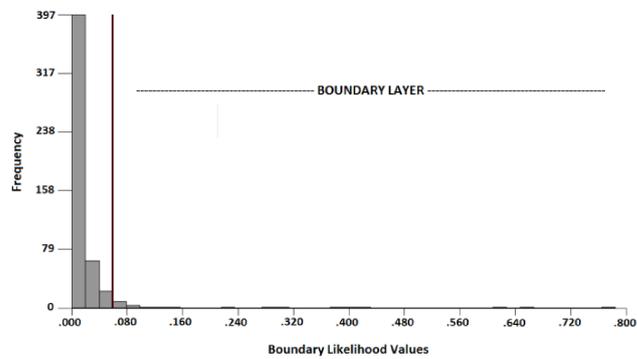
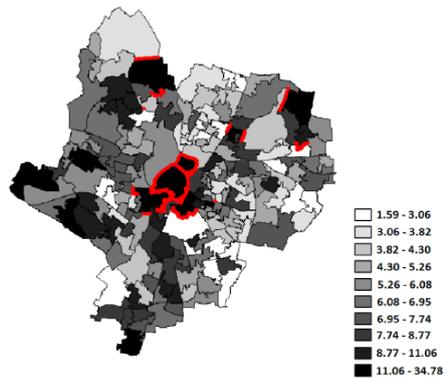
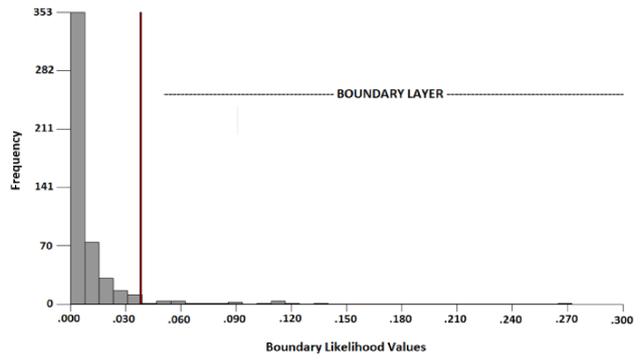
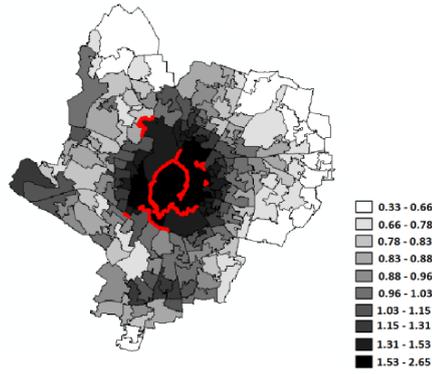
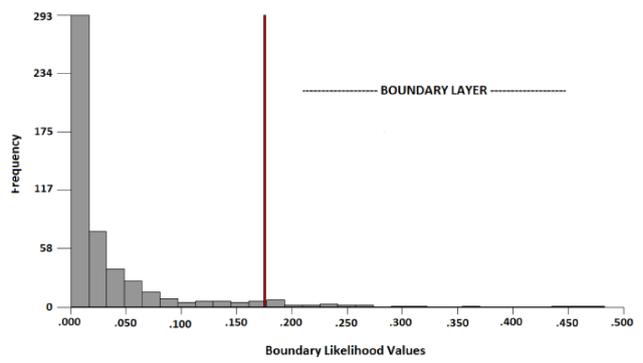
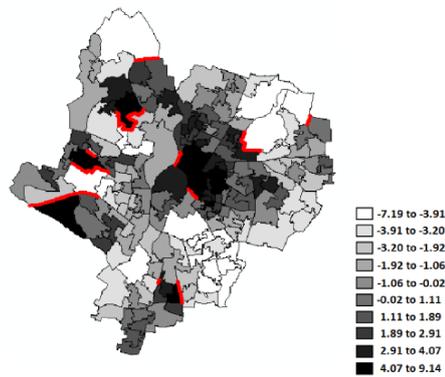


FIGURE 6.1: Maps illustrating the top 5% of Boundary Elements (red) across respective decile distributions of annual average children's respiratory admissions (J00-99, J00-06, J20-22): Leicester 2000-09

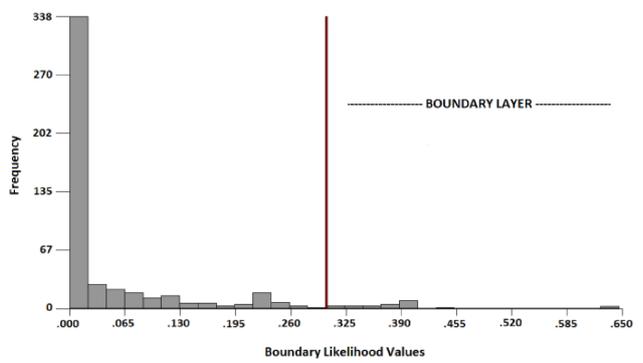
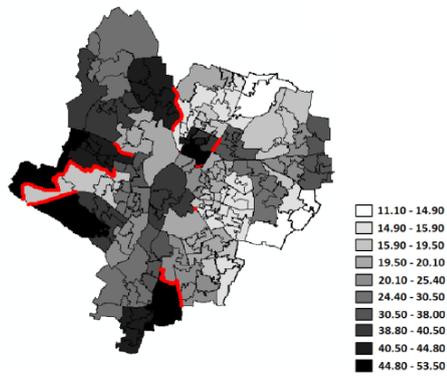
TPM₁₀ Emissions (t/yr.)



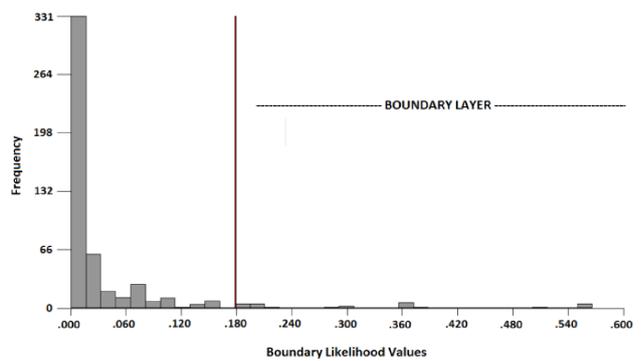
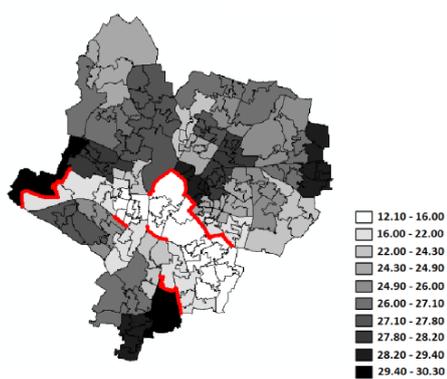
Carstairs Index [Leicester]



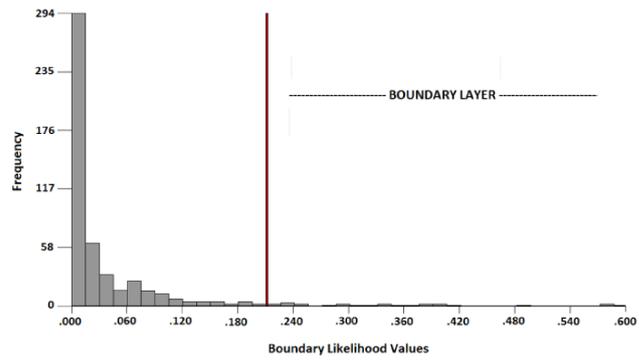
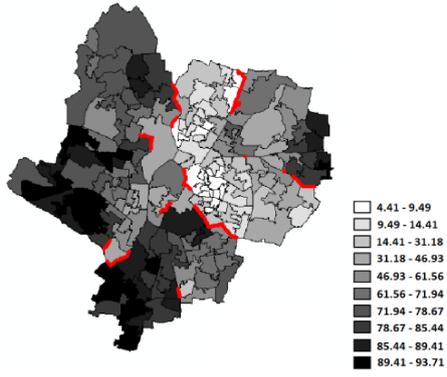
Adult Smoking Prevalence (%)



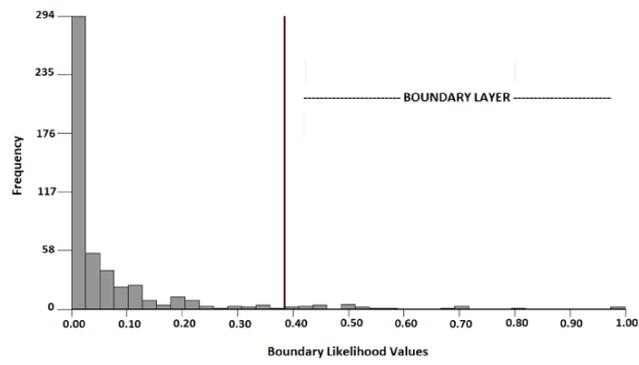
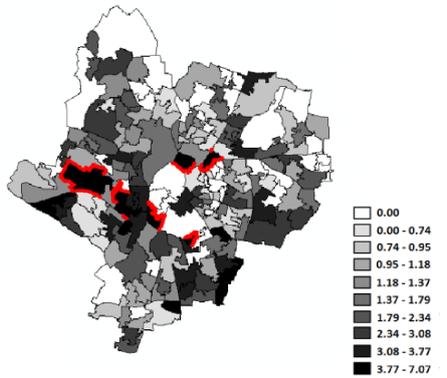
Adult Obesity Prevalence (%)



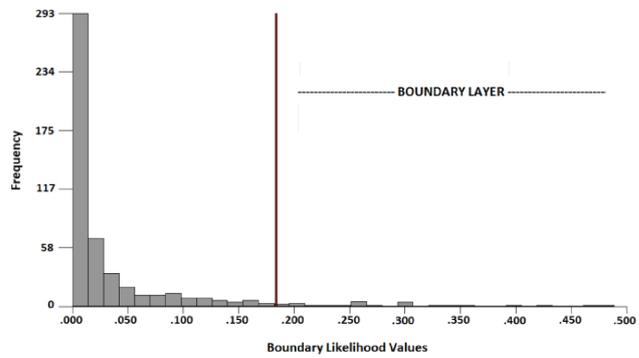
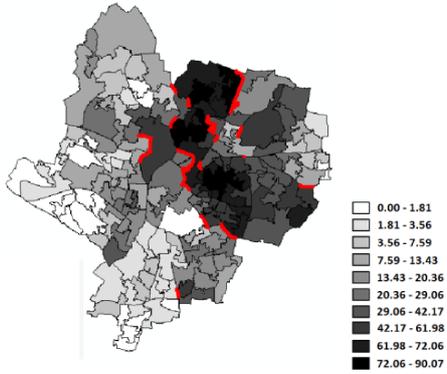
White British Children (%)



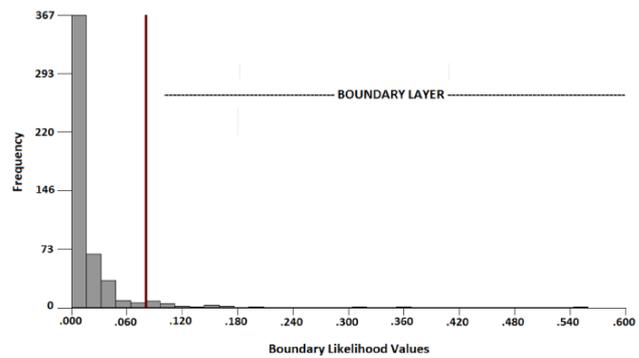
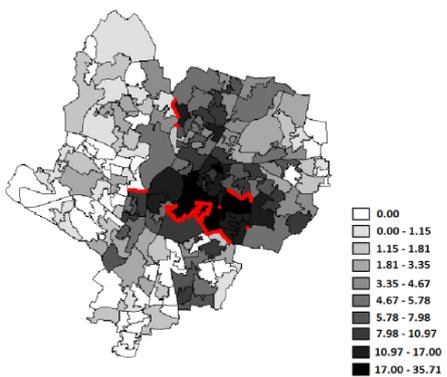
White Non-British Children (%)



Indian Children (%)



Other South Asian Children (%)



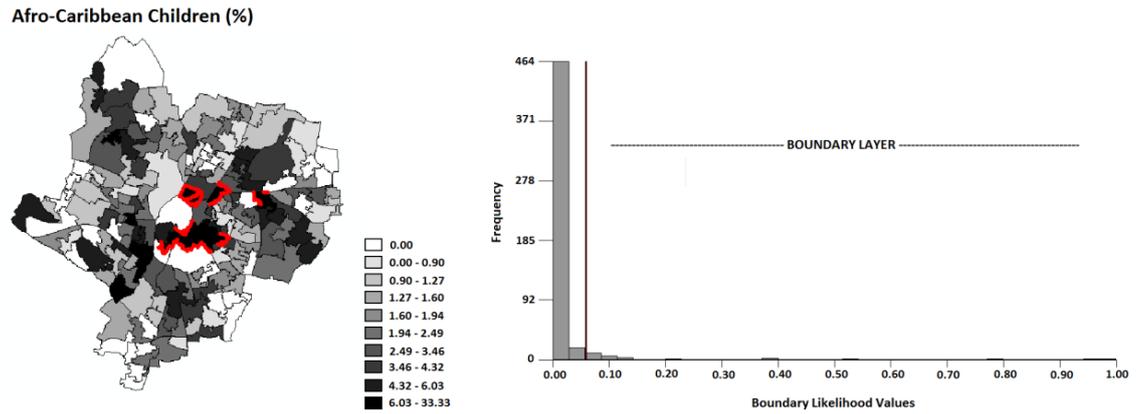


FIGURE 6.2 [PAGES 229-231]: Maps illustrating the top 5% of Boundary Elements (red) across respective decile distributions of environmental and socioeconomic-ethnic independent variables previously identified to be of interest of via spatial regression models

TPM₁₀ emission boundaries were associated with BLV's of 0.04-0.27, with their distribution once again focusing upon Leicester's inner city locales (Figure 6.2). One may note that a primary set of boundary's containing the respiratory LLSOA of interest and two other adjacent southern communities, separates extremely high from high pollutant zones. It would then appear that several fragmented outer boundaries represent a shift from high to moderate pollutant zones, suggesting that a two-phase boundary process operates across Leicester's central district. Such a system was just reported in relation to respiratory outbreaks, which would imply that spatial transitions in pollutant levels are a key influence in determining respiratory health gradients.

The largest reported TPM₁₀ BLV (0.27) may be found on the north-western face of the central LLSOA denoting a shift from extremely high to high pollutant zones. The second and third highest BLV's exist within the inner (0.13) and outer (0.12) boundary sets towards the south of this locale, marking smaller differences in residential TPM₁₀ exposure levels. It may be noted that the normalised differences between pairs of LLSOA TPM₁₀ emission exposures are of a slightly reduced value compared to other socio-environmental and health variables (Table 6.1). A certain degree of smoothing was inadvertently introduced through the spatial interpolation of the 1x1km NAEI emission dataset to the LLSOA level. Nevertheless, the distribution of BLV's would suggest that the reported boundary elements clearly represent rapid transitions, in the wake of plausible instances of over-smoothing.

In contrast, Carstairs Index boundaries provided BLVs of a consistently moderate strength ranging from 0.18-0.48. Furthermore, there is a less centric focus, with boundaries in relative levels of deprivation appearing to exist predominantly in several key pockets across the cities western periphery (Figure 6.2). Some minor formations are also observed extending

out from the inner city focal area and across the eastern fringes of Leicester's central district. The highest deprivation related BLV (0.48) was identified along the border of Western Park and New Parks, with the second highest BLV (0.46) also located within the western district, this time along the border of Braunstone and Western Park. However, the third highest BLV (0.46) is located in the inner city, where Spinney Hills and Abbey meet, in the area identified as a direct extension of the city's leading respiratory health problems. Meanwhile the fourth (0.36) and fifth highest deprivation BLVs (0.35) were identified along a minor cluster of northern LLSOAs along the border of Beaumont Leys and Abbey, which appear to coincide with respiratory health gradients outside of the crucial inner-city zone. It would therefore appear that determinants of socio-economic status unlike other key burdens are not location restricted, but they rather operate throughout the city in a way that regulates moderate shifts in respiratory health.

Boundaries in levels of adult smoking prevalence, representative of passive smoking exposures during childhood, appear to operate almost exclusively across peripheral areas positioned within the western half of city (if one was to split the city into two). These boundaries contain moderate-high BLV's of 0.30-0.65, which form in relation to the visually clear-cut spatial contrasts of alternate extremes in the variables decile structuring. The largest differences in smoking prevalence are identified to occur across Leicester's western periphery, where Braunstone separates from New Parks. Here two boundaries produce a substantially high average BLV of 0.65, representative of the obvious difference between third order and tenth order smoking deciles. Meanwhile, the third to sixth largest magnitude boundaries are positioned towards Leicester's northern periphery, where they produce an average BLV of 0.42. Once more, such high dissimilarities are produced by sharp spatially extensive differences between second and ninth order deciles.

Likewise, adult obesity boundaries thought to be indicative of childhood activity and dietary intake, appear to exist between neighbourhoods exhibiting a sharp contrast in decile classification, as indicated by BLV's of 0.18 - 0.57. The six largest shifts in magnitude are recorded along the north, eastern and western faces of the inner-city focal point, which itself experiences extremely low obesity levels (second decile) compared to the adjacent communities (seventh+ decile); therefore it is of little surprise that the average BLV of these six boundaries is logged as 0.56. The second most striking transition between two homogeneous clusters of low and high obesity rates may be observed across Leicester's southern periphery, however this occurs at a far lower magnitude as observed by an average BLV of 0.37 across the six boundary elements in question.

Children of 'White British' ethnicity are observed to predominantly reside within the cities western and southern quarters, with transitional zones typically occurring across central regions of Leicester (Figure 6.2). In particular, one may note that a section of boundaries run from north-to-south through inner city locales almost to create an interlocking structure, segmenting the eastern half of the city from the west. This structure consists of thirteen boundary elements, which provide an average BLV of 0.40. Furthermore, four of these boundaries are logged as containing the highest BLV's recorded for this particular ethnic group. Meanwhile, in this partitioned eastern zone a second peak of 'White British' children may be observed along the city limits, around the missing link of the cities outer ring-road. This reoccurrence of 'White British' residents would appear to form a banded structure, in which rapid transitions in minority groups are universally observed as one moves towards inner city locales. Overall, boundaries in 'White British' children were recorded to range from 0.21 to 0.60.

In contrast, levels of 'White Non-British' children were observed to be high across multiple pockets of minor clustering, typically in the form of a singular LLSOA positioned outside the inner-city. Because of such high outliers, BLV's for 'White Non-British' children were recorded to start from 0.39, in some circumstances reaching the maximum value of 1.00. In particular, a high residency cluster across a portion of Western Park contains six out of the top eight BLV's for this social group, producing an overall average BLV of 0.72. Around the inner-cities fringes, three minor transitional pockets may be observed, of which the two northern boundary zones provide average BLV's of 0.52 and 0.46, with the southern cordon providing an average BLV of 0.49.

For 'Indian' children, one may note that a section of boundaries run from north-to-south around inner city locales almost matching the dividing structure between 'White British' residents and ethnic minorities. This arrangement is constructed from twelve 'Indian' boundary elements, which together provide an average BLV of 0.31. Such a value is noticeably smaller to what was recorded in relation to shifts in 'White British' residency levels, indicative of the selective integration between 'Indian' and other ethnic minority groupings discussed with Chapters 4 and 5. In this respect, these boundaries in 'Indian' residency represent a clear separation between 'White British' and ethnic minority children. However one should note that ten boundaries illustrate fluctuating levels of 'Indian' residency across the eastern half of the city of a similar magnitude, as recoded in an average BLV of 0.29. Yet such boundaries would tend to relate to interactions between other ethnic groups, thus suggesting that aspects of segregation are also likely to occur between minority groups. Overall, boundaries in 'Indian' children were recorded to range from a low 0.18 to a moderate 0.49.

Boundaries in 'Other South Asian' children's residency levels were associated with BLV's of 0.08-0.56. These boundaries are shown to almost exclusively mark the fringe of the south-eastern section of Leicester's inner-city, with minor boundaries also capping the northern section of this ethnic group's residential areas of choice throughout Stoneygate. These five northern boundaries provide an average BLV of 0.12, which would imply that whilst reductions in 'Other South Asian' residency levels occur in relation to other minority groups, such changes are relatively minor and occur in a relatively smooth fashion. Two other minor transitions in 'Other South Asian' residency levels may be observed around a northern LLSOA within Belgrave (BLV 0.1), and within a LLSOA east of the inner-city focal point of prior interest (BLV=0.08).

Meanwhile, boundaries in 'Afro-Caribbean children's residency levels were also defined in relation to low BLV's of 0.06, yet in some circumstances displayed extreme dissimilarities in neighbouring residency levels reaching the maximum univariate BLV value of 1.00. Specifically, a cluster of two LLSOAs recognised to represent the northern extent of respiratory boundaries is observed to be completely encased by this groups top six boundary elements, which deliver an average BLV of 0.67. Interestingly levels of Afro-Caribbean residency are recorded to be 0.00% within the inner city focal point, which would suggest Afro-Caribbean residents to be strongly removed from the main driving forces behind a child's debilitated respiratory responses. In particular, chapters 4 and 5 previously established TPM_{10} emissions to cause a localised burden of an extreme magnitude unique to this specific LLSOA. Despite this the two adjacent north-western LLSOAs housing large numbers of 'Afro-Caribbean' children, falls well within the general pollutant zone of concern, in addition to sharing common elevated boundaries with all respiratory conditions under investigation.

Still, a secondary spatial expanse of ten boundaries is observed towards the southern fringes of the outer-city centre, recording an average BLV of 0.12 in relation to 'Afro-Caribbean' residency levels centrically increasing from a first to tenth order decline. One may recall that such a boundary is in-line with the staged transition in deteriorating respiratory health and elevated pollutant levels. Collectively, these observations reveal that although 'Afro-Caribbean' children are not exposed to extreme values of TPM_{10} fallout, yet their residency remains strongly associated with unfavourable exposure levels. Furthermore, one should not rule out that the lifestyle of this social group may also have a minor role in influencing the extent to which these respiratory outcomes occur.

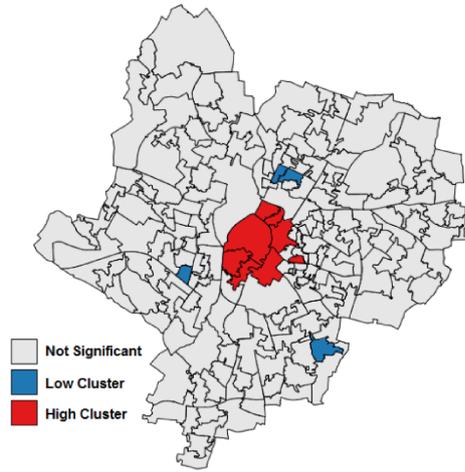
Local indicators of spatial autocorrelation (LISA) in the form of a first order neighbour Getis-Ord G_i^* statistic were subsequently conducted, in-order to externally cross-validate whether the edges of spatially homogeneous pockets correspond to our detected boundaries

(Figure 6.3, Appendix E1). Unlike the Local Moran's statistic which detects regions of homo and heterogeneity, the Getis-Ord G_i^* statistic simply categorises spatial clusters to represent either 'hot' or 'cold' spots dependant on the direction in which the local statistics substantially deviates from the global statistic; In contrast, the Local Moran's statistic compares the local situation to that of its neighbours. It is believed that the less descriptive Getis-Ord G_i^* statistic presents a more conservative approach for upper tier boundary validation, in that the prominence of local outliers will often be restricted by its global threshold.

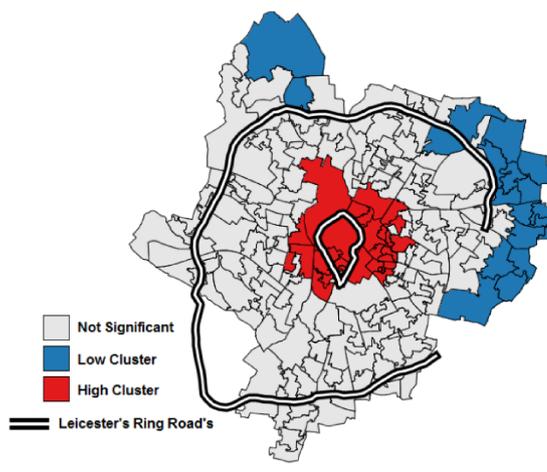
The G_i^* statistic identified the existence of a significantly high clustering of children's J00-99 respiratory hospital admissions within the city centre of Leicester, which closely mimic the outer range of wombled boundaries (Figure 6.1, 6.3). In-fact, observed annual J00-99 rates across the inner city zone of high clustering averaged 76.01 admissions per 1,000 children, which is distinctly higher than Leicester's LLSOA global average of 39.43 admissions per 1,000 children. Furthermore, the south-eastern cold-spot located within Knighton is marked by a boundary on its northern face; whilst the Westcotes cold-spot appears to represent a central low point, for which a proximal boundary falls neutrally in-between this and the inner-city hot-spot of concern. Nevertheless the northern boundary sections around Belgrave and Beaumont Leys, appear to have gone undetected by the G_i^* analysis, which might suggest that the 5% threshold could be restricted further.

Yet if one was to recall the Local Moran's J00-99 outputs (Figure 4.2), they would notice that the Belgrave boundaries define a highly outlying locale, and are therefore of an appropriate nature. Such observations just confirm ones earlier opinion that the G_i^* analysis offers a stricter inquiry for spatial objects. As with the entire respiratory set, G_i^* outputs for the individual J00-06 and J20-22 subsets successfully illustrated their respective inner-city expanses of homogeneity, in-line with the main polygon wombling analysis. Yet one may note that other external boundaries for both subsets have remained undetected, yet from the quantile plots still represent exceptionally localised spatial shifts albeit of a reduced magnitude.

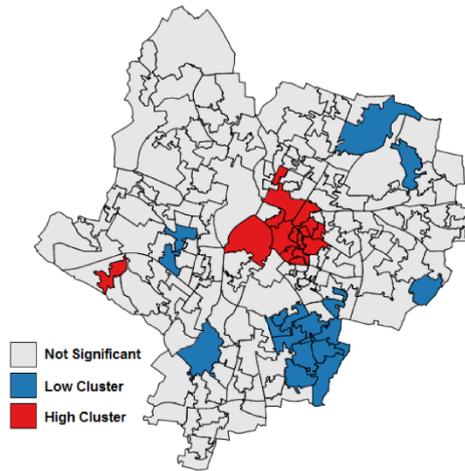
J00-99 Admissions Per 1,000 Children



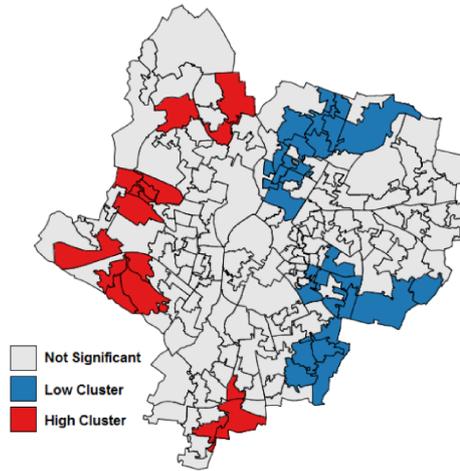
TPM₁₀ Emissions (t/yr.)



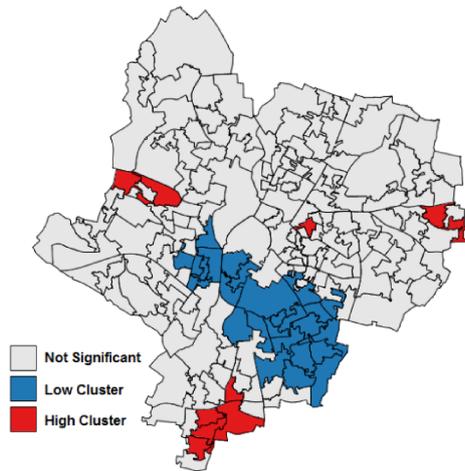
Carstairs Index [Leicester]



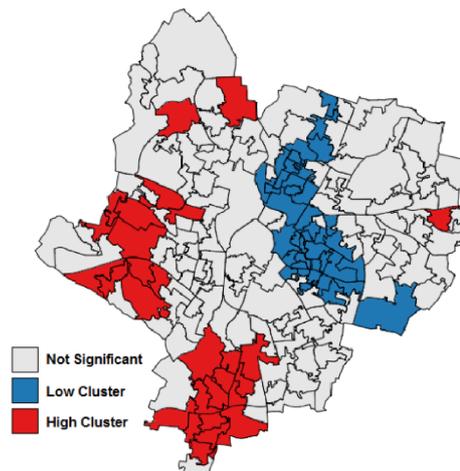
Adult Smoking Prevalence (%)



Adult Obesity Prevalence (%)



White British Children (%)



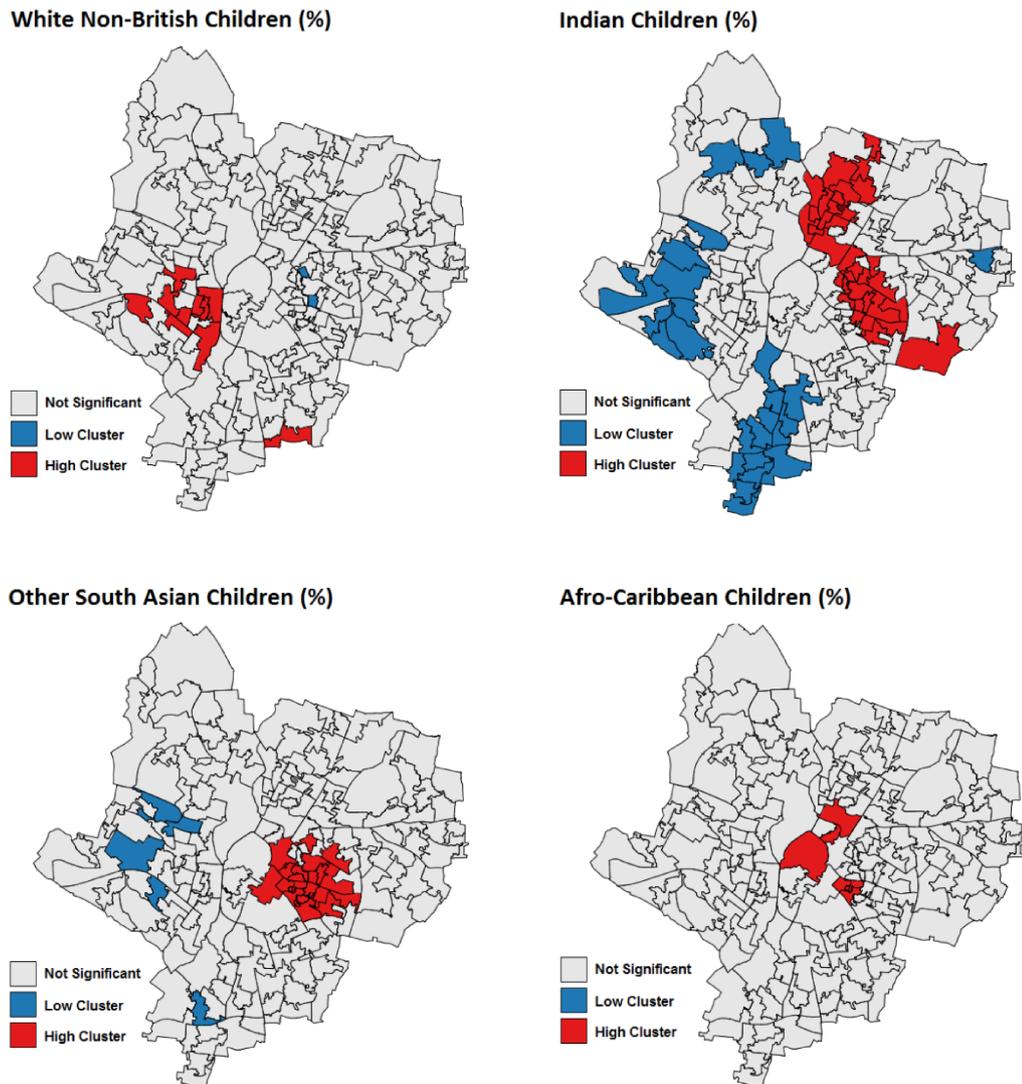


FIGURE 6.3 [PAGES 236-237]: Getis-Ord G_i^* hot-spot analysis ($P = 0.05$) of annual average children's admissions of the complete respiratory set (J00-99), and other socio-environmental independent variables explored by the boundary analysis

Of particular spatial importance is the distribution of PM_{10} road-transport emissions (TPM_{10}), which also exist at elevated levels within the inner city, extending slightly beyond the eight LLSOAs coined as hot-spots for children's respiratory cases (Figure 6.3). This area of interest contains the city's inner ring-road (A594) and several key arterial roads linked to the M1 and the city's outer ring-road (A563). Residentially experienced TPM_{10} emissions levels within the hot-spot of respiratory cases were identified to occur at levels 49-154% higher than average citywide emission rate of 1.04t/yr. It would seem as if the primary components of the inner-city TPM_{10} emission boundaries closely depict the circular extent of Leicester's inner ring-road. Meanwhile the G_i^* cold-spots have not been represented by boundary detection, due to their smooth transitional nature as depicted in Figure 6.2's TPM_{10} decile plots.

As previously reported in Chapters 4 and 5, a crossover between children's respiratory cases and TPM₁₀ emissions, with Carstairs Index deprivation levels may also be observed from the hot-spot analysis; thus presenting the potential avenue for a double burden of social and environmental issues collectively impacting on a child's wellbeing. In particular affluent areas along the cities south and eastern peripheries averaging Carstairs Index scores of -4.06, experience TPM₁₀ emission levels approximately 0.11t/yr. below typical citywide rates, and record 30.42 J00-99 admissions per 1,000 children. Whereas the central deprivation hot-spot presents an average Carstairs Index score of 4.62 and TPM₁₀ emissions 0.60t/yr. above typical residential exposures, which coincide with a J00-99 burden of 50.27 admissions per 1,000 children. However in contrast to the two other described elements, a comparison of boundary and Gi* analysis outputs would imply that issues of deprivation present themselves across a much smoother and expansive spatial scale (Figure 6.2, 6.3). Here boundaries appear within the nearby vicinity of the north-eastern and south-eastern cold-spots, which are thought to not represent the edge of homogeneous areas, but rather the heart of these smooth spatial shifts in magnitude. The construction of polygon boundaries along Leicester's western periphery, whilst not clearly defined, also remain plausible in that minor central elements of alternative homogeneous clusters are documented by the Gi* analysis. Nevertheless, several north-western boundary elements remain unconfirmed by hot/cold spot outputs.

The potential existence of environmental inequalities may be further brought into question upon examining the distribution of Leicester's ethnic minority groups, with 'Afro-Caribbean', 'Indian' and 'Other South Asians' tending to reside within inner city areas denoted to experience elevated deprivation and traffic emissions levels. Across Leicester, 'Afro-Caribbean', 'Indian' and 'Other South Asian' residents aged 0-15 years, respectively account for 2.67%, 30.00% and 5.88% of this age groups citywide populace. However within their individual inner city double-burden hot-spots, 'Afro-Caribbean', 'Indian' and 'Other South Asian' children correspond on average, to 10.27% 55.59% and 20.49% of each LLSOAs residential populace. Furthermore, children of White-British ethnicity, on average only explain 15.52% of the residential populace across inner city LLSOAs defined by the hot-spot analysis as experiencing elevated deprivation and TPM₁₀ emission levels. Although regression modelling indicated children from 'Indian' communities to globally experience fewer respiratory admissions, spatial analysis identified a tendency for this community to reside within areas experiencing overruling adverse socio-environmental influences. These observations would imply that certain ethnic minority groups experiencing environmental injustices possess favourable social abilities to mitigate such health outcomes.

The previously reported 'White British' boundaries describing sharp transitions in relation to levels of ethnic minority residency appear to be clearly recognised by the Gi* analysis (Figure 6.2, 6.3). In-fact, boundary locations appear positioned precisely along the fringes of the ethnic enclave band, which runs from north-to-south, cutting through sections of central Leicester's problem area. The Gi* statistic confirms that this banded structure largely relates to increased levels of 'Indian' residency. Likewise, this ethnic groups strongest boundary structures closely mimic both sides of this residency band, with a minimal detection of magnitude shifts around residential cold-spots. One may notice that several 'Indian' boundaries also exist within this band principally relating to shifts in 'Afro-Caribbean' residency. Gi* outputs describing the residential tendencies of 'Other South Asian' and 'Afro-Caribbean' residents, would also appear to confirm the positioning of boundary elements. Yet, areas deemed to house low levels of 'Other South Asian' children, as with other ethnic minority groups, were found to inadequately differ from neighbouring locales to warrant a boundary placement. This would confirm that ethnic minority children have a strong residential disassociation with 'White British' children, whereby they almost universally reside within the eastern quadrant the city. However, the one exception to this rule involves the settlement of 'White Non-British' residents whom appeared scattered on the western edge of central Leicester.

Areas experiencing high levels of smoking prevalence, presented as likely candidates for passive smoking amongst children, tended to exist along the cities western periphery predominantly occupied by the White British populace. Locations of elevated smoking prevalence appeared spatially unrelated to the respiratory hot-spot and the plethora of detrimental socio-environmental factors identified to be of significance through regression modelling in Chapters 4 and 5. In-fact, J00-99 GWR models only identified levels of smoking prevalence to influence 3.82- 4.37% of children's citywide cases. It was only in relation to J20-22 spatial models that passive smoking presents itself as a thorn of concern, for cases outside of the central zone. Yet, it is quite possible that the benefits of avoiding second-hand smoke have been masked, due to the strong dissociations between passive smoking and children of Indian ethnicity, whom are less likely to be admitted to hospital for respiratory symptoms. Once should note that the wombled boundaries identified to represent sufficient levels of rapid change for smoking prevalence, almost uniquely exist towards the western periphery in relation to hot-spots. Likewise, levels of adult obesity thought to be indicative of childhood activity and dietary intake, appear elevated in peripheral areas of limited respiratory concern, predominantly housing 'White British' children. Gi* outputs also confirm the presence of boundaries across inner city locales, inclusive and south of the respiratory focal point of

concern. Yet, these boundaries would appear to closely mimic those defining transitions in ‘White British’ residency levels, which would imply that the beneficial and detrimental impacts of obesity during childhood is purely a ‘White British’ problem.

Table 6.1, collectively summarises the comparative strength of the aforementioned boundaries relevant to each individual variable, in addition to linking their main transitional shifts to a specific classification of homogeneity.

Normalised 0-1	Squared Euclidean BLV (Maximum = 1)			Boundary Associated Getis-Ord Gi* Outputs	
	Min.	Med.	Max.	Primary Fringes	Primary Locales
Children’s J00-99 Admissions	0.06	0.09	0.59	Hot-Spot	Inner-City
Children’s J00-06 Admissions	0.03	0.06	0.73	Hot-Spot	Inner-City
Children’s J20-22 Admissions	0.06	0.10	0.78	Hot-Spot	Inner-City
TPM ₁₀ Emissions	0.04	0.07	0.27	Hot-Spot	Inner-City
Carstairs Index	0.18	0.23	0.48	Mixed	Various
Adult Smoking Prevalence	0.30	0.37	0.65	Hot-Spot	West Periphery
Adult Obesity Prevalence	0.18	0.30	0.57	Cold-Spot	South Central
White Non-British Children	0.39	0.51	1.00	Mixed	Various
Indian Children	0.18	0.26	0.49	Hot-Spot	Eastern
Other South Asian Children	0.08	0.11	0.56	Hot-Spot	Central
Afro-Caribbean Children	0.06	0.11	1.00	Hot-Spot	Inner-City

TABLE 6.1: Descriptive summary of the comparative strength and favoured positioning of social, environmental and health boundaries

6.3. BOUNDARY OVERLAPS OF THE COMPLETE RESPIRATORY SET (ICD10: J00-99)

Overlap statistics were subsequently employed to establish whether the boundaries defined by areal wombling across different variables, occupied similar localities at a level greater than what would occur by chance. The extent and likelihood of boundary overlap was evaluated via the application of four overlap statistics based on the average minimum distance from boundaries in one variable to the nearest boundary in the other variable of interest (Jacquez 1995, Jacquez & Greiling 2003). The overlap statistic O_S measures the frequency of which boundaries within two datasets intersect, and is thus defined as the number (cardinality count) of elements that are located within in both boundary sets G and H. O_G is the mean distance from any location in the boundaries for G to the nearest location in the boundaries for H. O_H is the mean distance from any location in the boundaries for H to the nearest location in the boundaries for G. O_{GH} is the average distance from the location in the G or H boundaries to the nearest location in the other (G or H as appropriate) boundary.

Boundary overlap analysis techniques were subsequently used to determine whether boundaries in children's respiratory hospitalisation incidents (ICD-10: J00-99) existed closer than one would expect to influential socio-environmental regression variables. In effect the boundary overlap analysis aims to determine whether factors significantly affecting health operate intensely across spatial pockets, or smoothly at a global scale unaffected by spatial positioning. Of particular interest are the univariate distributions of TPM₁₀ emissions, 'Afro-Caribbean' children, 'Other South Asian' children and J00-99 respiratory incidents, all of which appear to experience significant spatial gradient shifts almost exclusively across inner city centre locations (Figures 6.1 - 6.3). It should also be noted that boundaries across children of 'Indian' ethnicity also appear to occur just outside of the inner city zone, perhaps acting as a buffer between the 'White British' and other ethnic minority groups. Meanwhile, boundaries of deprivation denoted by the Carstairs Index appear to be dispersed throughout the city, thus suggesting that the variables effects on children's respiratory health potentially has a weaker spatial dependency than other influential factors.

Statistically significant overlap was identified to occur between boundaries in TPM₁₀ emissions and children's respiratory health (Table 6.2), with the two variables sharing an additional 10 Boundary Elements than what would be expected by chance (OS P \uparrow <0.01). The indicated average minimum distance of 283m from a boundary in TPM₁₀ emissions to a children's respiratory health boundary (OG P \downarrow <0.01), was identified to be of a significantly smaller distance than expected under a null hypothesis of no spatial patterning. In addition, the mean distance from locations in either boundary to the nearest location in the opposing boundary was identified to occur across a significantly small distance of 471m (OGH P \downarrow =0.03), thus acknowledging the presence of significant boundary overlap. Conversely the average minimum distance from boundaries of children's respiratory health to boundaries of traffic emissions lacked statistical significance, thus indicating that additional attributes are likely responsible for a proportion of the spatial dissimilarities experienced in children's respiratory health.

The overlap statistics of respiratory health boundaries alongside 'Indian' ethnicity, 'Other South Asian' ethnicity and Carstairs Index deprivation measures, suggest corresponding areas of rapid change across such variables to be of limited significance (Table 6.2). The previously reported importance of deprivation within the citywide prediction of children's respiratory health but limited presence of coinciding localised magnitude shifts, suggest that this influential factor operates relatively smoothly across the city. Meanwhile, whilst 'Indian' and 'Other South Asian' residency exhibits strong signs of spatial dependency, such groups are not purely restricted to inner-city locales experiencing health, social and environmental

burdens. In-fact, several minority boundaries of relevance exist throughout eastern Leicester in accordance to intra-minority relationships and the reappearance of 'White British' residences. In particular, boundaries in 'Indian' residency generally align with those of the 'White British', which collectively run north-to-south, demonstrating the strong residential separation between children of ethnic majority and minority groups. Nevertheless, both 'White British' and 'Indian' residents appear to decline within inner-city locales of concern, suggestive of the appearance of another minority group to whom 'Indian' residents are disassociated with.

Children of 'Afro-Caribbean' ethnicity were the only social group to experience significant overlap with boundaries in respiratory health, which both variables rising across inner-city locales (Table 6.2). These two variables were identified as sharing an additional 11 Boundary Elements than what would be expected by chance ($O_S P < 0.01$). Zones of rapid change in population levels of 'Afro-Caribbean' children were designated to occur at an average minimum distance of 296m from a respiratory health boundary ($O_G P < 0.01$), thus rejecting the null hypothesis of no spatial patterning. Furthermore, mean distances from locations in either boundary to the nearest location in the other boundary were identified to exist across a modest distance of 556m ($O_{GH} P = 0.05$).

Interestingly, the boundary overlap statistics identified smoking prevalence shifts within the adult populace to occur at significantly distant localities for boundaries in children's respiratory health ($O_{GH} P \uparrow = 0.04$). Perhaps a hefty proportion of smokers recognise the detrimental effects of second hand smoking and thus take suitable actions around children, and/or levels of smoking prevalence are under-recorded or overshadowed by other detrimental factors within certain segments of society. Such matters will be discussed in greater detail within the concluding sections of this chapter.

Boundaries Overlapped (G,H) H= J00-99 Admissions Per 1,000 Children	Statistic	Observed (meters)	Expected (meters)	P	P
(G) TPM ₁₀	O _G	283	893 (±285)	1.00	0.00*
	O _H	658	902 (±327)	0.75	0.25
	O _{GH}	471	898 (±255)	0.97	0.03*
	O _S (count)	14	3 (±2)	0.00*	1.00
(G) Carstairs Index of Deprivation	O _G	1100	893 (±255)	0.20	0.80
	O _H	580	703 (±265)	0.64	0.36
	O _{GH}	840	798 (±211)	0.36	0.64
	O _S (count)	5	4 (±2)	0.31	0.83
(G) Smoking Prevalence	O _G	1352	891 (±249)	0.05*	0.95
	O _H	904	690 (±232)	0.17	0.83
	O _{GH}	1132	792 (±189)	0.04*	0.96
	O _S (count)	3	4 (±2)	0.68	0.51
(G) Obesity Prevalence	O _G	885	904 (±274)	0.47	0.53
	O _H	997	716 (±262)	0.13	0.87
	O _{GH}	939	811 (±212)	0.24	0.76
	O _S (count)	7	4 (±2)	0.10	0.94
(G) White British Children	O _G	1136	906 (±255)	0.18	0.82
	O _H	580	664 (±240)	0.60	0.40
	O _{GH}	858	785 (±202)	0.28	0.72
	O _S (count)	4	3 (±2)	0.43	0.75
(G) White Non-British Children	O _G	777	900 (±304)	0.64	0.36
	O _H	869	869 (±314)	0.42	0.58
	O _{GH}	823	884 (±256)	0.55	0.45
	O _S (count)	7	4 (±2)	0.13	0.93
(G) Indian Children	O _G	690	870 (±261)	0.76	0.24
	O _H	512	707 (±241)	0.77	0.23
	O _{GH}	601	788 (±202)	0.83	0.17
	O _S (count)	6	4 (±2)	0.18	0.90
(G) Other South Asian Children	O _G	707	908 (±336)	0.69	0.31
	O _H	877	978 (±338)	0.61	0.39
	O _{GH}	792	943 (±274)	0.68	0.32
	O _S (count)	5	3 (±3)	0.33	0.79
(G) Afro-Caribbean Children	O _G	296	902 (±355)	1.00	0.00*
	O _H	817	42 (±371)	0.71	0.29
	O _{GH}	556	971 (±298)	0.95	0.05*
	O _S (count)	15	4 (±3)	0.01*	1.00

* P 0.05

TABLE 6.2: Univariate boundary overlap analysis with annual average children's admissions of the complete respiratory set (J00-99)

Multivariate boundaries of evenly weighted TPM₁₀ emissions and individual socioeconomic-ethnic factors were subsequently formed, in order to assess the influence of social attributes on the relationship between road-transport emissions and health boundaries (Appendix E2, Table 6.3). Such bivariate BLV's were simply constructed from the summation of Squared Euclidean dissimilarities for the two individual normalised components X1 and X2 across locations i and j (i.e. $[X1_i - X1_j]^2 + [X2_i - X2_j]^2$). Under such a premise the maximum BLV will never exceed the number of variables used within the boundaries construction for variables normalised 0-1.

In general, the addition of TPM₁₀ emission transitions appeared to provide only a limited impact towards BLV consistencies recorded by social influences in their univariate format (Tables 6.1, 6.3). A consequence, primarily attributed to the highly localised nature of TPM₁₀ emissions which may only raises BLV's found around inner-city locales, and secondly due to the somewhat smoothed nature of TPM₁₀ boundaries which may have a reduced influence alongside other spatially pronounced variables. Nevertheless, the combination of TPM₁₀ emissions and 'Afro-Caribbean' residency levels was observed to markedly raise the ethnic group's univariate minimum and maximum BLV's from 0.06 to 0.10, and 1.00 to 1.09 respectively. It would appear that the combination of these two factors has strengthening their classification of the outer extent of the inner-city problem zone, with only a few boundaries placing focus upon the transitional shifts occurring inside (Appendix E2).

2X Normalised 0-1	Squared Euclidean BLV (Maximum = 2)		
	Minimum	Median	Maximum
TPM ₁₀ Emissions & Carstairs Index	0.18	0.24	0.49
TPM ₁₀ Emissions & Smoking Prevalence	0.34	0.40	0.66
TPM ₁₀ Emissions & Obesity Prevalence	0.19	0.37	0.78
TPM ₁₀ Emissions & White British Children	0.24	0.35	0.60
TPM ₁₀ Emissions & White Non-British Children	0.40	0.53	1.00
TPM ₁₀ Emissions & Indian Children	0.20	0.30	0.50
TPM ₁₀ Emissions & Other South Asian Children	0.11	0.15	0.56
TPM ₁₀ Emissions & Afro-Caribbean Children	0.10	0.15	1.09

TABLE 6.3: Descriptive summary of the comparative strength of bivariate boundaries recording the combined spatial dissimilarities of TPM₁₀ emissions and social variables

Levels of obesity prevalence were the only other variable to see drastic changes from its univariate BLV, which when combined with TPM₁₀ emissions caused an increase in maximum BLV from 0.57 to 0.78. However, these changes in the maximum value would appear more coincidental, in that a select few common boundary's exist across inner city locales, with overall boundary patterning unmoved from what was defined by obesity as a singular factor.

One should also note that these common boundaries around the focal LLSOA represent alternate responses between these two factors of interest, which combine to create an extreme BLV that conveys a mixed message; in that low obesity levels unlike TPM_{10} emissions are not thought to be disadvantageous. Such findings would therefore suggest obesity prevalence to be highly detached from TPM_{10} issues.

In terms of visual changes, only recordings of deprivation, 'Other South Asian' and 'Afro-Caribbean' residency levels, experience substantial alterations to aspects of their boundary positioning when combined with TPM_{10} spatial surfaces (Figure 6.2, Appendix E2). Univariate 'Other South Asian' boundaries, previously established that particularly high residency levels were favoured inside of the inner-city respiratory hot-spots exterior transition zone, where moderate-high burdens are experienced. In combining 'Other South Asian' and TPM_{10} gradient surfaces, the resulting boundary outputs would appear to clearly define the southern extent of our previously established respiratory, social and environmental burdens. A similar consolidation was previously observed in regards to 'Afro-Caribbean' residency.

On another note, 'Other South Asian' residency levels were not previously associated to provide sufficiently strong boundaries towards the north of our inner-city focal point, despite decile plots here also indicating moderate-high levels of residency. Yet, when combined with TPM_{10} spatial surfaces, their bivariate boundary outputs may firmly establish the northern cap of Leicester's zone of concern, due primarily to the overwhelming change in TPM_{10} emissions experienced here. It is believed that the northern extent of this respiratory hot-spot is almost entirely caused by elevated TPM_{10} emissions at the plumes point of origin, in that other explanatory variables are unable to clearly define this transitional zone. The magnitude of change is at such a level, that it is consistently adopted into all of the bivariate boundary models, even where social influences are in strong disagreement throughout the city.

As with the previous variables, bivariate boundaries relating to Carstairs index levels of magnitude appeared to generally occupy the same locales to what was recorded for its univariate counterpart boundaries (Figure 6.2, Appendix E2). In-fact all of the broad deprivation boundary structures were maintained except within one area of eastern Leicester, positioned well away from the inner ring-road. The only major change brought about by the combination with TPM_{10} surfaces, consists of the addition of five boundaries around the city centre, in an area where only two deprivation boundaries had previously existed. The average bivariate BLV across these seven inner-city boundaries was recorded at 0.29, a level substantially above the average BLV of 0.18 recorded by univariate Carstairs Index boundaries across the same locales. These boundaries are thought to represent the interior transition

zone of the inner-city respiratory hot-spots, where very elevated respiratory effects are induced through the double burden of deprivation and TPM₁₀ emissions.

Elsewhere in the city boundaries in deprivation appear to have experienced little additional influence from gradients in TPM₁₀, which suggest that such a double burden is only apparent across inner-city locales. For example, the six boundaries within the vicinity of Western Park, representative of some of the highest deprivation gradients, were observed to have average BLV's only change from 0.29 to 0.30 under a bivariate scheme. Yet, in combining TPM₁₀ and deprivation gradient shifts, one may observe that the designated bivariate boundaries adequately capture the majority of inner and outer city respiratory transitions, signalling the importance of both elements. It may be noted that the greatest health risks are to be found where the two factors centrally coincide, high risks occur where TPM₁₀ operates independently on the fringes of the inner-city, and moderate risks occur where deprivation exclusively functions in peripheral locales.

The bivariate overlap analysis statistically identified boundaries of the combined elements of road-transport emissions and 'Afro-Caribbean' ethnicity, to transpire within significantly closer distances to children's respiratory boundaries than taking both components individually (Table 6.4). It was observed that 'Afro-Caribbean' ethnicity reduced the distance of TPM₁₀ emission boundaries to rapid changes in respiratory incidents by 38m ($O_G P < 0.01$). Likewise, TPM₁₀ emissions were observed to reduce the distance to which boundaries in either 'Afro-Caribbean' residency or J00-99 symptoms coincide, by 15m ($O_{GH} P = 0.05$). However it should be noted that boundaries in all J00-99 admissions (O_G) still do not fall within a proximal distance of significance. Although levels of TPM₁₀ emissions and 'Afro-Caribbean' lifestyle choices instigating the bulk of respiratory complaints across inner-city locales, such outcomes would suggest that, other factors have an influential role in determining the cities wider respiratory issues.

Boundaries Overlapped (G,H) H= J00-99 Admissions Per 1,000 Children	Statistic	Observed (meters)	Expected (meters)	P	P
(G) TPM ₁₀ & Carstairs Index	O _G	871	897 (±294)	0.45	0.55
	O _H	334	831 (±296)	0.99	0.01*
	O _{GH}	602	864 (±239)	0.89	0.11
	O _S (count)	9	3 (±2)	0.03*	0.99
(G) TPM ₁₀ & Smoking prevalence	O _G	1334	891 (±281)	0.08	0.92
	O _H	626	778 (±277)	0.69	0.31
	O _{GH}	980	835 (±228)	0.25	0.75
	O _S (count)	4	4 (±2)	0.49	0.67
(G) TPM ₁₀ & Obesity prevalence	O _G	894	879 (±268)	0.43	0.57
	O _H	990	753 (±268)	0.17	0.83
	O _{GH}	942	816 (±221)	0.24	0.76
	O _S (count)	6	4 (±2)	0.19	0.89
(G) TPM ₁₀ & White British Children	O _G	994	888 (±253)	0.28	0.72
	O _H	463	627 (±224)	0.76	0.24
	O _{GH}	729	757 (±190)	0.50	0.50
	O _S (count)	6	3 (±2)	0.17	0.91
(G) TPM ₁₀ & White Non-British Children	O _G	777	886 (±298)	0.61	0.39
	O _H	869	850 (±303)	0.42	0.58
	O _{GH}	823	868 (±245)	0.50	0.50
	O _S (count)	7	3 (±2)	0.11	0.95
(G) TPM ₁₀ & Indian Children	O _G	621	880 (±252)	0.88	0.12
	O _H	355	703 (±257)	0.95	0.05*
	O _{GH}	488	791 (±209)	0.96	0.04*
	O _S (count)	8	4 (±2)	0.05*	0.97
(G) TPM ₁₀ & Other South Asian Children	O _G	478	884 (±328)	0.93	0.07
	O _H	910	936 (±331)	0.45	0.55
	O _{GH}	694	910 (±272)	0.80	0.20
	O _S (count)	9	4 (±3)	0.05*	0.97
(G) TPM ₁₀ & Afro-Caribbean Children	O _G	245	924 (±358)	1.00	0.00*
	O _H	837	1116 (±402)	0.71	0.29
	O _{GH}	541	1020 (±322)	0.96	0.04*
	O _S (count)	14	3 (±3)	0.00*	1.00

* P 0.05

TABLE 6.4: Bivariate boundary overlap analysis of TPM₁₀ emissions and an individual social parameter, with annual average children's admissions of the complete respiratory set (J00-99)

The grouping of TPM₁₀ emissions with Carstairs Index values revealed the factors to not be within significant proximity of respiratory admission boundaries (O_G P>0.05), thus reflecting the smooth distribution of deprivation (Table 6.4). However zones of extreme changes in children's respiratory admissions share a considerable amount of BEs (O_S P =0.03) and arise within close proximity to areas marking substantial disparities of environmental and economic influence (O_H = 334m, P <0.01). This would indicate that these two factors have substantial

explanatory power in defining the locations of children's respiratory health across the city. Significantly similar trends were also noted to exist when unifying the variables of traffic emission and 'Indian' ethnicity for overlapping with respiratory boundaries ($O_H = 355m$, $P = 0.05$). Upon viewing the univariate 'Indian' boundaries (Table 6.2), one may see that this ethnic group itself has no associations with poor respiratory health ($P > 0.1$), as previously confirmed via spatial modelling in Chapters 4 and 5. As formerly detailed, persons of Indian ethnicity tend to reside within areas fringing the inner city, thus marking respiratory risk zones of elevated traffic emission inhabited by other ethnic minority groups. It is believed that 'Afro-Caribbean' residents are the minority group at greatest risk to such burdens, although to a lesser degree some other 'Other South Asian' residents may also be placed at risk; as observed through their bivariate TPM_{10} boundaries sharing a significant proportion of J00-99 boundaries ($O_S P = 0.05$).

In constructing trivariate boundary sets containing readings of TPM_{10} , deprivation and another final social influence, one may observe four every-day socio-environmental exposure candidates that are collectively responsible for the spatial existence of marked boundaries in respiratory health (Appendix E3, Table 6.5). The two-trivariate boundary sets of interest contain 'Afro-Caribbean' or 'Other South Asian' residency levels as their final component, both of which visually resemble the layout of univariate J00-99 health boundaries (Figure 6.1, Appendix E3). One should note that the 'Afro-Caribbean' trivariate boundary would appear to continue to segregate the inner and outer gradient stages of the city centre hot-spot, whereas the 'Other South Asian' trivariate boundary set purely defines the central zones outer limits.

Both of these trivariate sets of interest are identified to share a substantially high number of BE's with J00-99 outcomes ($O_S P = 0.02$), with such health boundaries also appearing to fall within a short distance of these combined socio-environmental influences ($O_H = 343m$, $P = 0.01$). Furthermore, a significantly close O_{GH} score of 528m ($P = 0.03$) for the 'Afro-Caribbean' trivariate set, would suggest that this combination of socio-environmental factors somewhat consistently result produce a detrimental health gradient. In contrast, the 'Other South Asian' trivariate set neither provided a significant O_{GH} or O_G score, which would indicate that health gradients are not universally associated with such socio-environmental influences.

Boundaries Overlapped (G,H) H= J00-99 Admissions Per 1,000 Children	Statistic	Observed (meters)	Expected (meters)	P	P
(G) TPM ₁₀ , Carstairs Index & Smoking Prevalence	O _G	1248	884 (±280)	0.11	0.89
	O _H	562	696 (±239)	0.68	0.32
	O _{GH}	905	789 (±205)	0.23	0.77
	O _S (count)	4	4 (±2)	0.48	0.67
(G) TPM ₁₀ , Carstairs Index & Obesity Prevalence	O _G	965	901 (±275)	0.35	0.65
	O _H	776	699 (±253)	0.34	0.66
	O _{GH}	870	800 (±218)	0.31	0.69
	O _S (count)	7	4 (±2)	0.11	0.95
(G) TPM ₁₀ , Carstairs Index & White British Children	O _G	904	889 (±255)	0.43	0.57
	O _H	434	667 (±229)	0.86	0.14
	O _{GH}	669	778 (±189)	0.69	0.31
	O _S (count)	7	4 (±2)	0.10	0.96
(G) TPM ₁₀ , Carstairs Index & White Non-British Children	O _G	897	900 (±297)	0.45	0.55
	O _H	878	784 (±283)	0.31	0.69
	O _{GH}	887	842 (±239)	0.40	0.60
	O _S (count)	7	4 (±2)	0.12	0.94
(G) TPM ₁₀ , Carstairs Index & Indian Children	O _G	985	874 (±277)	0.31	0.69
	O _H	406	671 (±243)	0.90	0.10
	O _{GH}	696	772 (±208)	0.58	0.42
	O _S (count)	6	4 (±2)	0.21	0.90
(G) TPM ₁₀ , Carstairs Index & Other South Asian Children	O _G	813	892 (±309)	0.52	0.48
	O _H	318	878 (±311)	0.99	0.01*
	O _{GH}	565	885 (±257)	0.92	0.08
	O _S (count)	10	3 (±2)	0.02*	0.99
(G) TPM ₁₀ , Carstairs Index & Afro-Caribbean Children	O _G	714	888 (±303)	0.70	0.30
	O _H	343	947 (±332)	0.99	0.01*
	O _{GH}	528	918 (±255)	0.97	0.03*
	O _S (count)	13	4 (±3)	0.00*	1.00

* P 0.05

TABLE 6.5: Trivariate boundary overlap analysis of TPM₁₀ emissions, Carstairs Index measurements of deprivation and an individual social parameter, with annual average children's admissions of the complete respiratory set (J00-99)

6.4. URTI BOUNDARY OVERLAPS (ICD10: J00-06)

For children's URTI's, statistically significant overlap was once again identified to occur between boundaries in TPM₁₀ emissions (Appendix E4), with the two variables sharing 11 additional Boundary Elements to what would be expected by chance (OS P_↑ <0.01). The indicated average minimum distance of 257m from a boundary in TPM10 emissions to a children's respiratory health boundary (OG P_↓ <0.01), was identified to be of a significantly

smaller distance than expected under a null hypothesis of no spatial patterning. In addition, the mean distance from locations in either boundary to the nearest location in the opposing boundary was identified to occur across a significantly small distance of 315m ($O_{GH} P < 0.01$), thus acknowledging the presence of significant boundary overlap.

One should note that critical thresholds in TPM_{10} emissions show closer spatial ties with URTI's, than what was recorded in relation to children's outcomes of the complete respiratory set (J00-99). This may be observed through the respective reduction in the critical O_G and O_{GH} distances of 26m and 156m. Upon previously establishing the importance role of TPM_{10} emissions on localised dose-response relationships in respiratory health during childhood, it should come of little surprise that its impact has a more immediate influence on the most minor of respiratory complaints. The strength of such associations are firmly cemented through overlap scores which record URT health boundaries to be universally located within close proximity to TPM_{10} emission gradients of interest ($O_H = 372m, P = 0.03$). Such findings were not previously observed for the complete respiratory set, and would suggest that TPM_{10} emissions have an aspect of involvement within all major URT outbreaks across Leicester.

As before, deprivation boundaries on their own were generally shown to have no statistical significance with URTI gradients (Appendix E4), despite prior regression models recognising such exposures as substantially influencing respiratory health throughout Leicester. This lack of association amongst spatial surfaces once again portrays the relatively smooth nature in which deprivation pockets form across the city. Yet, unlike its associations with J00-99 health gradients, deprivation was identified to share 5 more BEs with URTIs than to be expected by chance ($O_S P \uparrow = 0.04$); potentially identifying deprivation as having a more influential role on URT outcomes, when combined with rapid transitions in other socio-environmental exposures. As before, such concepts will be explored via the constructed bivariate and trivariate boundary outputs (Appendix E2, E3). Unlike the univariate J00-99 overlaps, elevated levels of smoking prevalence were not observed to occur in locales distant from URT complaints, instead recording no significantly correlated spatial patterning. A lack of significant spatial patterning was also recorded in relation to univariate boundaries in 'Indian' and 'Other South Asian' residency, in line to prior outputs for the complete respiratory set (Appendix E4).

Interestingly, the boundary overlap statistics identified obesity prevalence shifts within the adult populace to share a significant number of Boundary Elements with children's URT health ($O_{GH} P = 0.05$). Despite this influence also occupying a number of mutual J00-99 boundary locales, their spatial connection was statistically defined to not be of importance.

Placing focus back on the URT subset, these common boundaries around the focal LLSOA and eastern side of the inner-city zone of concern are a source of some confusion, in that low obesity levels are associated with poor URT health. Perhaps these low levels of obesity mark communities whom have an inadequate dietary intake, or more likely is that the actual beneficial influences of a healthy diet are overshadowed by a plethora of arduous socio-environmental burdens.

Unique to the J00-06 subset, is the proximal association with Leicester's 'White Non-British' residents, whom appear to reside within 313m of such health fronts ($O_G P < 0.01$). Furthermore, the mean distance of 373m separating opposing boundary locations, was deemed to be of a significantly smaller distance than expected under a null hypothesis of no spatial patterning ($O_{GH} P \downarrow < 0.01$). Such close ties have likely been influenced through the sharing of respiratory boundary elements solely related to the J00-06 subset, towards the cities western periphery around Western Parks, and on the east-central outskirts of Charnwood (Figures 6.1, 6.2). Whilst both sets of boundaries depict these two specific locales as experiencing mutual gradient transitions from low to high levels, one should recall that Chapter 5 found limited evidence of 'White Non-British' lifestyles influencing URT outcomes. This mixed response becomes apparent on investigating 'White Non-British' boundaries located along the southern fringes of the inner-city, which depict an inverse response with URT outcomes. Therefore, it is most likely that sections of this ethnic group are at a higher risk of exposed to other detrimental socio-environmental influences, rather than the groups lifestyle choices bearing a noteworthy impression on URT health.

As with the complete respiratory set, children of 'Afro-Caribbean' ethnicity were recorded to experience significant overlap with boundaries in URT health (Appendix E4). These two variables were identified as sharing an additional 14 Boundary Elements than what would be expected by chance ($O_S P < 0.01$), a figure 3 elements above what was recorded in the J00-99 boundary analysis. Furthermore, zones of rapid change in population levels of 'Afro-Caribbean' children were designated to occur within 245m from a URTI health boundary ($O_G P \downarrow < 0.01$), with an average distance of 391m also separating opposing boundary locations ($O_{GH} P = 0.01$). Such values reflect a reduction on the complete respiratory sets critical O_G and O_{GH} distances by 51m and 165m, respectively. What's more, a universal proximity of URT health boundaries to this ethnic minority group ($O_H = 391m, P = 0.05$), would suggest that such outcomes are not solely based upon the groups expected, but not exclusive residence, within neighbourhoods facing a heightened risk to detrimental socio-environmental exposures. Previously, the lifestyle choices of 'Afro-Caribbean' residents were believed to moderately influence respiratory health during childhood (Chapters 4 and 5). Therefore, it should come of

little surprise that such lifestyles provide a greater influence on the most minor of respiratory complaints, as presented via a reduction in the group's proximity to URTI health fronts.

As with the complete respiratory set, a bivariate overlap analysis statistically identified collective TPM₁₀ emission and 'Afro-Caribbean' residency boundaries, to transpire within a reduced proximity of children's URT boundaries when compared to their individual contributions (Appendix E5). It was observed that 'Afro-Caribbean' ethnicity reduced the distance of TPM₁₀ emission boundaries to rapid changes in respiratory incidents by 48m (O_G $P < 0.01$). One should also note that bivariate TPM₁₀ emission and 'Afro-Caribbean' boundaries appear to exist 36m closer to URTI health fronts than to those of the complete respiratory set (J00-99). Yet unlike their individualistic boundaries, transitions in URT health are revealed to no longer fall within a proximal distance of significance to areas marking a mutual excess of both TPM₁₀ and 'Afro-Caribbean' levels (O_H). This would suggest that although communities characterised by these two components are positively associated with URT health gradients, such areas only represent a portion of the cities zones of concern. Although it should not be forgotten that such areas are thought to occur alongside health gradients of the highest magnitude.

Once more, trends were also noted to exist when unifying the variables of traffic emission and 'Indian' ethnicity for overlapping with respiratory boundaries ($O_S = 8$, $P = 0.04$). It would appear that while the combination of these socio-environmental variables had limited influence on establishing URT health fronts (O_G , O_{GH} , $P > 0.05$), such influences would appear to broadly mark zones of respiratory risk ($O_H = 304m$, $P = 0.03$). As previously discussed the 'Indian' ethnic itself has no associations with poor respiratory health, yet marks transition zones between 'White British' and ethnic minorities, in addition to certain intra-ethnic minority transitions around central locales. In combining with TPM₁₀ surfaces, structural focus is placed on marking the fringes of these central locales, predominantly housing other minority groups whom experience elevated socio-environmental burdens. Interestingly, bivariate TPM₁₀ and 'Other South Asian' boundaries would appear to share a substantial proportion of boundary elements and fall within significant proximity of selective URT health transitions (O_G , O_{GH} , $P \downarrow < 0.05$). Whereas before, their associations in a bivariate manner with the complete respiratory set were weak, in that only a selective amount of boundary elements were shared. These increased associations are likely representative of the increased structural presence of J00-06 surfaces, which consolidate the southern and uniquely define the eastern face of the centre respiratory hot-spots outer limits, where this group predominantly resides.

It would therefore appear that communities characterised by these two components are through to clearly and positively determine spatial transitions in children's URT health,

however this combination of factors is not the main driving force behind Leicester's URT fronts ($O_H P \downarrow > 0.05$). On a final note relating to the bivariate ethnic minority boundaries, it would appear as if the addition of TPM_{10} emissions hardly affects the univariate elements representative of 'White Non-British' surfaces. Here moderate ethnic elements coincide with dissimilarities along the outer limits of the central TPM_{10} area, whilst westerly boundaries representative of strong residential dissimilarities prevail despite experiencing reduced TPM_{10} levels. Identical findings are recorded to those of the univariate 'White Non-British' surfaces.

Finally, the grouping of TPM_{10} emissions with Carstairs Index values revealed the factors to not be within significant proximity of respiratory admission boundaries ($O_G P > 0.05$), thus reflecting the smooth distribution of deprivation. However zones of extreme changes in children's respiratory admissions share a considerable amount of BEs ($O_S P = 0.03$) and arise within close proximity to areas marking substantial disparities of environmental and economic influence ($O_H = 79m$, $P \downarrow < 0.01$). This would indicate that these two factors have substantial explanatory power in defining the locations of children's respiratory health across the city. One should also note that bivariate TPM_{10} and deprivation boundaries appear to exist a staggering 256m closer to URTI health fronts than to comparable elements of the complete respiratory set (J00-99). This somewhat spatially immediate response would indicate that URTI's are initiated based upon relatively clear thresholds of socio-environmental exposures.

In constructing trivariate boundary sets containing readings of TPM_{10} , deprivation and another final social influence, one may observe that this double burden has interesting interactions with all five of the explored ethnic groups (Appendix E6). Following on from prior knowledge obtained within this project, one may observe that the trivariate boundary sets of 'Indian' and 'White British' residency clear define east-west divisions between ethnic minority and majority groups; in addition to outlying aspects of segregation between other minority groups across inner city locales (Figure 6.3, Appendix E3). Consequently, the addition of these ethnic groups only act to assist in describing the outer limits of this inner-city problem zone, which affects Leicester's most vulnerable minority groups. URT health transitions are indicated to fall within 277m ($O_H P = 0.02$) of trivariate boundaries relating to both 'Indian' and 'White British' residency.

Interestingly, in exploring the three other ethnic minority groups whose lifestyles hypothetically contribute to such burdens (Appendix E6), one may observe that URT health transitions are recorded within a significantly close distance to trivariate boundaries in 'White Non-British' residency ($O_H = 410m$, $P \downarrow = 0.03$). Yet such a separation distance is actually 133m greater than the outer limits of the socio-environmental ethnic problem areas denoted by 'Indian' and 'White British' residents. Although this should be of limited surprise when

considering that 'White Non-British' residency is sporadically focused outside of the key inner-city zone of concern, to which transitions in 'Indian' and 'White British' are thought to define such outer limits. In-fact, 'White Non-British' residency appears only associated with minor transitions in URT health, to which an unclear direction of influence was previously shown by the univariate boundary analysis of this group. Trivariate 'White Non-British' boundaries may therefore only define minor URT health gradients, without offering a descriptive element of such interactions.

The two minority trivariate boundary sets of particular interest contain 'Afro-Caribbean' or 'Other South Asian' residency levels as their final component, both of which to a certain extent visually resemble the citywide layout of univariate URTI boundaries (Figure 6.1, Appendix E3). One should note that 'Afro-Caribbean' trivariate boundary would appear to continue to segregate the inner and outer gradient stages of the city centre hot-spot, whereas the 'Other South Asian' trivariate boundary set purely defines the central zones outer limits. Interestingly the transitions between these two ethnic groups would appear to share associations with the structured gradients of respiratory health, which rapidly decreases with increased centricity. In-fact, spatial transitions in URT health are indicated to respectively fall within 67m and 50m ($O_H P \downarrow < 0.01$) of trivariate boundaries relating to both 'Other South Asian' and 'Afro-Caribbean' residency. In exploring a more citywide stance of these interactions, boundaries in 'Other South Asian' ethnicity appear to sway URT outcomes within a distance of 429m compared to 309m for 'Afro-Caribbean' residents ($O_{GH} P < 0.01$). 'Afro-Caribbean' residents are thus show to experience the brunt of the cities socio-environmental burdens, with 'Other South Asian' residents experiencing moderate burdens and the lifestyle choices of 'Indian' residents appearing to acting to actively inhibit such burdens. Such interactions have previously been touched upon within chapter 4.

6.5. LRTI BOUNDARY OVERLAPS (ICD10: J20-22)

For children's LRTI's, statistically significant overlap was once again identified to occur between boundaries in TPM_{10} emissions (Appendix E7), with the two variables sharing 12 additional Boundary Elements to what would be expected by chance ($O_S P < 0.01$). The indicated average minimum distance of 185m from a boundary in TPM_{10} emissions to a children's respiratory health boundary ($O_G P \downarrow < 0.01$), was identified to be of a significantly smaller distance than expected under a null hypothesis of no spatial patterning. Such a distance reveals that elevated TPM_{10} emission levels stimulate LRT outcomes within a range

72m closer than the pollutants critical response for URT outcomes. These findings would appear to be in-line with conclusions drawn from chapter 5, which state that LRTI's only occur where persistently high exposures exist or short sharp episodes occur; whereas URTI's may operate across moderate and persistent exposures, hence the TPM_{10} emissions smoother associations with URT health outcomes. However, unlike URT outcomes, the average minimum distance of children's LRT health boundaries to those of traffic emissions lacked statistical significance, thus indicating that additional attributes are likely responsible for a proportion of the spatial dissimilarities experienced in children's LRT health. One may note that a similar conclusion was drawn from boundaries relating to the complete respiratory set (J00-99).

Deprivation boundaries on their own were generally shown to have no statistical significance with LRTI gradients, despite exposures influencing such health outcomes throughout Leicester (Appendix E7). Yet, unlike its associations with J00-99 health gradients, deprivation was identified to share 6 more Boundary Elements with LRTIs than to be expected by chance ($O_S P \uparrow = 0.01$). As with the URT subset, this outcome may identify deprivation as having a considerably influential role on LRT outcomes, when combined with rapid transitions in other socio-environmental exposures. A lack of significant spatial patterning was also recorded between LRT and ethnic minority univariate boundaries representative of 'White Non-British', 'Indian' and 'Other South Asian' residency, in line to prior outputs for the complete respiratory set. In addition, the boundary overlap statistics identified smoking prevalence shifts within the adult populace to occur at significantly distant localities for boundaries in children's LRT health ($O_{GH} P \uparrow = 0.02$). As before, perhaps matters of smoking prevalence are under-recorded or overshadowed by other detrimental factors within certain segments of society.

As with the complete respiratory set, children of 'Afro-Caribbean' ethnicity were the only social group to experience significant overlap with boundaries in LRT health (Appendix E7). These two variables were identified as sharing an additional 11 Boundary Elements than what would be expected by chance ($O_S P \uparrow < 0.01$), a figure identical to what was recorded in the J00-99 boundary analysis. Furthermore, zones of rapid change in population levels of 'Afro-Caribbean' children were designated to occur within 214m from a URTI health boundary ($O_G P \downarrow < 0.01$), with an average distance of 464m also separating opposing boundary locations ($O_{GH} P = 0.01$). This reduced proximity of elevated 'Afro-Caribbean' levels to LRT rather than URT outcomes by a distance of 31m ($O_G P < 0.01$), falls in-line with the classification of TPM_{10} exposures, which state that LRTI's only occur where high magnitudes of exposure exist. As portrayed through the short distance of socio and environmental causes and LRT effects compared to the somewhat gradual causal-effect scheme for URT outcomes. Akin to the

complete respiratory set, but unlike the URT subset, health boundaries in children's LRT outcomes universally lacked significant proximity to those of 'Afro-Caribbean' residency; which would suggest that several other attributes of considerable strength command this respiratory subset.

As with the previously explored respiratory conditions, a bivariate overlap analysis statistically identified collective TPM_{10} emission and 'Afro-Caribbean' residency boundaries, to transpire within a reduced proximity of children's LRT boundaries when compared to their individual contributions (Appendix E8). It was observed that 'Afro-Caribbean' ethnicity reduced the distance of TPM_{10} emission boundaries to rapid changes in respiratory incidents by 89m ($O_G P < 0.01$). One should also note that bivariate TPM_{10} emission and 'Afro-Caribbean' boundaries appear to exist 113m closer to LRTI health fronts than to those of the URT set (J00-06), further highlighting the immediate and somewhat spatial more restricted area of influence of agents on LRT conditions. As with their individual boundaries, transitions in LRT health were recorded to still lack substantial associations with areas marking mutual excesses of TPM_{10} and 'Afro-Caribbean' levels (O_H), despite habitually occurring alongside health gradients of the highest magnitude. Likewise, locations describing a sizable shift in the level of 'Other South Asian' residents were observed to share a considerable amount of BEs ($O_S P = 0.01$) and arise within close proximity to children's LRTI health fronts ($O_G = 344m, P < 0.01$).

Meanwhile, the grouping of TPM_{10} emissions with Carstairs Index values revealed the factors to not be within significant proximity of respiratory admission boundaries ($O_G P > 0.05$), children's LRT cases were recorded to share a considerable amount of BEs ($O_S P < 0.01$). Whilst the combined burden of deprivation and TPM_{10} emissions were found to not always manifest reductions in children's LRT health, it would appear that Leicester's existing LRTI boundaries unanimously arise within close proximity to areas exhibiting such characteristics ($O_H = 264m, P = 0.01$). It is believed that this double burden of exposure to environmental pollutants and deprivation is the main driving force behind severely reduced respiratory health within Leicester.

Reasoning for why boundaries of this double burden lacks consistent acquaintances with LRTI structures, are thought lie within the nature of their bivariate boundaries construction; in that suitable elements were able to exist across western Leicester where only disparities in deprivation were high. The definition of these elements has been strongly influenced by the unavoidable use of interpolated TPM_{10} surfaces, which characteristically depict somewhat smoothed dissimilarity values compared to other factors (Table 6.1). Consequently, normalised TPM_{10} emission BLV's have a severely reduced ability to sway the vote in multivariate boundary constructions where extremely sharp transitions in additional

factors occur. Following this concept it is possible to suggest that some of the peripheral boundaries are not 'true' representatives of this double burden, and that in reality spatial shifts relating to levels of both TPM₁₀ and deprivation do comprehensively define respiratory outcomes across Leicester.

In constructing trivariate boundary sets containing readings of TPM₁₀, deprivation and another final social influence, one may observe that this double burden has interesting interactions with only three of the explored ethnic groups (Appendix E9). Unlike URT complaints, only shifts in 'Indian' residency would appear to define the outer limits of the inner-city problem zone relating to LRTI's, which primarily impacts upon Leicester's most vulnerable minority groups. Here LRTI health transitions may be found on the other side of trivariate boundaries relating to 'Indian' residency ($O_S P = 0.05$), and are typically indicated to fall within 359m of such multivariate transitions ($O_H P = 0.05$). The two trivariate boundary sets of particular interest once more involve 'Afro-Caribbean' and 'Other South Asian' residency levels as their final component. As previously observed, the 'Afro-Caribbean' trivariate boundary lies within the inner and outer gradient stages of the city centre hot-spot, whereas the 'Other South Asian' trivariate boundary set purely defines the central zones outer limits. These observations are to a certain extent shown through spatial transitions in LRT health, which respectively fall within 248m and 217m ($O_H P < 0.01$) of trivariate boundaries relating to both 'Other South Asian' and 'Afro-Caribbean' residency. Furthermore, the distance separating either a causal or effect boundary from the nearest opposing boundary, for trivariate 'Other South Asian' and 'Afro-Caribbean' structures, respectively occurred within the significantly proximal distances of 505m and 264m ($O_{GH} P = 0.05$); thus acknowledging the presence of significant and influential boundary overlap.

6.6. SOCIO-ENVIRONMENTAL BOUNDARY OVERLAPS

On a final point of interest, overlap statistics were employed to statistically summarise the spatial interactions and associations between the individual socio-environmental variables (Appendix E10 – E17). As expected elevated levels of 'Afro-Caribbean' residency are to be found within close proximity to TPM₁₀ emissions and vice versa, both of which were recorded in a spatially significant manner (Appendix E10). In-fact out of Leicester's core ethnic groups, 'Afro-Caribbean' residents were observed to reside within the closest proximity to TPM₁₀ boundaries ($O_G = 351m, P = 0.02$). In contrast, 'White British' residents were overall shown to favour residing within peripheral locales away from detrimental TPM₁₀ surfaces ($O_G = 1397m,$

$P = 0.05$). Although there would appear to be cases where a subset of this ethnic group, most likely comprising of the most vulnerable individuals, falls within close proximity to TPM_{10} exposures ($O_H = 384m$, $P > 0.05$). Yet, whilst this distance is similar to that displayed through Afro-Caribbean residency, one should note that such findings are not of statistical significance.

Meanwhile, spatial transitions in 'White Non-British' residency were themselves not universally related to TPM_{10} emissions ($O_G P > 0.05$), yet central pollutant boundaries were observed to show a tendency of existing within close proximity to this particular minority group ($O_H = 258m$, $P < 0.01$). However, where this occurs elevated 'White Non-British' residency levels are found to fall outside of the core TPM_{10} zone of respiratory burdens. For this ethnic group, similar or inconclusive exposures exist involving the collective range of Leicester's socio-environmental influences (Appendix's E10, E11, E12, E14), which when combined with the groups lifestyle choices, produces an indistinguishable direction of respiratory sway. Neither 'Indian' nor 'Other South Asian' residency levels globally shared associations with TPM_{10} surfaces ($O_G, O_H P > 0.05$). This lack of spatial significance is something of a surprise particularly for 'Other South Asian' residency boundaries, which positively combine with the outer-city centre TPM_{10} boundaries in a bivariate fashion to share associations with URT and LRT health fronts. Likewise, surfaces in deprivation were neither found to significantly occur at proximal or distant locations to TPM_{10} boundaries, despite previously signalling that their combination of effects provide a substantial double burden on respiratory health. Although this lack of significance likely relates to the fact that this double burden only influences inner-city communities, whereas issues of deprivation are not spatially restricted and occur throughout the city.

Elevated community levels of smoking prevalence, in-line with earlier findings, are found to occur in distant locations from the high levels of inner-city TPM_{10} emissions ($O_{GH} = 1313m$, $P = 0.02$), which are one of the main driving forces behind the prescribed respiratory hot-spot. It is therefore most likely that the minor to moderate health burdens of smoking have been overshadowed, rather than non-existent, as local regression analysis reveals smoking as an influential factor behind respiratory health outside of this zone (Chapter 5). In contrast, obesity prevalence boundaries overall appear unrelated to TPM_{10} emissions ($O_G, O_{GH} P > 0.05$), although central TPM_{10} boundaries would appear to have some associations ($O_S P = 0.02$). Such observations have previously been discussed, in that both factors were shown to present alternative surface responses, and therefore do not cooperate to diminish a child's respiratory health.

Upon exploring the interactions of social variables with levels of deprivation, one may observe that elevated levels of smoking prevalence spatially coincide with unfavourable socio-

economic situations particularly around western peripheral locales, which house 'White British' residents (Appendix's E11, E12). Yet the proximity of smoking prevalence to deprivation, asks questions regarding the under-reporting of such health lifestyle influences amongst certain minority groups; as to accurately report such lifestyles one would have to initially access the relevant public services, and secondly feel comfortable in discussing such matters. In following this concept, it is likely for measurements of deprivation to capture general attitudes towards smoking, and therefore inadvertently act as a proxy for the effects of passive smoking. Lakshman et al (2010) have previously reported similar observations in their assessment of the 2008 East of England Lifestyle Survey. Following on from this line of investigation, Appendix E12 indicates transitions in smoking prevalence to be comprehensively distant from 'Afro-Caribbean' and 'Other South Asian' communities ($O_H P = 0.05$). Yet such observations are in conflict to findings from the Health Surveys for England 2006-08, which report smoking uptake to be high amongst these two specific ethnic groups (Better Health 2011). Such factors are to be explored within greater detail throughout the conclusion section of this chapter.

One should note that while issues surrounding deprivation (Figure 6.2) affect many 'White British' communities, such boundaries have a tendency to fall away from the fringes of these spatial blocks (Appendix E11), which would suggest that the general lifestyle choices of the ethnic majority are not the main cause behind socio-economic transitions. Interestingly, boundaries in deprivation were typically observed to occur within distant locations from transitions in 'Afro-Caribbean' and 'Other South Asian' residency levels ($O_H P < 0.05$), while no significant global associations in spatial surfaces were observed in the opposite direction. Yet, cluster analysis has consistently identified both of these ethnic minority groups to reside within deprived neighbourhoods (Figure 6.4). Again such observations would suggest that detrimental socio-environmental influences are typically detached across Leicester as a whole, yet when such influences do combine then serious respiratory impacts have been felt.

Overlap analysis confirms the previously discussed strong segregation that occurs between ethnic majority and minority groups, within an earlier section of this chapter. In particular, the expanse of north-to-south running boundaries in 'Indian' and 'White British' residency are shown to occur within proximal distance, reflective of these short sharp ethnic transitions (Appendix E14). From here, communities comprised of predominantly of 'Indian' residents are found to share central and eastern wards with other minorities, as indicated by the earlier hot-spot analysis outputs (Figure 6.3) and close boundary proximity (Appendix E16). Nevertheless, intra-minority divisions would also appear to exist, as indicated by the reverse residential gradients of centrally focused 'Afro-Caribbean' and 'Indian' residents. Meanwhile

transitions in 'Other South Asian' residency levels tend to occur within 'Indian' strongholds, representative of a far greater level of residential coalescence. In-fact, the societal integration between ethnic minorities appears to represent how individual group's lifestyles impact upon respiratory health; with isolated 'Afro-Caribbean' communities experiencing and perhaps contribute to a wealth of burdens whereas 'Indian' community's lifestyles actively thwart respiratory issues.

6.7. CONCLUSIONS

In this chapter, I set out to spatially analyse intra-urban patterns of environmental and social risk, through applying techniques conventionally applied within the fields of genetics and environmental conservation to define how objects naturally shift in magnitude across spatial fields. Traditionally, the distance-threshold impact of pollutants emitted from mobile sources on respiratory health are explored through comparing measurements of health amongst external and internal inhabitants of arbitrarily defined buffer zones placed upon locations predetermined in response to their social or environmental attributes. Boundary analysis techniques remove the selection bias of traditional applied proximity methods, through the exploration of pollutant and health gradient shifts across an entire cityscape in the construction of a distance-threshold relationship. Furthermore, such tools uniquely provide a means to quantify the extent to which certain social groups modify such thresholds, an issue previously left unexplored through their treatment of confounding factors. However the technique should not be viewed as a direct replacement of subject-level proximity based enquiries, but rather means of identifying priority areas for intervention that require exploration at a more individualistic level.

Cluster analysis techniques validating constructed boundary sets, identified a disproportionate distribution of children's respiratory hospital admissions across Leicester, of which inner city children experiencing considerably greater respiratory health burdens. Noticeable hot-spots of residentially experienced road-transport emissions, deprivation and certain ethnic groups appeared to also coexist across such inner city localities in general agreement with multilevel modelling. Such findings appear to be in agreement with Pearce et al.'s (2010) 'triple jeopardy' of social, health and environmental inequalities. Naturally occurring boundaries of TPM_{10} emissions marking significant changes in magnitudes of environmental pollutant levels, were identified to occur within 283m of children's J00-99 hospital admission boundaries across Leicester UA ($O_G P \downarrow < 0.01$). Substantial shifts in the

percentage of 'Afro-Caribbean' children at the LLSOA were also identified to exist within close proximity to boundaries in children's overall respiratory health ($O_G=296m$, $P <0.01$). Meanwhile, the overlap statistics of J00-99 respiratory health boundaries alongside 'Indian' ethnicity, 'Other South Asian' ethnicity and Carstairs Index deprivation measures, seem to suggest corresponding areas of rapid change across such variables to be of limited significance. However in evaluating spatially aggregated health and socio-environmental exposure datasets, lies the potential for ecological fallacy, in assuming that associations observed at the community level universally hold for individuals residing within such areas. Whilst ecological studies cannot be used as substitutes for individual correlation studies, this does not indicate that ecological studies are etiologically useless, rather ecological variables are to be viewed as a necessity in the examination of structural, contextual, and sociological influences of disease development (Schwartz 1994). Therefore, these results may only indicate whether boundaries formed from TPM_{10} and social structures modify distance to health boundaries compared to those of TPM_{10} boundaries alone. Yet, in establishing critical distance-response thresholds, our research provides an intuitive foundation for future investigations on an individualistic basis.

A recent meta-analysis of 33 peer-reviewed studies identified average distance decay values for mobile sources to generally exist in the order of 100-400m for elemental carbon or particulate matter mass concentration, 200-500m for NO_2 , and 100-300m for ultrafine particle counts (Zhou et al 2007). Epidemiological investigations of proximity to major road links, defined by arbitrary distance buffers, would appear to agree with the suggested distance threshold of 283m separating elevated children's respiratory admissions in relation to naturally occurring boundaries in traffic emissions. Furthermore, a study appraising the impact of truck traffic pollutants on the lung functionality of 1,092 Dutch Children living along motorways, reported Forced Vital Capacity (FVC) and Peak Expiratory Flow (PEF) 300m from motorways to diminish by -3.6% and -7.7% respectively, compared to reductions of only -2.0% and -1.7% at 1000m (Brunekreef et al 1997). Residential exposure to traffic and 8-year lung development across 3,677 Californian children has also identified substantial variations in relation to freeway proximity, with resident's 500m and 1000-1500m experiencing declines in FVC of -63ml and -19ml respectively (Gauderman et al 2007). Meanwhile an asthma survey of 1,080 Californian children residing at distances below 300m of major freeways identified odds ratios of 1.25 at a distance buffer of >150m and 300m, increasing to 3.80 at distances 75m (Kim et al 2008). This would indicate that although traffic pollutants may operate across wider geographical areas, significantly detrimental health effects occur within relatively close distance to emission sources as defined by the threshold of this study.

Substantial shift in magnitudes of 'Afro-Caribbean' children were identified to reduce the distance between TPM_{10} boundaries and respiratory incidents by 38m ($P < 0.01$), thus implying that these two independent variables operate across spatially similar neighbourhoods. Whilst bivariate boundaries of TPM_{10} emissions and 'Afro-Caribbean' lifestyle choices share links with a majority of inner-city respiratory hospitalisations, J00-99 health fronts were not uniformly located within proximal distances to such features ($O_H P > 0.01$). This would indicate that additional influences have a role in determining the cities wider respiratory issues. In combining surfaces of deprivation with TPM_{10} emissions, one would appear to be able to adequately capture J00-99 health fronts caused by such a double burden, in addition to minor peripheral health fronts triggered solely by issues involving community deprivation levels ($O_H=334m, P = 0.01$). Yet bivariate boundaries in deprivation and TPM_{10} were found to not universally trigger a J00-99 health front ($O_G, O_{GH} P > 0.05$), a phenomenon possibly introduced by an over-smoothed TPM_{10} dataset being unable to restrict deprivation driven boundary placements.

In constructing trivariate boundary sets containing readings of TPM_{10} , deprivation and either 'Afro-Caribbean' or 'Other South Asian' residency levels as their final component, a close visual resemblance of the J00-99 health boundaries was obtained ($O_H = 343m, O_S = 10 BEs$). One should note that 'Afro-Caribbean' trivariate boundary would appear to continue to segregate the inner and outer gradients of the city centre respiratory hot-spot, whereas the 'Other South Asian' trivariate boundary set purely defines the zones outer limits. Furthermore, a significantly close O_{GH} score of 528m ($P = 0.03$) unique to the 'Afro-Caribbean' trivariate set, would suggest that this combination of socio-environmental factors somewhat consistently result produce a detrimental health gradient. It would therefore appear that Pearce et al.'s (2010) 'triple jeopardy' of social, health and environmental inequalities, operates across Leicester in a distinct and spatially measurable manner.

A substantial proportion of Leicester's ethnic minority groups have traditionally occupied low skilled manual labour jobs, with an influx of migrants from the Afro-Caribbean and Asian colonies originally occurring during Britain's post-war reconstruction. However, the collapse of Leicester's manufacturing industries in the 1970's and 1980's would have significantly affected these migrant communities, potentially explaining their tendency to reside within deprived areas. Sizeable Afro-Caribbean migration has also occurred within the last decade through the movement of Somalis from the Netherlands, as low skilled economic migrants. The economically disadvantaged and recently arrived ethnic groups are thought to characteristically possess a limited knowledge of and access to public services, which would be of importance in explaining adverse health outcomes.

Such conclusions have previously been observed within a UK study of 5,494 primary school children, which identified children of Afro-Caribbean and South Asian subcontinent origins as less likely to receive asthma treatments in the form of β_2 agonists (Duran-Tauleria et al 1996). In an examination of health care access across 6,648 English children and young adults aged 2-20, Saxena et al (2002) identified that certain ethnic minority groups appeared able to access primary care, but rarely receive secondary care which could aid manage severe health conditions. For instance, children of Indian ethnicity scored respective AOR's for hospital and GP visitations of 0.72 and 1.86, whereas Afro-Caribbean children produced corresponding values of 1.19 and 1.09 (Saxena et al 2002). This would concur that a certain element of confusion likely exists with regards to accessing the early tiers of the health care system amongst certain individuals.

Collectively localised regression, hot-spot and boundary overlap statistics predominantly identified smoking prevalence within the adult populace to occur across significantly distant localities from areas experiencing high children's respiratory health issues. Perhaps a hefty proportion of smokers recognise the detrimental effects of second hand smoking and thus take suitable actions around children. Alternatively, the impacts of smoking may be clouded by stronger influences on health in the form of deprivation and ethnicity, which are often linked to smoking rates. An analysis of 25,739 adults aged over 16 years who participated in the 2008 East of England Lifestyle Survey, identified neighbourhoods with the highest rates of deprivation to experience respectively low Odds Ratios (OR) for non-smoking (0.45) and 5+days/week of fruit and vegetable consumption (0.70) (Lakshman et al 2010).

Interestingly, a recent report by the Equality and Human Rights Commission (EHRC) identifies poverty, defined as persons earning 60% beneath the median income after accounting for housing costs, to be higher among minority ethnic groups in the UK during 2006-08. In-fact only 19% of White and 26% of Indian ethnicities were classified to be disadvantaged, whereas levels of deprivation appeared substantial within Black (37%) and Pakistani/Bangladeshi (56%) communities (EHRC 2011). Furthermore age-standardised assessments of men's smoking prevalence from the Health Surveys for England 2006-08, identify particularly high rates amongst Black Caribbean's (37%) and Bangladeshi's (36%), moderate rates across White English (27%) and Pakistani's (25%), and low rates for persons of Indian (15%) ethnicity (Better Health 2011). This would imply that a group's socio-economic position tends to act as a determinant of smoking prevalence, particularly for groups whom feel unable to discuss such issues in confidence. Relating to this piece of research it was observed that smoking prevalence cold-spots typically occurred within areas containing an elevated proportion of Indian residents, whom are associated with experiencing fewer

respiratory hospital incidents. However, the seemingly crude spatial variations of modelled obesity and smoking prevalence sets, which exist in blocks of comparable rates, would allude to the presence of ecological fallacy. Therefore, caution is advised in extrapolating information on these variables to an individual level.

One should note that subtle changes to the distance-response measures were observed between the URT and LRT subsets, when compared to those of the overall respiratory system. For instance, critical thresholds in TPM_{10} emissions displayed closer spatial ties with URTI's than outcomes of the complete respiratory set, as observed through the respective reduction in the critical O_G and O_{GH} distances by 26m and 156m ($P < 0.01$). Likewise, boundaries in 'Afro-Caribbean' residency provided corresponding reductions on the complete respiratory sets critical O_G and O_{GH} distances of 51m and 165m ($P = 0.01$) for URT health fronts. Upon establishing their significant interaction across a collective spectrum of respiratory illnesses, it should come of little surprise that such socio-environmental factors deliver a greater influence on the most minor of respiratory complaints, as presented via a reduction in the group's proximity to URTI health fronts.

Like all epidemiological studies exploring the impacts of air pollutants, this work has limitations, specifically involving exposure assessments assumed to be constant across each census units. However, it is reasonable to claim that children spend a larger proportion of time around home or attending educational facilities within close proximity, thus lending credibility to the application of residential emission levels as an exposure proxy. Secondly, estimates of annual vehicle emissions were obtained through vehicle flow models, which although vary spatially, do not account for the actual dispersion of vehicle pollutants. Nonetheless, the application of such datasets is superior to utilising distance from road links as a measurement of exposure. It should also be remembered that studies based around monitored pollutant concentrations and dispersion modelling often assume the overall contribution from traffic sources, whereas the application of emission datasets allows for the direct assessment of the road transport component.

Thirdly, within boundary analysis it is widely recognised that selecting a boundary threshold value (BLV) is subjective in nature. Nevertheless, through stringently classifying boundaries as the top 5% of BLVs, it was anticipated that any boundary preconceptions were minimal. Furthermore, the utilisation of naturally occurring boundaries is thought to offer a more realistic approach for determining the spatial impacts of motor-vehicle pollutants than traditionally applied artificial proximity buffers. This is of importance when considering the complexity of a cityscapes transport network, which likely contains several minor roads close to residential districts experiencing higher pollutant levels through congestion, than what

would be experienced at a predetermined major road. What's more, boundary analysis techniques can include supplementary social factors, thus exploring their influence within the modification of critical distance thresholds of pollutants on health outcomes.

In conclusion, this chapter has shown naturally occurring boundaries in road-transport emissions to result in elevated children's respiratory admissions within a distance of 283m ($P < 0.05$). These findings appear to be in accordance with peer-reviewed studies of average distance decay values for mobile sources at major freeways (Zhou et al 2007), but are noticeably greater than the US Environmental Protection Agency (EPA) buffer analysis threshold of 150m used by Maantay (2007). Additional boundary overlap analysis, identified the designated emission-health threshold to reduce in relation to certain ethnic groups, thus suggesting environmental injustices likely prevail within the model British multicultural City of Leicester. It is believed that this research presents the first study to define local social and environmental critical distance thresholds for factors effecting children's respiratory health. Furthermore, this research presents such findings within the context of a European urban environment, whereas distance-decay and buffer analysis research in the past has tended to be conducted within an American setting. The chapter's findings are considered to have promising applications within healthcare management for locating vulnerable populaces and for minimising health risks in future road network designs.

LOCALISED POLLUTER PAYS PRINCIPLES (PPP)

OVERVIEW

Spatial modelling, object identification and gradient association techniques have confirmed the existence of underlying structural patterning, whereby persons of minority and or lower socioeconomic status habitually reside within intra-urban areas experiencing elevated environmental burdens. In particular, a seemingly unjust 'double-burden' of deprivation and air pollutant exposure was identified as a key explanatory factor across a range of unfavourable respiratory outcomes. At its core 'Environmental Justice' seeks to provide equal access to a clean environment and equal protection from possible environmental harm irrespective of socioeconomic factors (Cutter 1995), both of which appear somewhat lacking across this multicultural British urban environment.

Thus, it is the intention of this chapter to examine the extent of such injustices in further detail, through a largely unexplored line of research, in which levels of environmental accountability are gauged to assess the extent to which one pays for ones actions (Polluter-Pays Principles). Whilst traditionally applied to international cases, this chapter intends to develop upon a localised implementation of such principles (Mitchell & Dorling 2003) across a collection of spatially detailed intra-urban communities, within the context of social, environmental and health outcomes. Upon conducting such procedures, this body of research as a whole, can confidently say that it has geographically located and measured (in a numerical and proximal form) those vulnerable intra-urban populations, whilst distinctively holding select communities to account in an environmental context. To conclude, a few partial solutions are offered to these prescribed problems of the post-industrial cityscape. In particular, this chapter covers objectives 7, 8 and 9 of this project outlined in Chapter 1.

7.1. INTRODUCTION

Contemporary research into the field of 'Environmental Justice' by scholars and policymakers has highlighted underlying structural patterning of society's vulnerable groups, whereby persons of minority and or lower socioeconomic status habitually reside within areas experiencing elevated environmental burdens. In particular, the seemingly unjust 'double-burden' of deprivation and air pollutant exposure is consistently identified as a key explanatory factor in defining health disparities throughout urban environments of the developed world (Crouse et al 2009, Kingham et al 2007, Naess et al 2007). Cutter (1995) defines 'Environmental Justice' (EJ) as a principle which guarantees equal access to a clean environment and equal protection from possible environmental harm irrespective of socioeconomic factors such as race, income and or class. Of significant importance is the fact that such equality measures embody mechanisms for assigning culpability, therefore shifting the burden of proof of contamination to the polluters not resident, a term coined as 'The Polluter-Pays Principle' (PPP). "Thus EJ research seeks to determine whether marginal and/or minority groups bear a disproportionate burden of environmental problems, and whether planning policy and practice affecting the environment are equitable and fair" (Mitchell & Dorling 2003, p909).

Under the OECD council's preliminary 1972 and ensuing 1974 recommendations, "the Polluter-Pays Principle means that the polluter should bear the costs of pollution prevention and control measures, the latter being measures decided by public authorities to ensure that the environment is in an acceptable state" (OECD 1992, p5). Yet, fundamentally the Polluter-Pays Principle is not a concept of equity, but rather a measure for ensuring economic efficiency and minimising distortions in international trade, by incorporating environmental costs in the decision-making process; thus optimising the use of natural resources and ending the cost-free use of the environment as a receptacle for pollution (Vicha 2011). At an international level, such concepts exist in the trading of greenhouse gas emission allowances, in that pollution costs are internalised (efficiency), but also that producers buy their allowances before they pass on those costs to consumers (equity) (Woerdman et al 2007).

Although the principle's precise legal definition for the purposes of practical application remains largely elusive, ironically the Polluter-Pays Principle often exists in practice without necessarily existing in theory, with enforcement agencies on a practical level simply applying the specificity of rules to a particular legal problem (Mann 2009). In this regard, corporate accountability in international environmental law has been traditionally dealt with, whether with a conscious nod to the Polluter-Pays Principle or otherwise, from taxation charges on toxic substances and dangerous goods, or more commonly through imposing

compensation to the victims of environmental harm typically from developing nations (Luken 2009, Luppi et al 2012).

Yet, a largely unexplored tangent of EJ literature is the focus towards whether a 'Localised' Polluter-Pays Principle (PPP) exists, whereby the community responsible for producing pollutants experiences proportional environmental and social burdens. This is somewhat of a surprise, when considering that personal mobile rather than corporate owned point sources account for a substantial proportion of detrimental pollutants found within the Post-industrial cityscape. To-date the focus of EJ research has commonly focused purely upon describing or quantifying how the socio-physical structures of the urban environment shapes health, with limited attention paid towards the origins, of the albeit complex environmental contributions imposed by personal sources. Recently, either a conscious nod to the Polluter-Pays Principle or simply a fortuitous offshoot from other policy, have brought aspects of local environmental responsibility to the average citizen, in the form of urban zoning charges.

In 2003, the London Congestion Charging Scheme (LCCS) became operational, in an attempt to alleviate traffic congestion throughout the cities central districts. Modelled repercussions, identified wards located within the congestion charging zone to experience a 1.3% reduction in NO₂ concentrations, amassing to 183 Years-of-life-gained per 100,000 persons over a 10-year period (YLG₁₀), compared to only 18 YLG₁₀ among the remaining wards (Tonne et al 2008). Whilst, the most deprived quintile recorded the greatest benefit, experiencing 60 YLG₁₀ through a 0.5% reduction on Pre-LCCS NO₂ concentrations of 46.77µg/m³, such outcomes were found to insufficiently reduce the socioeconomic inequalities of air pollution (Tonne et al 2008). In-fact, Pre-LCCS concentrations across affluent wards were already of a substantially lower magnitude (38.15µg/m³), prior to experiencing a further 0.05% reduction from the LCCS (Tonne et al 2008).

In contrast, Cesaroni et al's (2012) evaluation of two low-emission zones established in Rome across the period of 2001-2005, revealed well-off residents as experiencing the greatest level of health gains from zoning implementation. Here, residential reductions in NO₂ concentrations were observed to provide 687 YLG₁₀ for communities of high socioeconomic position, compared to benefits of only 163 YLG₁₀ experienced by residents of the most deprived quintile (Cesaroni et al 2012). Whilst it should be the attention of policy workers to minimise pre-existing societal gradients, here the Rome LEZ fails potentially introduces new issues, one should not rule out the ever so unrealistic scenario that perhaps the poor emit the most and thus the polluter is paying. If this is the case then perhaps an ethical approach beyond that of the Polluter-Pays Principle is required. Yet both zoning studies are unable to quell such concepts, in that they fail to provide information regarding the origin of residentially

experienced road-transport pollutants. Therefore, under an ideal scenario I recommend that focus should be placed on locating and defining communities of interest (in terms of pollutant creation and exposures), in order to improve the ethical efficiency of future traffic management schemes.

Mitchell & Dorling (2003), in an environmental justice analysis of British air quality across 10,444 electoral districts, uniquely explored the role of locally generated vehicle emissions in the air-quality poverty relationship to assist in the further understanding of such principles. Levels of NO_x contributed by each community were derived from 'static' modelling techniques combining, 1991 UK Census recordings of car ownership categorised into vehicle type by local DVLA fleet overviews, with emission factors and typical travel distances respective of vehicle age obtained from European Commission documents. While following a comprehensive method for calculating community contributions, such models are still restricted by their 'static' nature, in that they fail to account for actual population movements in favour of a uniform vehicle-age distance function. Still, the study revealed that although people residing within the areas of poorest air quality contributed the most emissions per car, a clear pattern emerged in which wards emitting the least NO_x, experience the greatest NO₂ concentrations and experienced higher levels of deprivation. These findings would suggest that strong socio-environmental inequalities prevail throughout modern Britain, igniting the previously highlighted need for ethical groundwork prior to the implementing traffic management schemes of the future. Whilst Mitchell & Dorling (2003) establish this tangent of EJ research, further research is required, as localised PPP issues have yet to be explored within the context of health outcomes, or across smaller intra-urban communities, which have highly variable demographics.

Therefore the primary intention of this chapter is to develop upon Mitchell & Dorling's (2003) concept of local PPP's, through exploring the interaction between intra-urban community generated vehicle emissions on residentially experienced levels of transport pollution, deprivation and respiratory health. The methodological enhancements are described within chapter 3, which relate to the incorporation of actual intra-urban workforce travel patterns, rather than assigning each community with a universal travel function. Additional study improvements are to be found through assessing local PPP's at a higher resolution census unit, and through incorporating health outcomes into the relationship.

7.2. CREATION-EXPOSURE RELATIONSHIP OF PERSONAL TRANSPORT SOURCES

Annual LLSOA estimates of road-transport PM₁₀ (TPM₁₀) emissions created from individual communities were derived through combining personal vehicle fleet composition counts with daily workforce trips, which were assumed to represent the significant proportion of population movements. This was achieved through the use of datasets and procedures of calculation outlined in Chapter 3. Cartographic plots of the total TPM₁₀ emissions created from personal transport modes, by the workforce within each local community across Leicester, are displayed in Figure 7.1.

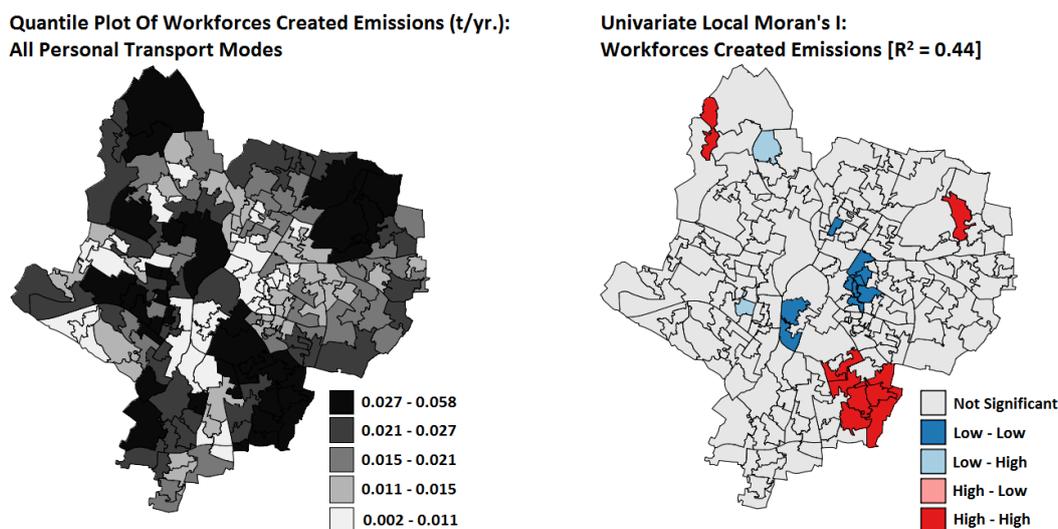


FIGURE 7.1: Cartographic plots of the total TPM₁₀ emissions created from personal transport modes, by local community workforces within Leicester UA

With the assistance of the Local Moran's I statistical test, one may notice that a moderately positive level of spatial structuring exists across Leicester ($R^2 = 0.44$) with respect to community emission contributions. In particular the 11 LLSOAs contained within the south-eastern ward of Knighton were found to create the greatest quantities of TPM₁₀ emissions from personal transport modes (0.022 - 0.046 t/yr.), yet experienced low annual average residential TPM₁₀ emissions of 0.92t/yr. In addition, children from such communities were largely of 'White British' ethnicity (61.54%) and from relatively affluent families (Rank of -4.70), whom typically experience reduced J00-99 admission rates (29.96 per 1,000). In contrast, residents from the 13 LLSOAs contained within the east-central ward of Spinney Hills were on average attributed with only creating 0.010t/yr. whilst experiencing TPM₁₀ emissions of 1.53t/yr. Meanwhile deprivation levels appeared raised (Rank of 4.20), proportions of 'White British' children fell (10.37%), and moderate J00-99 admission rates prevail (46.02 per 1,000). The cities greatest polluters appear to reside predominantly around the ward of Knighton and several other

satellite communities located around the cities periphery. However, those creating the least emissions reside in central locations, which experience the greatest socio-environmental burdens (Chapters 4 and 5). Although one may note that inhabitants of select inner-city locales contribute moderate levels of personal transport emissions, and in a certain respect pay for their consequences. Even so, such contributions are substantially outweighed by those made from external communities, whom do not appear to pay socially, environmentally or physically. The cities present traffic management strategy would therefore appear to be operating in an ethically unjust manner.

Bivariate Local Moran's I statistics were subsequently conducted, as to define the spatial relationships between community created transport emissions and surrounding respiratory health or social-environmental influences, recorded at first-order locations (Figure 7.2, Table 7.1). Upon exploring the connections between respiratory health and pollutant contributions from personal modes of transportation, one may once more view inner-city locales with interest. In-fact it would appear that the most central communities environmentally contribute towards their decline in respiratory health, whereas their adjacent communities emit relatively low levels yet are highly burdened by the contributions of others. For instance, the focal point of prior interest experiences the cities highest annual rates of children's J00-99 admissions (147.83 per 1,000), with its inhabitants contributing 0.022t/yr. in personal TPM₁₀ emissions; a figure ranking this community in the upper 35th percentile of Leicester's polluters. Meanwhile, its 3 north-eastern surrounding LLSOAs continue to exhibit high J00-99 admissions rates (77.18 per 1,000), yet they universally fall within the lowest 15th percentile of polluters, on average creating only 0.007t/yr. in personal TPM₁₀ emissions. Similarly the focal points 2 south-western surrounding LLSOAs experience moderate-high J00-99 admissions rates (58.77 per 1,000), whilst being amongst the lower 5th percentile of polluters in personal transportation terms (≤ 0.004 t/yr.).

Whilst the south-eastern peripheral communities were shown to be the primary contributors of personal TPM₁₀ emissions (Figure 7.1), their creation-respiratory relationship is less apparent, with minor interactions possibly caused by the passage of vehicles away from their places of residence. As previously indicated, by children from these affluent areas experiencing low-moderate levels of respiratory hospital admissions attributed to TPM₁₀ emissions (Figure 4.9) offset by other more favourable attributes. Nevertheless, a singular Knighton LLSOA community was identified as a high polluting-low respiratory outlier by the Bivariate Moran's I analysis, emitting 0.040t/yr. of personal TPM₁₀ emissions whilst experiencing a J00-99 admission rate of only 29.24 per 1,000 children; a value noticeably below the expected LLSOA rate of 39.43. In a minor deviation, the spatial significance of these

communities within Knighton and its adjacent peripheral locales appears somewhat expanded upon for LRTI incidence rates (J20-22). In terms of residents emitting low levels of TPM₁₀ emissions and reaping the respiratory benefits, one should view the 7 LLSOAs between the wards of Belgrave and Latimer, situated just off one of the cities arterial roads. Here such communities typically emit only 0.013t/yr. of TPM₁₀ and experience J00-99 rates (26.70 per 1,000) below the citywide average.

If one may refer back to the cluster detection analysis presented in chapter 4 (Figure 4.2), they should recall the existence of two High-Low J00-99 admission outliers, which have interestingly reappeared within this section of the analysis. Here, the northern outlier identifies the existence of a major road junction, whereby a primary radial corridor containing multiple restaurants and retail outlets (A47: Melton Road) intersects the cities outer ring-road. Subsequently this LLSOA was thought to represent a bottleneck in the flow, which only becomes noticeable during peak hours, and thus appears to have gone undetected by the 1x1km resolution NAEI model. This reasoning explains why children's respiratory health seems unusually impeded (55.37 per 1,000), when compared to average incident levels recorded by first order neighbours (24.25 per 1,000). Although Leicester's Local Transport Plan recognises this corridor for 2011-26 as a key problem area, frequently experiencing delays via buses and general traffic, one should also note that personally created TPM₁₀ emissions for this particular community (0.026t/yr.) are slightly above those of its immediate neighbours (0.020t/yr.). It would therefore appear that the direct actions of this community has a role to play within the extent to which such environmental burdens are felt, further exacerbating existing problems within the cities transport network.

In direct contrast, the previously exposed eastern High-Low J00-99 outlier marking the terminal junction of the outer ring-road (Figure 4.2), would appear to follow trend with its neighbours and emit low levels of personally created TPM₁₀ emissions. In-fact, at this easterly outlying respiratory location, personally created transport emissions (0.017t/yr.) are somewhat lower to those recorded by its first order neighbours (0.021t/yr.). Yet, the eastern target area experiences J00-99 admission rates of 46.12, whilst surrounding locales only record on average incident levels of 24.25 per 1,000 children. Further highlighting the unjust health response felt by this community, albeit at a substantially lower level to what is experienced by the previously discussed inner-city communities. Nonetheless, one might conclude that that the effects of this second bottleneck are therefore unfairly placed upon this community, though they could potentially be reduced through completion of the missing link of the outer ring-road. In addition to increasing the flow here, traffic would also be discouraged from enter

the inner city, potentially mitigating the environmental burdens felt by those most vulnerable communities.

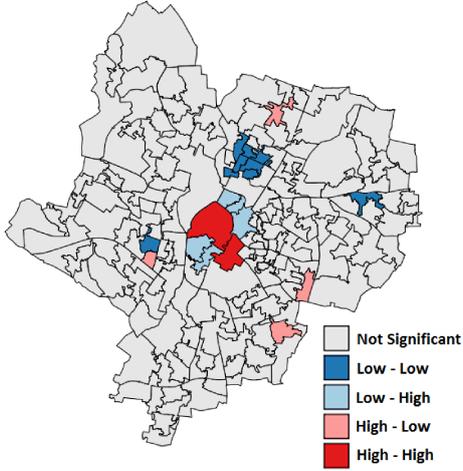
A second Bivariate Local Moran's I statistical test was then applied to compare workforce created TPM₁₀ emissions to neighbouring residentially experienced TPM₁₀ emissions (Figure 7.2). Within the previously explored city centre cluster of high respiratory admissions (Figure 4.2), it is possible to observe that residents within the heart of the cluster experience and create high levels of emissions, thus exacerbating their own environmental problems. Therefore, for this area it may be deemed that a polluter pays situation is already in operation for the socially vulnerable, who reside within areas of high traffic emissions and contributing the most emissions per vehicle. However output areas towards the fringes of this high respiratory admissions cluster, experience real environmental equity issues, with such areas producing low levels of private transport emissions, yet they still experience high levels of residential TPM₁₀ emissions. For instance, residents from the 12 east-central LLSOAs of relevance are found to be relatively deprived (Rank 4.97) and experience residential TPM₁₀ emissions 0.59t/yr. above the citywide average (1.04t/yr.), whilst only emitting 0.009t/yr. from personal transport modes. Meanwhile, socially content communities along Leicester UA's eastern periphery are identified to create high levels of traffic emissions yet experience disproportionately low TPM₁₀ emissions. Here 11 LLSOAs found across Evington and Humberstone on average, portray a deprivation rank of -2.64, and personally contribute 0.027t/yr. of TPM₁₀ whilst residentially experiencing only 0.60t/yr. of TPM₁₀ emissions. This would support the prior notion of environmental injustices existing within intra-urban areas.

	Bivariate: Local Moran's Statistic			
	R ²	Pearson's R	I Value	Z-Score
Children's J00-99 Admissions	0.08	-0.28	-0.17	-3.82
Children's J00-06 Admissions	0.07	-0.24	-0.16	-3.46
Children's J20-22 Admissions	0.07	-0.27	-0.16	-3.43
TPM₁₀ Emissions	0.11	-0.30	-0.26	-5.83
Carstairs Index	0.34	-0.59	-0.42	-9.42
Adult Smoking Prevalence	0.02	-0.14	-0.11	-2.38
Adult Obesity Prevalence	0.11	-0.34	-0.28	-6.27
White British Children	0.07	0.25	0.22	5.17
White Non-British Children	0.02	0.16	0.08	1.94
Indian Children	0.05	-0.22	-0.19	4.19
Other South Asian Children	0.07	-0.26	-0.23	-4.94
Afro-Caribbean Children	0.04	-0.19	-0.09	-2.00

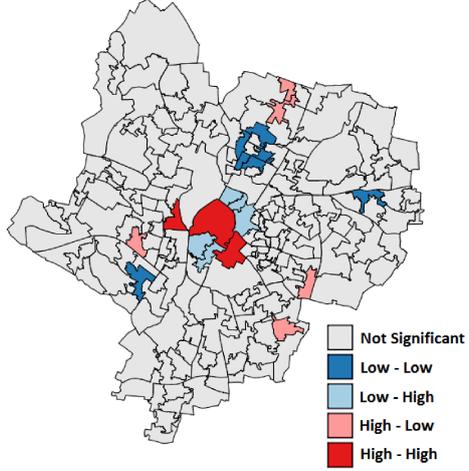
TABLE 7.1: Summary of the Bivariate Local Moran's statistical analysis, revealing the spatial associations between created TPM₁₀ emissions from the workforce's personal transportation modes (i) and health/socio-environmental influences experienced by surrounding locales (j)

Bivariate Local Moran's I: (i) Workforces Created TPM₁₀ Emissions ; (j)

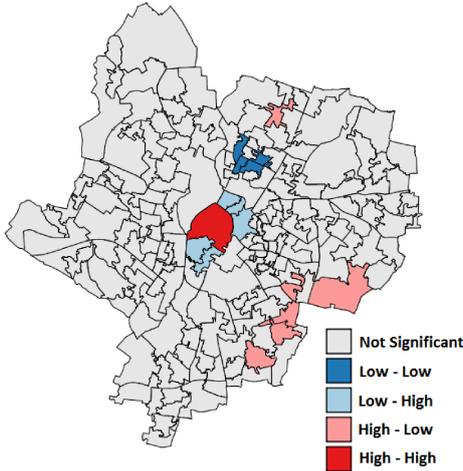
(j) J00-99 Admissions Per 1,000 Children [R² = 0.08]



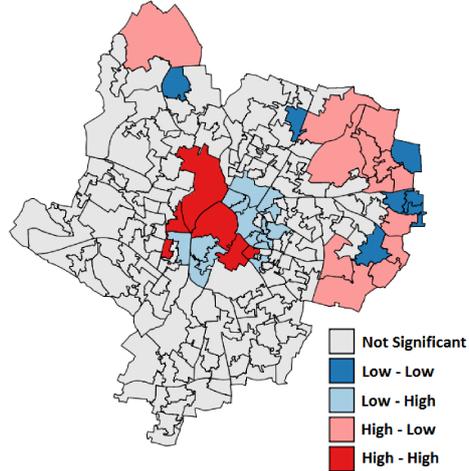
(j) J00-06 Admissions Per 1,000 Children [R² = 0.07]



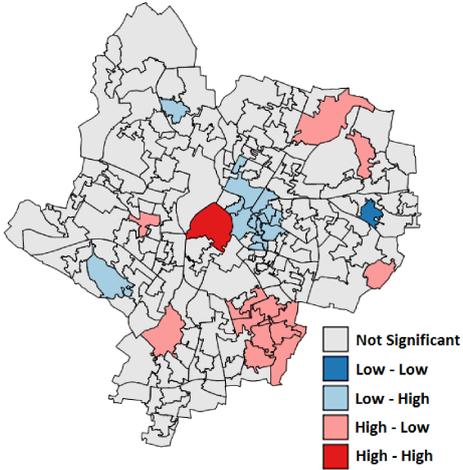
(j) J20-22 Admissions Per 1,000 Children [R² = 0.07]



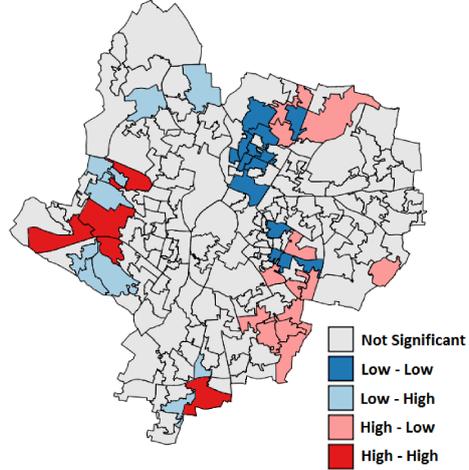
(j) Residentially Experienced TPM₁₀ Emissions [R² = 0.11]



(j) Carstairs Index Of Deprivation [R² = 0.34]

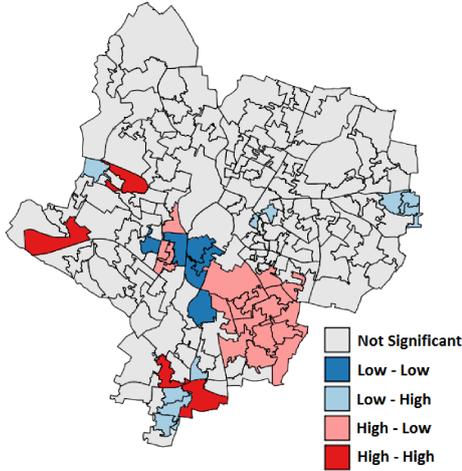


(j) Adult Smoking Prevalence [R² = 0.02]

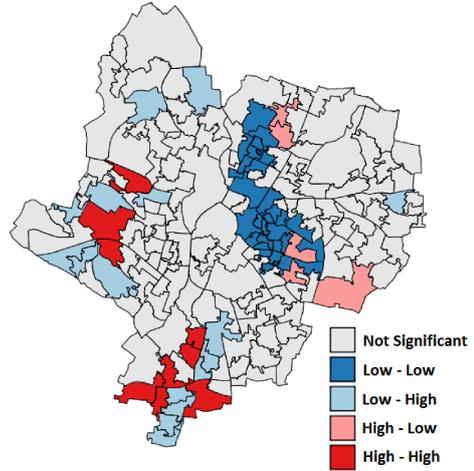


Bivariate Local Moran's I: (i) Workforces Created TPM₁₀ Emissions ; (j)

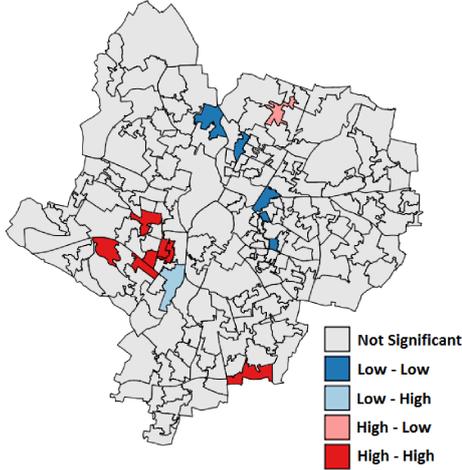
(j) Adult Obesity Prevalence [R² = 0.11]



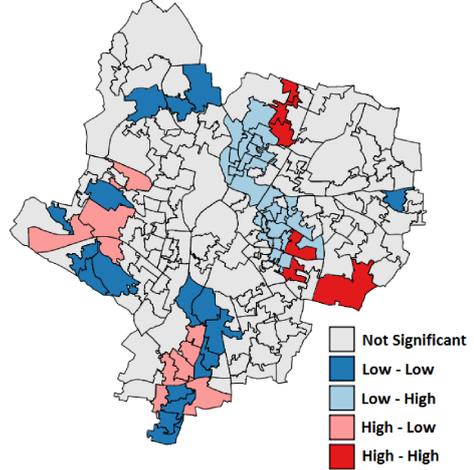
(j) White British Children [R² = 0.07]



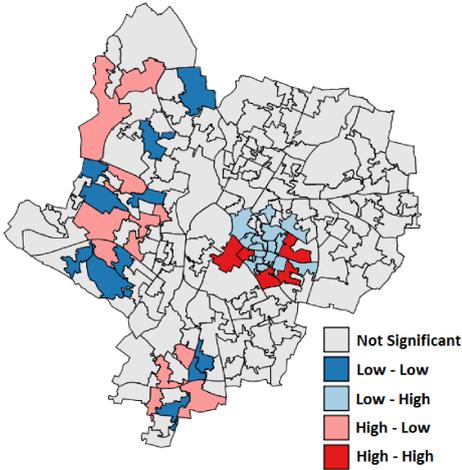
(j) White Non-British Children [R² = 0.02]



(j) Indian Children [R² = 0.05]



(j) Other South Asian Children [R² = 0.07]



(j) Afro-Caribbean Children [R² = 0.04]

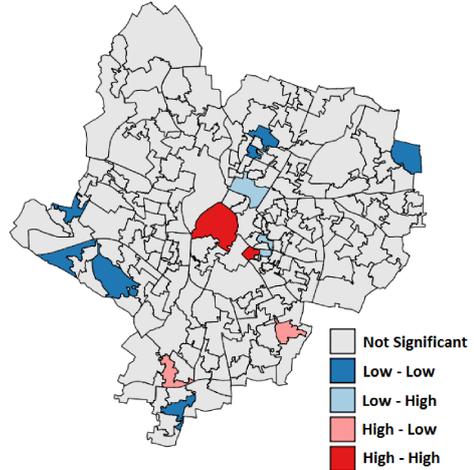


FIGURE 7.2 [PAGES 274-275]: Bivariate Local Moran's I cluster and outlier analysis exploring the first-order relations involving community created transport emissions, and surrounding respiratory or social-environmental measurements

Following on from this, the Bivariate Local Moran's I statistical test was then applied to compare local workforce created TPM₁₀ emissions with neighbouring levels of deprivation (Figure 7.2). Once again, in focusing on the previously explored cluster of high respiratory admissions within the city centre, it is possible to observe that the eastern section of the cluster produces relatively low levels of private traffic emissions and is within an area experiencing elevated levels of deprivation. Meanwhile the focal LLSOA of continued interest is exclusively representative of a deprived community, generating a substantial amount of unfavourable environmental pollutants from personal transportation choices. Another zone of importance is the cluster towards the south-eastern sector of the city, within the ward of Knighton, which consistently experiences low levels of deprivation yet creates significantly high road-transport emissions. As previously discussed, it would appear that the consequences of these elevated personal transportation emissions are not sufficiently felt in a physical or environmental manner by these peripheral communities.

In viewing Table 7.1, the Bivariate Local Moran's I Z-scores indicate that the health, social and environmental indicators of interest, on the whole represent a significant negative relationship ($P \leq 0.05$) with respect to community created TPM₁₀ emission levels (with the exception of White-British, White Non-British and Indian residencies). For instance, as personal transportation emissions increase one may observe a decline in respiratory hospitalisations and community levels of deprivation. Whilst of significance, a majority of these relationships would appear to yield weak structures at a global scale, with the exception of the somewhat clear-cut spatial relationship associated with community deprivation levels (I-Value -0.41). To a lesser extent, similar negative global interaction structures of a weak-moderate strength may also be observed with regards to residentially experienced TPM₁₀ (I-Value -0.26) and levels of obesity prevalence (I-Value -0.28). Nevertheless for the most part one is to accept that the relationships between emission creation and various societal classifications are not universal but rather localised to specific residential pockets.

The Bivariate Local Moran's I analysis of locally created TPM₁₀ emissions from a communities workforce with neighbouring levels of adulthood obesity (Figure 7.2), identifies a substantial clustering of highly polluting yet physically healthy inhabitants, comprising of 16 south-easterly LLSOAs from Knighton and aspects of Castle. Here typical levels of adult obesity prevalence of 14.61% are substantially beneath expected citywide rates of 24.30%, yet these communities created on average 0.033t/yr. of TPM₁₀ emissions from personal transport. In fact, this cluster contains five out of the city's top 10 polluting communities, with all cluster observations falling within the upper 35th percentile of polluters in personal transportation terms. One should also note that this affluent cluster (Rank of -4.22) also typically experiences

low levels of TPM_{10} emissions (0.97t/yr.), and reduced annual rates of children's J00-99 admissions (32.78 per 1,000).

In contrast to this pocket and the overall global trend (Table 7.1), a second minor cluster of 4 physically healthy inner-city communities (15.10% obesity prevalence) are observed to emit low levels of personal TPM_{10} emissions (0.003t/yr.). Such communities broadly portray the cities expected level of socio-economic status (Rank of 0.22), yet are burdened by substantially high levels of residentially experienced TPM_{10} emissions (2.13t/yr.), and face high rates of children's J00-99 admissions (54.08 per 1,000). Within this Low-Low bivariate cluster, levels of commuting to work via private transport are thought to be low, perhaps coinciding with a favouring of physical transportation measures by populaces with a slightly below par socioeconomic status, hence their low obesity rates. If this is the case, then one might expect these inhabitants to face elevated exposure periods with the environmental burdens placed on to them by others, which appears to be captured by the reduced respiratory health of vulnerable community members (i.e. children).

As previously discussed, local cluster detection would indicate that a somewhat weak-moderate global trend would exist in explaining that as obesity decreases level of private transport emissions increase. However, this relationship would appear mainly driven by the affluent and healthy south-eastern communities of the city. Towards the cities southern limits of Eyres Monsell, exist 6 LLSOAs experiencing high levels of adult obesity (29.00%). Yet, out of these noteworthy communities, only two are denoted to be high polluters from personal transportation modes (0.021-0.024t/yr. TPM_{10}). In-fact, the rest of these communities were deemed to be low emitters, averaging only 0.003t/yr. of personally created TPM_{10} emissions. It is somewhat unlikely that obese communities would regularly partake in physical modes of transport; therefore, it is highly probably that the obese favour the use of public transport.

Bivariate Local Moran's I outputs comparing workforce created TPM_{10} emissions to neighbouring residential levels of 'White British' inhabitants, is once again representative of the clear divide between western majority and east-central minority groups (Figure 7.2). In viewing Table 7.1, the Bivariate Local Moran's I Z-scores labels 'White British' residency to share a significant positive global relationship ($P \leq 0.05$) with respect to community created TPM_{10} emissions, at an albeit weak level. In that, as personal transportation emissions increase so does the community composition level of White British residents. An expansive section of low 'White British' residency and low creation of personal TPM_{10} emissions may be observed, particularly across the east-central wards of Latimer, Spinney Hills and Stoneygate.

In Stoneygate, 6 out of its 11 LLSOAs were positioned within this cold spot of interest, in which the workforce personally created approximately 0.016t/yr. of TPM_{10} yet unfairly

experienced 1.12t/yr. of TPM₁₀ emissions. Levels of 'White British' children (10.42%) appeared to be replaced by those of 'Indian' (54.50%), 'Other South Asian' (22.09%) and 'Afro-Caribbean' (6.14%) ethnicities. In Spinney Hills, 11 out of a possible 14 LLSOAs were of interest, whereby the workforce personally created only 0.009t/yr. of TPM₁₀ yet unfairly experienced 1.50t/yr. of TPM₁₀ emissions. As before, levels of 'White British' children (7.08%) typically appeared replaced by those of 'Indian' (66.80%), 'Other South Asian' (17.28%) and 'Afro-Caribbean' (3.51%) ethnicities. Across Latimer, 4 out of 8 LLSOAs were signalled to be of importance, in that the workforce normally created around 0.012t/yr. of TPM₁₀ yet unfairly experienced 1.40t/yr. of TPM₁₀ emissions. Once more, average levels of 'White British' children (8.85%) appeared substituted by those of 'Indian' (75.49%), 'Other South Asian' (6.83%) and 'Afro-Caribbean' (1.53%) ethnicities. One may also note that children's J00-99 admission rates were observed to decline in response to Indian residency in spite of residential TPM₁₀ exposures, as observed by rates of those explored communities in Stoneygate (43.80 per 1,000) and Latimer (23.32 per 1,000).

However, communities predominantly constructed from White British families are not necessarily to blame for the majority of personal transportation emissions created by the cities workforce. For instance, upon exploring the 'White British' stronghold towards the cities southern limits, one may observe that the 13 LLSOAs contained within this pocket of interest evenly represent low emitting (7/13) and high emitting (6/13) communities. Here, the 7 Low-High LLSOAs predominantly housing children of 'White British' ethnicity (88.33%), created only 0.013t/yr. and residentially experienced 0.91t/yr. of TPM₁₀ emissions. Such communities were typically of a somewhat deprived nature (Rank of 1.63), with high levels of smoking (40.30%) and obesity (28.04%) characteristically prevailing amongst residents. Meanwhile, the 6 High-High LLSOAs contained comparable levels of 'White British' children (87.28%), yet they created 0.024t/yr. whilst residentially experienced a measly 0.95t/yr. of TPM₁₀ emissions. These communities appeared of a similar somewhat deprived nature (Rank of 1.38), once more exhibiting high levels of smoking (40.30%) and obesity (28.04%) prevalence. A similar story of high and low emission creation would appear to occur for 'White British' communities around the wards of Braunstone and New Parks, representative of the cities western outer limits.

Whilst these observations are of a contradictory manner, in that many characteristics are shared between the alternate responses, one should note in terms of ethnicity, that the test highlights the cities slightly deprived 'White British' residents to be of particular interest. In that financially they can afford personal transportation modes, however this is most likely to consist of ageing stock that exhibit less stringent emission standards. Perhaps their choice of vehicle is limited by other financial strains relating to healthy lifestyle choices, causing such

communities to teeter on the edge of choosing between either persisting with public transport or purchasing their own poor vehicle stock. Furthermore, these privately run vehicles would appear to not directly impact their own suburban communities, but it is quite possible that such a fleet would become an issue within the more compact central urban environments that are travelled through. Within the city's traffic management plans it could be of future interest to target such a social group in a strategic manner, through adopting schemes which improve and or encouraging the use of public transportation, or through providing assistance towards the maintenance of their vehicle stock.

However, it would appear that it is not race but deprivation, which is the key driving factor behind the creation on personal TPM₁₀ emissions; although elements of race, appear involved in determining affluence. For instance, Leicester's most affluent communities (Lower 10% of Carstairs Index), personally created a hefty 0.035t/yr. of TPM₁₀, yet residentially experiencing only 0.85t/yr. of TPM₁₀ emissions. Whilst, the composition of children from such communities remains predominantly of 'White British' origin (70.34%), these areas are also representative of elements of successful integration with the 'Indian' community (17.30%). Furthermore, J00-99 admissions were on average recorded at the low rate of 32.88 per 1,000 children, 6.55 cases bellow expected. These 10% most affluent areas were primarily located around the wards of Knighton and Humberstone, with some minor pockets around Western Park, and a single element existing at the top section of Beaumont Leys. In exploring all LLSOA communities within the ward of Knighton, one may observe that a far greater level of integration has occurred, whereby 'White British' and 'Indian' ethnicities respectively account for 61.53% and 22.20% of the child inhabitants. Overall, this would suggest that the most affluent communities in terms of health, financial, environmental and cultural terms, are accountable for creating a substantial proportion of environmental issues that affect those less fortunate. In conclusion, the greatest polluters are currently not adhering to the PPP's. In contrast to prior recommendations, a stronger more direct action would be most preferential across those communities, whom are financially able to alter their mode of travel, if such services are sufficient.

Bivariate Local Moran's I outputs comparing workforce created TPM₁₀ emissions to neighbouring residential levels of ethnic minorities, would also appear to unanimously highlight those modestly deprived 'White British' communities along western and southern peripheral areas of ethnic interest. It was previously discussed that the financial strains in such areas, seemingly worsened by participation in unhealthy lifestyle activities, left these inhabitants open to purchasing low-cost environmentally unfavourable vehicle stock rather than travelling via public modes. As such, communities are not the city's main cause for

concern, but rather they represent a preventable future burden of minor-moderate force; it was recommended that travel choices within these financially strained swing communities be addressed by a cautious approach, as to not restrict their human rights to freely travel. In focusing on the distribution of 'Indian' children, one may see that the emission creation relationship portrays a strong mirror opposite to that described by families of 'White British' origins. As described in prior chapters, 'Indian' residency is particularly high across the east-central (outer-city centre) wards of Latimer, Spinney hills and Stoneygate, which have been associated with creating low emission levels from personal transportation. However, select eastern peripheral communities towards Evington and Rushey Mead, located along the northern and southern tips of this 'Indian' cluster, uphold these high levels of 'Indian' residency (63.25%) yet contrastingly emit high levels of personal TPM_{10} (0.023t/yr.). Interestingly, these High-High communities' face up to the city's most affluent and polluting wards of Knighton and Humberstone, which characteristically represent a successful integration of the cities elite 'White British' and 'Indian' families. Perhaps such locales mark the next wave of ethnic minority families to move up the social ladder, encouraging further integration with the surrounding affluent 'White British' populaces.

Upon examining the 'Other South Asian' communities of high residency around Stoneygate, it would appear that this minority group also tends to emit relatively low levels of pollutants from personal modes of transportation. Across this groups 15 LLSOAs of low TPM_{10} creation (0.012t/tr.) and high residency (20.52%), both deprivation levels (Rank of 2.88) and residential TPM_{10} exposures (1.38t/yr.) were recorded at a moderately high magnitude. However, 5 nearby LLSOAs with similar ethnic residency levels (20.40%) were identified to create substantial TPM_{10} emissions (0.012t/tr.), whilst residentially experiencing noticeably reduced rates of deprivation (Rank of -0.05) and TPM_{10} emission exposures (1.33t/yr.). This would appear to provide further strength to the argument that, communities on the threshold of affording personal forms of transportation perhaps require additional measures of assistance in maintaining vehicle upkeep, or incentives to switch to public modes of travel.

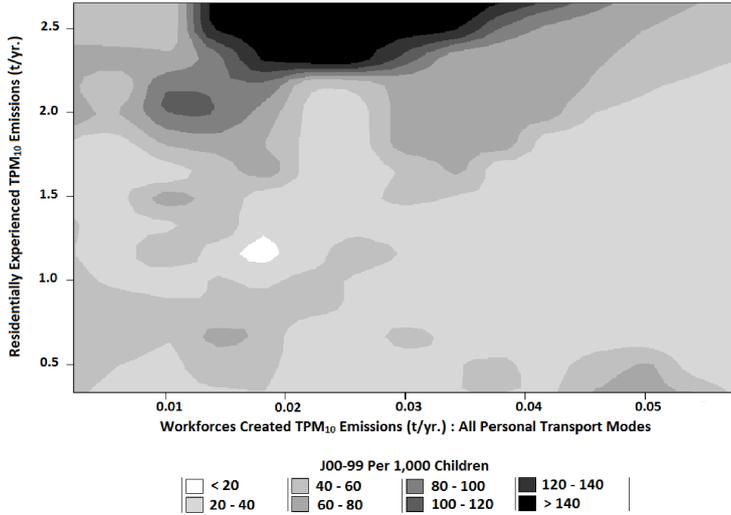
From the Bivariate Moran's I plot comparing workforce created TPM_{10} emissions to neighbouring residential levels of 'Afro-Caribbean' inhabitants, it is hard to define how this social group participates in the creation of personal transportation emissions; as made evident by its global measures (Table 7.1). Whilst low levels of 'Afro-Caribbean' spatially typically coincide with low emission creation, a much more diverse response may be observed around central locales where high residency levels occur. One may note that the inner-city focal community of interest is deemed to be a hot-spot on both accounts, deemed to be within the upper 35th percentile of personal TPM_{10} contributors (0.022t/yr.) whilst being the most

polluted of Leicester's LLSOA communities. However, 'Afro-Caribbean' residency within this spot is recorded at 0.00%; rather the locale spatially signifies the group's high residency level at its 9 immediate surrounding neighbours (10.28%).

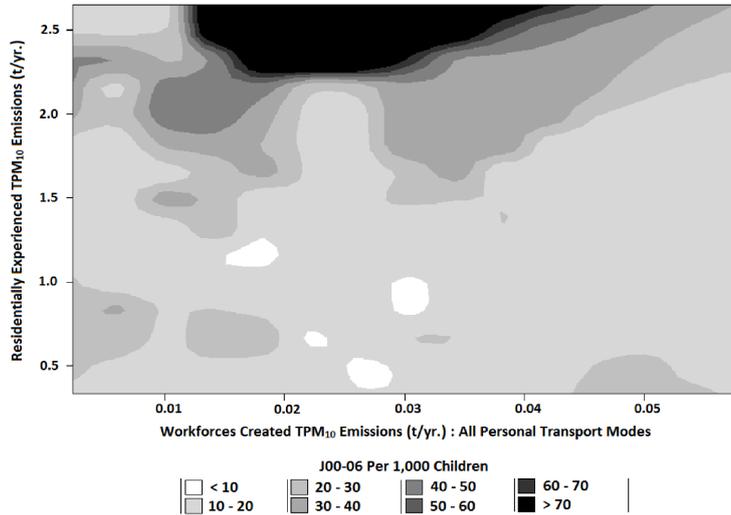
Spatial patterns of environmental injustices are illustrated in further detail through a set of contour plots, simultaneously examining created and residentially experienced TPM_{10} emissions against a measurement of respiratory or social status (Figure 7.3). In assigning children's overall respiratory hospital admissions (J00-99) as the final factor, one may observe from the bottom right quadrant that highly polluting communities whom residentially experience relatively low levels of TPM_{10} emissions, typically experience fewest respiratory issues. As previously shown in the spatial regression analysis (Chapters 4-5), children's respiratory cases rise in relation to increased residentially experience TPM_{10} emissions, the effects of which generally appear to be felt in a more meaningful manner by those communities which emit lower levels of pollutants from personal modes of transportation. One should note that children with severely reduced respiratory health (≥ 80 J00-99 admissions per 1,000), tended to be housed within communities contributing modest amounts of transport related pollutants, which in a just situation would result in only moderate health implications. However, at present these residents appear unfairly plagued by the contributions of external communities, whom health wise pay very little. Similar respiratory health patterns emerge in relation to the spread of both URT and LRT infections during childhood.

Contour plots holding deprivation as a third factor, clearly reveal a socially banded structure to be in existence within the City of Leicester; identifying affluent residents to generally contribute the highest levels of private transport emissions whilst residing in areas experiencing low levels of road-transport emissions (Figure 7.3). The social bands, whilst remaining visually present, would appear to represent a wider range of creation-exposure scenarios as one reaches expected citywide socio-economic levels, indicating that a smooth deprivation gradient is in operation. Meanwhile, those most deprived are found almost exclusively within the upper left quadrant, experience elevated environmental burdens whilst personally emitting few TPM_{10} emissions. Interestingly the deprivation contour plot reveals a minor pocket of modestly affluent residents whom both emit and experience few TPM_{10} emissions, however it would appear that only a singular LLSOA within the ward of Freeman at present sufficiently follows suit. In raising environmental awareness amongst the city's most affluent residents, it would be hoped that many of the cities transport related burdens could be reduced. At present, strong inequalities seemingly occur with respect to road-transport emissions across this British intra-urban environment.

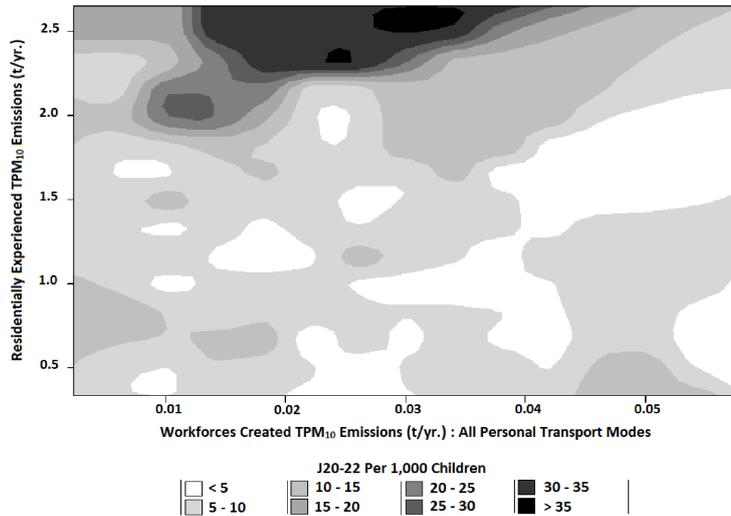
Contour Plots Of Children's J00-99 Hospital Admissions Vs Created & Experienced TPM₁₀ Emissions



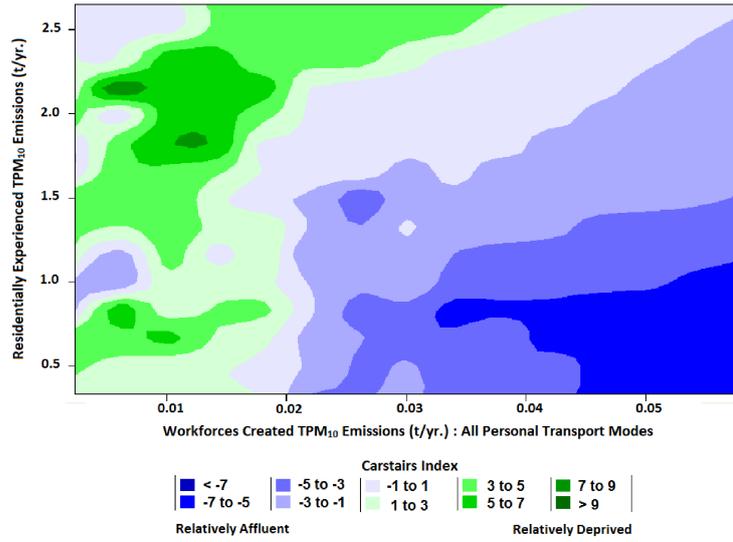
Contour Plots Of Children's J00-06 Hospital Admissions Vs Created & Experienced TPM₁₀ Emissions



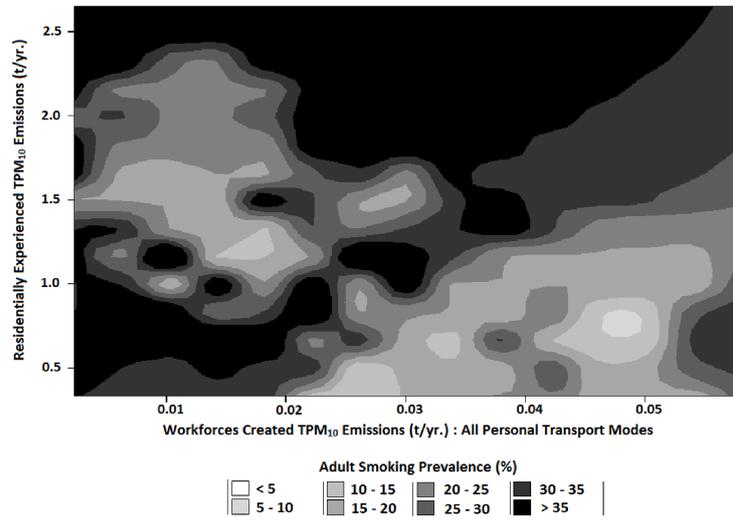
Contour Plots Of Children's J20-22 Hospital Admissions Vs Created & Experienced TPM₁₀ Emissions



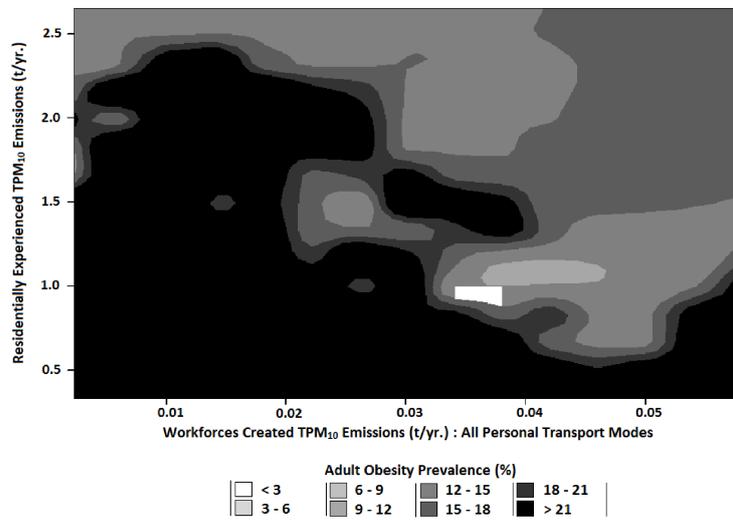
Contour Plots Of Deprivation Vs Created & Experienced TPM₁₀ Emissions



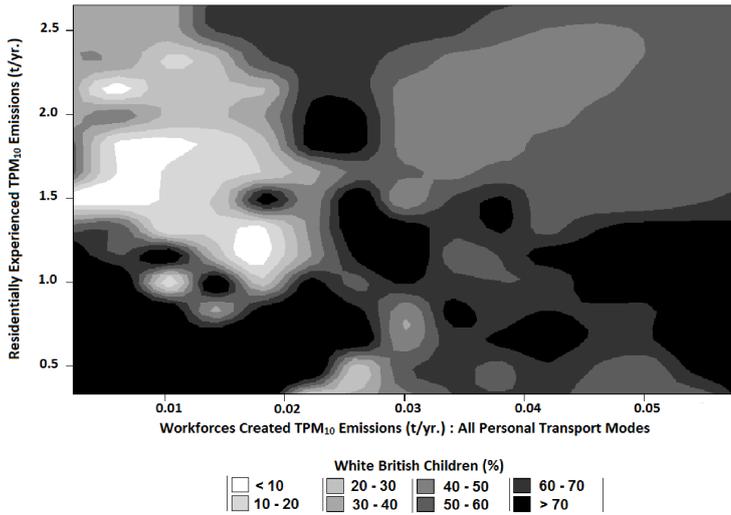
Contour Plots Of Smoking Prevalence Vs Created & Experienced TPM₁₀ Emissions



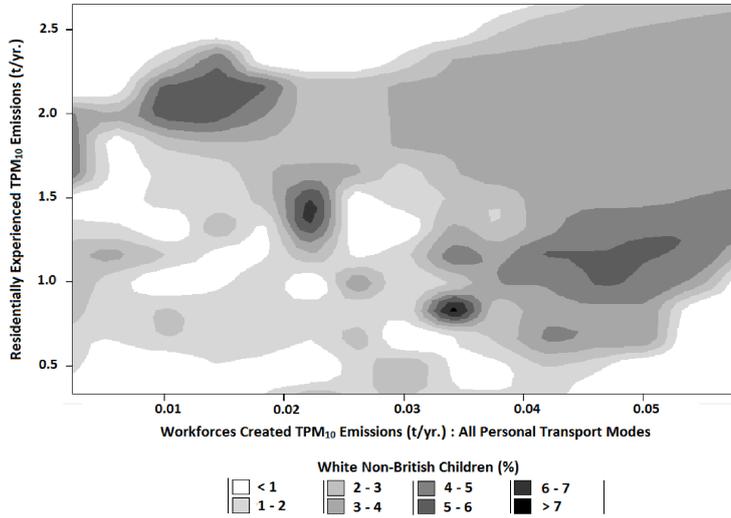
Contour Plots Of Obesity Prevalence Vs Created & Experienced TPM₁₀ Emissions



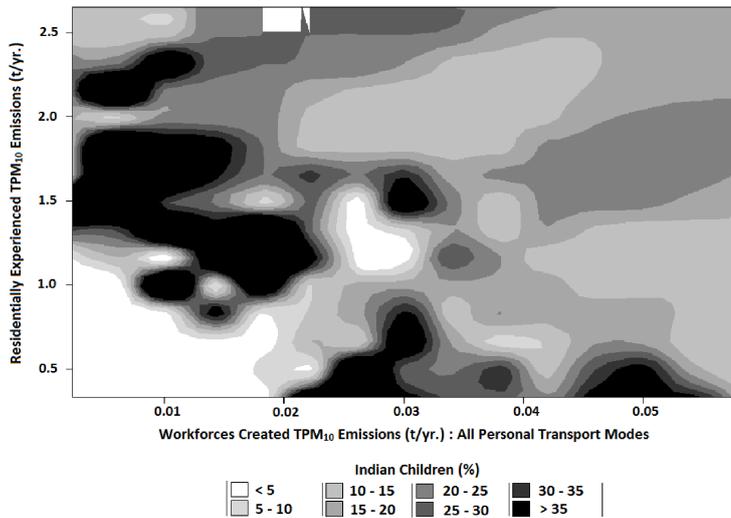
Contour Plots Of White British Residency Vs Created & Experienced TPM₁₀ Emissions



Contour Plots Of White Non-British Residency Vs Created & Experienced TPM₁₀ Emissions



Contour Plots Of Indian Residency Vs Created & Experienced TPM₁₀ Emissions



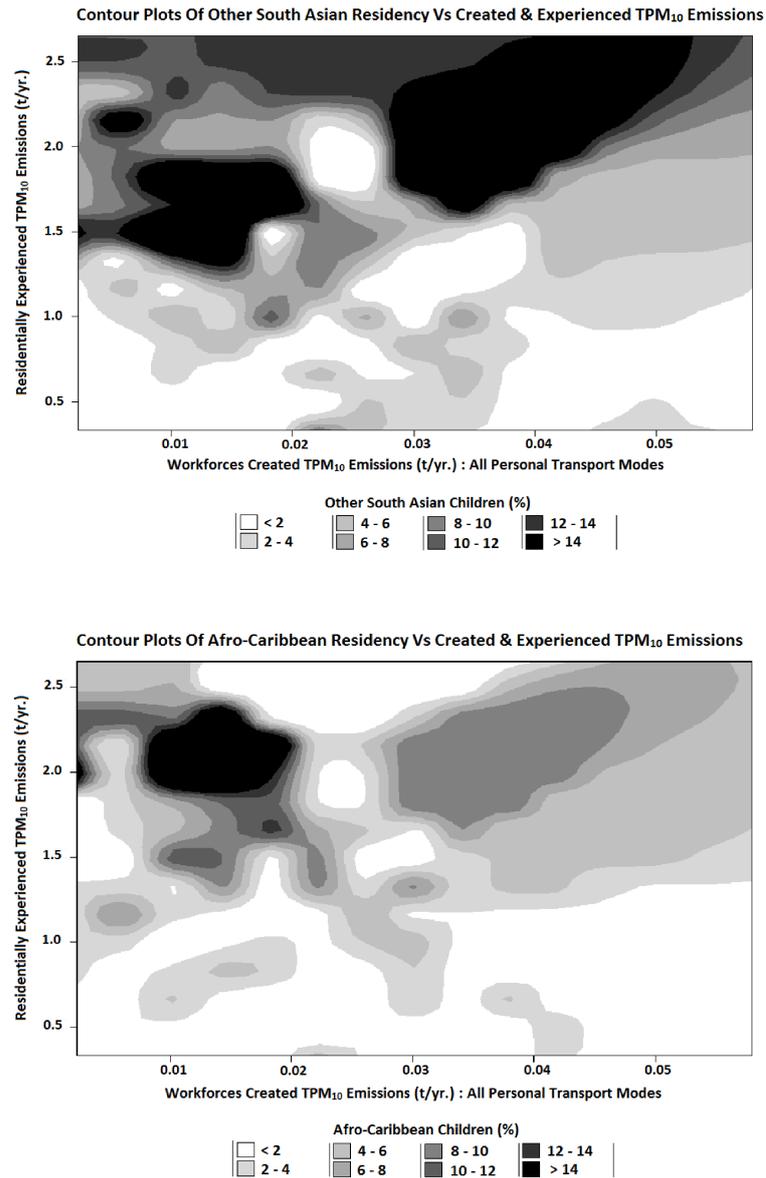


FIGURE 7.3 [PAGES 282-285]: Contour plots, simultaneously examining created and residentially experienced TPM₁₀ emissions against a measurement of respiratory or social characteristic

In exploring whether lifestyle choices determine the thought process behind a communities environmental contribution, one may observe that the contour plots have detected some rather broad structural bands in relation to smoking prevalence (Figure 7.3). Here low levels off smoking tend to occur within those communities whom residentially experience low levels of TPM₁₀ emissions, whilst heavily contributing towards the cities wider TPM₁₀ emissions problems. In particular, these relatively affluent and healthy communities are to be found around Leicester's south-eastern periphery. Although these communities are self-conscious in mitigating the level of risk placed on their children (i.e. passive smoking), they appear unaware or simply uninterested in how they shift their transport burdens onto others; perhaps a case of

out of sight out of mind. A careful targeting of information towards these communities, informing one of their actions may in the short term, raise answerable questions on their preferential mode of transportation. Meanwhile the banding structure defining elevated levels of smoking prevalence appears less clear, as this diagonal band concentrating on low emitting yet highly exposures communities is split by an expansive pocket that conflictingly to the prior statement, chose not to smoke. These non-smoking, deprived inhabitants, who are unfairly burdened by others private transport pollutants, are typically representative of minority communities residing around central Leicester. In general, this contour plot would indicate that smoking prevalence is highest amongst those communities which contribute moderate levels of TPM_{10} emissions, with such levels rising in accordance to other environmental exposures at their place of residence.

In terms of obesity prevalence, one may observe that a clear pattern emerges, identifying those most obese as emitting the fewest pollutant from personal transportation measures (Figure 7.3). In addition, LLSOA communities residentially experiencing TPM_{10} emission levels beneath expected citywide exposures ($\leq 1.04t/yr.$) are also more likely to be classified as obese. To a certain extent this is an unexpected outcome (on the understanding that obese persons are less likely to travel via physical modes), as an increased use of public transport would potentially be associated with moderate residential exposure rates, caused by an increase in the frequency of heavy vehicles entering such residential neighbourhoods. It is thought that these observations represent both deprived and affluent suburban communities, whereby public transport facilities are positioned away from the place of resident, and as such are unable to accumulate within residential street canyons (which are typically less confined within suburban areas). If this is indeed the case, then those environmentally unexposed to the TPM_{10} problem, are also to an extent impacting external communities through their use of public transport, while unfairly feeling relatively few environmental burdens themselves.

To conclude this section, contour plots were created in order to further explore the pollutant interactions between the cities majority and key minority groups of interest (Figure 7.3). Typically 'White British' families reside within communities experiencing moderate to low residential TPM_{10} exposures. In particular LLSOA communities housing >70% of children classified to be of 'White British' origins, are residentially exposed to $<1.5t/yr.$ in TPM_{10} emissions. However, communities constructed from predominantly 'White British' families are not fundamentally accountable for Leicester's environmentally unjust predicament, as only a small segment of this societal group highly pollute and residentially experience few discharges. Nevertheless it should be noted that the top left quadrant, denoting low contributions and high exposures, typically house few children of 'White British' origins (0-50%). This quadrant

appears to be primarily occupied by those families of 'Afro-Caribbean' and 'Other South Asian' ethnicity. In addition, graphical outputs reveal that a full if not slightly excessive force, of environmental burdens are felt by these two ethnic minority groups, when they are found to inhabit communities providing moderate-high contributions of personal TPM₁₀ emissions. In contrast, other social groups across the city look as if they either completely avoid or experience only a mild impact in relation to their actions. Such findings would appear to confirm that sections of the cities ethnic minority groups are comprehensively burdened, in a socially unjust manner, by the contributions of others.

Whilst families of 'Indian' origins maintain a graphical distribution of communities, whom emit little yet face the environmental effects of others, such unjust burdens are of a reduced scale within these communities; marked by this ethnic subsets standing in-between the upper and lower TPM₁₀ exposure quadrants. However, unlike the two preceding ethnic minority groups, families of 'Indian' origins have a second key distribution across communities, which face few but emit moderate-high quantities of personal TPM₁₀ emissions. To a lesser extent, persons of 'White Non-British' residency mimic these two distributions portrayed by the 'Indian' populace. From these findings, it is possible to conclude, that those accountable for the major environmental burdens felt by inner city communities, are of affluent communities representative of a successful integration of the ethnic majority with persons of 'Indian' and 'White Non-British' origins. The outputs of these contour plots are in agreement with those of the Bivariate Moran's I cluster analysis, which primarily identified the ward of 'Knighton' as housing residents whom do not presently adhere to the PPP's.

7.3. RE-EXAMINATION OF THE 'DOUBLE BURDEN'

In examining the collective influence of socio-environmental influences on the complete set of respiratory conditions, Chapter 4 found environmental inequities to prevail selectively across sections of the model British multicultural city of Leicester. In particular, children from lower social class households tended to reside within areas experiencing relatively high levels of road-transport emissions, thought to be substantially created by external affluent communities. Such interactions are in accordance with a preceding body of EJ research, which have consistently reported the 'double-burden' of deprivation and air pollutant exposure as a key explanatory factor in defining health disparities (Crouse et al 2009, Kingham et al 2007, Naess et al 2007). Following on from this, an in-depth exploration of specific respiratory conditions within Chapter 6, documented the combined accountability of deprivation and

road-transport emissions in the decline of children's respiratory health across entire communities. It was proposed that certain socio-environmental factors were particularly adept at infiltrating a child's undeveloped immunological system, often resulting in the initiation of an URTI episode, with prolonging recovery times likely occurring from sustained exposures. If a sufficient level of recovery was not reached in time for the cold season, then the child may then become host to a viral infection, exacerbating previous respiratory complaints, potentially resulting in LRT conditions of greater severity. Thus underlining how this 'double burden' affects all aspects of respiratory health during childhood.

Within the above section of this chapter, graphical plots of deprivation against intensities of residentially created and experienced TPM_{10} emissions, clearly identified elements of social banding across the City of Leicester. Here affluent residents contributed the highest levels of private transport emissions whilst residing in areas experiencing low levels of road-transport emissions, whereas the reverse of this relationship occurred in large across those communities of a deprived nature. To further investigate this 'double burden' a new set of contour plots were constructed, simultaneously examining levels of deprivation and residentially experienced TPM_{10} emissions against a third measurement of respiratory or social status (Appendix F1). In assigning children's overall respiratory hospital admissions (J00-99) as the final factor, one may observe that the bottom left quadrant representative of decidedly affluent communities whom residentially experience relatively low levels of TPM_{10} emissions, typically experience the fewest respiratory issues. In contrast, children residing within communities represented by the top right quadrant, which are characteristically deprived and residentially experience relatively high levels of TPM_{10} , were identified to exclusively experience the most severe reductions in respiratory health (≥ 80 J00-99 admissions per 1,000). Prior contour plots (Figure 7.3), indicated that these children tended to be housed within communities contributing modest amounts of transport related pollutants, which in a just situation would result in only moderate health implications. However, at present these residents appear unfairly plagued by the contributions of external communities, whom health wise pay very little. Respiratory health would appear to decline as either deprivation or residential pollutant exposure increases, with the worst effects felt when both influences combine.

In terms of lifestyle choices, the contour plots would occur to confirm that by enlarge those most affluent and least polluted, through shifting their personal transportation burdens onto others, generally exhibit low smoking rates (Appendix F1). As before, central locales containing some of the most deprived communities, whom face moderate-excessive residential levels of TPM_{10} , also displayed low levels of smoking uptake; perhaps in recognition

of the multitude of burdens already thrust upon them. Peaks in smoking prevalence may be found across those most deprived communities where residentially experienced TPM_{10} is low, representative of the cities western suburbs primarily occupied by 'White British' families. A second peak is also observed across highly polluted communities, whereby smoking prevalence exists irrespective of socio-economic status. Overall, this lifestyle choice appears somewhat detached from the 'double burden' driving children's respiratory outcomes.

With respect to levels of obesity prevalence, it was previously shown that those most obese communities emitted the fewest pollutants from personal transportation measures. This remark is reconfirmed here, as those deprived communities (Rank of >0) are typically the most obese; with the exception of half of the upper right quadrant (representative of inner city inhabitants), which displays low levels of obesity across considerably deprived and polluted locales (Appendix F1). Overall those least obese would tend to reside within residentially polluted settings, irrespective of social status. Perhaps it is the highly active nature of some deprived communities, which magnifies their environmental exposures. Here the low cost (if any) of physical transportation measures promote physical wellbeing, at the expense of increasing roadside exposure periods, which could go some way in explaining why children's respiratory health is substantially diminished across these communities.

Contour plots, identified that 'White British' residents by enlarge do not face this 'double burden', with deprived communities from this ethnic majority typically residing across suburban areas, experiencing low levels of transport pollutants (Appendix F1). In addition, it would seem as though affluent 'White British' families characteristically experience moderate pollution exposures, and as such some of these communities partially pay for their contribution of personal transport emissions. As previously discussed, it was observed that the most prosperous communities characterised by the successful social integration of select minority groups with a prevailing 'White British' majority, were observed to highly pollute shifting such burdens onto other communities. Social status rather than ethnicity would therefore appear to define a community's environmental attitude and attributes, although the two mechanisms are often intertwined.

As before, peaks in 'Indian' residency form across two residential distributions (Appendix F1). The minor distribution is located within the bottom left quadrant, representative of those that have socially integrated with certain affluent 'White British' communities, whom emit high levels yet unjustly experience few environmental burdens. A second rather extensive distribution mode is defined by moderately social and environmental burdens, although this distribution does in part expand into the upper right quadrant indicative of the 'double burden'. However when this occurs, 'Indian' communities would

appear less effected by such burdens (<80 J00-99 admissions per 1,000) compared to other inhabitants (≥ 80 per 1,000). In examining the cities other ethnic minority groups, no clear pattern emerged in relation to those children of 'White Non-British' origins. Meanwhile, contour plots identified 'Other South Asian' and 'Afro-Caribbean' children as comprehensively occupying those most polluted communities, of which their greatest distributions are to be found within locales experiencing a 'double burden' of deprivation and pollution.

In a concluding thought, when examining the four conceivable quadrants individually associated with a spectrum of 'double burden' interactions, questions may arise asking whether severely environmentally burdened affluent communities actually exist within Leicester. On closer inspection of the dataset, one should note that only 4 LLSOAs exist which portray characteristics of highly polluted neighbourhoods ($TPM_{10} > 1.5$) containing inhabitants whom are not socially deprived (Rank > 0). These communities are to be found around the southern fringes of the city centre, three of which are located within the ward of Westcotes and one within Castle. Each observation respectively experiences residential TPM_{10} emission levels of 1.96, 1.67, 1.54 and 1.87t/yr., whilst recording corresponding deprivation ranks of -0.25, -0.39, -2.02 and -0.02. In comparison to the three other quadrants, observations are extremely sparse, and where available they are confined to only the upper left quartile. Therefore, an element of caution should be taken when drawing conclusions from the bottom left quadrant, with explanatory outputs for an environmentally disadvantaged yet socially affluent community existing in purely a theoretical manner (derived from distant contours). Such findings relating to these theoretical communities therefore require further scrutiny, with further research conducted across additional intra-urban environments to confirm their existence.

7.4. BOUNDARY ANALYSIS OF COMMUNITY CREATED TRANSPORT EMISSIONS

Following on from the procedures set out in Chapter 6, univariate crisp polygon wombling techniques were applied to detect boundaries in community created transport emission levels (from private modes). This was conducted in order to establish whether significant proximity based thresholds exist in relation to emission production, and spatial gradients in children's respiratory outcomes or social-environmental influences. If such thresholds indeed exist, then they are of interest for (a) confirming preceding statements, and (b) determining whether locally created TPM_{10} emissions burden proximal or distant residents.

Boundaries in community created TPM₁₀ emissions were associated with BLV's ranging from 0.18-0.61. The sharpest transitions were observed to occur around the upper section of the city's most northerly ward of Beaumont Leys, marking its border with Abbey (Figure 7.4). Here, three boundaries separating an individual LLSOA from Beaumont Leys from communities contained within Abbey, were shown to record an average BLV of 0.43. In-fact, two of these boundaries fell within the cities top five community transitions in personally created transport emissions. Within the Beaumont Leys LLSOA deprivation levels (-5.26), created TPM₁₀ (0.058t/yr.) and experienced TPM₁₀ (0.58t/yr.) emissions were registered to exist at noticeably distinct levels, to those corresponding values of 1.80-2.73, 0.014-0.019t/yr. and 0.64-0.69t/yr. recorded across Abbey's LLSOA communities. From these readings, boundary detection techniques would appear to confirm the previously explored, distinguishing community characteristics, involved within the creation of pollutants from personal transportation modes.

One may also note that additional boundaries are to be found between Beaumont Leys intra-ward communities, specifically this relates to moderate levels of TPM₁₀ creation within the wards core facing up to a sharp singular reoccurrence of high TPM₁₀ creation at the wards base. Here, deprivation levels (-3.32), created TPM₁₀ (0.040t/yr.) and experienced TPM₁₀ (0.85t/yr.) emissions recorded by the wards lower LLSOA, once again noticeably differed from those corresponding values of 3.49-5.71, 0.010-0.017t/yr. and 0.76-0.96t/yr. recorded within the heart of Beaumont Leys. These observations highlight a rather complex set of localised shifts in pollution dynamics by Leicester's northern communities (relating to minor pockets of affluence), which starkly contrast to the broader somewhat uniform intra-ward contributions of the cities south-easterly communities.

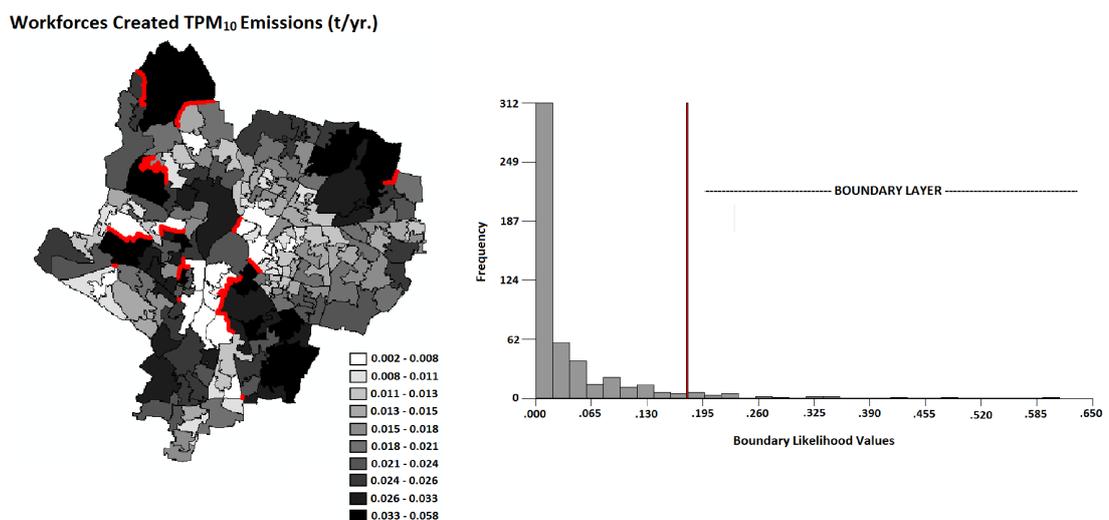


FIGURE 7.4: Map illustrating the top 5% of Boundary Elements (red) across respective decile distributions of community created transport emission levels (from private modes)

Whilst the south-easterly ward of Knighton is universally representative of high TPM_{10} contributions, one may note that the adjacent northerly communities track a gently diminishing transition in personal pollutant contributions (Figure 7.4). It is not until the inner-city communities are reached that a noticeable cut-off is witnessed, as determined by three boundary elements providing an average BLV of 0.28. In addition, all three elements fall within the cities top ten community transitions in personally created transport emissions. It would appear that two of these elements form a wider extent of six boundaries (BLV 0.23-0.32), which focus on the partitioning off of Castles southern section of moderate-high polluters from those whom contribute little along the outer-city centre wards of Westcotes and Freeman (Figure 7.4). Here, Castles three southern LLSOA communities respectively record average deprivation, TPM_{10} creation and TPM_{10} exposure levels of -2.55, 0.031t/yr. and 1.43t/yr., compared to their opposing community values of approximately -0.31, 0.002t/yr. and 1.66t/yr. Whilst the distinguishing features of polluting communities remain, they are of a slightly blurred nature when compared to those along the northern created emission boundaries. As explained this is an outcome of a geographically smoother transition between polluting communities in southern Leicester; created primarily in accordance to a more gradual change in social class.

It is down to such smooth transitions that the northern edge of Knighton is not partitioned off in this boundary analysis; this follows on from the cluster detection outputs recognition of a transition from low to moderately polluting 'Indian' communities inhabiting these adjacent locales (Figure 7.2), which has arose on account of increased affluence. In-fact this gradual geographic transition in affluence and pollution may be observed across the LLSOAs contained in Spinney Hills (Carstairs Index = 4.24, Created TPM_{10} = 0.009t/yr., Experienced TPM_{10} = 1.53t/yr.), Stoneygate (Carstairs Index = 0.22, Created TPM_{10} = 0.018t/yr., Experienced TPM_{10} = 1.17t/yr.), and Knighton (Carstairs Index = -4.70, Created TPM_{10} = 0.033t/yr., Experienced TPM_{10} = 0.92t/yr.) as one enters more peripheral locales. Similar trends may be observed in and around Humberstone, which also marks a staged transition in socioeconomic status matched by an integration of 'Indian' families into 'White British' communities.

On a final descriptive note, there would also appear to be eight fragmented boundaries of interest outlining the westerly ward of Western Park, which on average provide a BLV of 0.24 (Figure 7.4). This ward consists of 7 LLSOAs, typically housing persons of moderate affluent (Rank of -3.29) who create high levels of personal TPM_{10} (0.028t/yr.), yet residentially face environment burdens close to expected intensities (1.09t/yr.). What makes this unique from the previously explored emission contribution clusters, is that levels of 'White

Non-British' children are substantially raised ($\geq 7.07\%$) above that of Leicester's standard LLSOA profile (1.56%). Overall, one may conclude that the detected boundaries are of a comparative description to those presented via contour plots and within the spatial cluster analysis. Where it was revealed that affluent communities, representative of a successful integration of the ethnic majority with families of 'Indian' or (to a lesser extent) 'White Non-British' origins, were accountable for generating and passing on excessive levels of personal TPM₁₀ emissions to other communities.

Overlap statistics were subsequently employed to establish whether boundaries in community created transport emission levels (from private modes), spatially corresponded to geographic gradients in children's respiratory outcomes and social-environmental influences of interest outlined in Chapter 6. In examining the relationship between created TPM₁₀ emission boundaries and the entire range of children's respiratory outcomes (J00-99), it would appear that none of the distance based overlap metrics provide any information of particular interest (Table 7.2). Although a significant amount of boundary elements were shown to overlap, indicative of some interaction, with the two variables sharing an additional 6 Boundary Elements than what would be expected by chance ($O_5 P \uparrow = 0.03$). Of particular interest are the three common boundary locations separating an individual LLSOA of Beaumont Leys from communities contained within Abbey (Figures 6.1, 7.4). At these locations the emission and health gradients are reversed, in that when personal TPM₁₀ contributions are high J00-99 admissions are low (22.86 per 1,000), whereas the adjacent low contributing communities are found to experience a sharp elevation in the amount of J00-99 cases (57.05-65.04 per 1,000). These outputs build a strong case for the select existence of relatively localised shifts in environmental burdens, passed onto neighbouring communities by the frequent passage of vehicles en-route to work along a standard set of road links. Meanwhile the limited collection of mutually corresponding inner-city boundaries, would infer that these residents are unable to successfully shift their burdens onto neighbouring communities to the same extent.

For children's URTI's (J00-06), statistically significant overlap was once again identified to occur between boundaries in TPM₁₀ emissions (Table 7.2), with the two variables sharing 8 additional Boundary Elements than expected by chance ($O_5 P \uparrow \leq 0.01$). However, unlike the complete respiratory set, an average minimum distance of 248m from a boundary in TPM₁₀ emissions to a children's URT health front ($O_6 P \downarrow \leq 0.01$), was identified to be of a significantly smaller distance than expected under a null hypothesis of no spatial patterning. Furthermore, the mean distance from locations in either boundary to the nearest location in the opposing boundary were recognised to occur across a significantly small distance of 489m (O_{GH}

$P_{\downarrow}=0.02$), thus acknowledging the presence of significant boundary overlap between both factors.

As with the complete respiratory set, a common boundary was observed to separate a highly polluting community with low URT rates (10.80 per 1,000) in Beaumont Leys, from a LLSOA in Abbey, which emits fewer TPM_{10} emissions yet registers a higher quantity of children's URT cases (29.81 per 1,000). In contrast to the boundary analysis of the entire respiratory set, URT boundaries are detected to replicate such inverse relationships in personal TPM_{10} production and respiratory outcomes, across first-order communities throughout the city of Leicester. A more comprehensive inclusion of the community created TPM_{10} boundary set has also seen the emergence of significant distance based overlap metric outputs, clarifying ones observations in a more descriptive fashion and recognising that a global rather than locally restricted interaction is in operation (Table 7.2). Other key first-order interactions (Figures 6.1, 7.4) involve the separation of a highly polluting community (0.033t/yr.) with low URT rates (13.03 per 1,000) in Western Park, from a LLSOA in New Parks, which emits fewer TPM_{10} emissions (0.007t/yr.) whilst once more registering a higher quantity of children's URT cases (30.73 per 1,000). With respect to inner-city locales, these interactions are not so apparent due, as it would appear that such locales experience an accumulation of environmental burdens from multiple distant communities. Nevertheless, a clear common boundary was observed to separate a highly polluting inner-city LLSOA (0.031t/yr.) with modest URT rates (22.09 per 1,000) in Abbey, from a community in Spinney Hills, whom emit some of the lowest levels of TPM_{10} (0.005t/yr.) whilst recording substantial magnitudes of children's URT cases (38.97 per 1,000).

As with children's URTI cases, a global rather than locally restricted relationship would appear to link the community created TPM_{10} boundary set with the occurrences of some LRTI health fronts. Here, the indicated average minimum distance of 410m from a boundary in community created TPM_{10} emissions to a children's LRTI health front ($O_G P_{\downarrow}=0.04$), was identified to be of a significantly smaller distance than expected under a null hypothesis of no spatial patterning. The mean distance from locations in either boundary to the nearest location in the opposing boundary, also occurred across a significantly small distance of 541m ($O_{GH} P_{\downarrow}=0.05$), thus acknowledging the presence of globally significant boundary overlap. One may note that these metric based overlap statistics relating TPM_{10} creation to LRT health fronts were recorded to occur at a greater distance, than what was recorded in relation to URT complaints. However, this is of little surprise, as one may expect a smooth emission dispersion gradient along the route of travel from a person's place of residence, only occasionally amassing at junctions, and or when stretches of congestion occur. As understood previously,

severe LRT complaints required a persistently moderate-high level of residential contact, or exposure to particularly sharp episodes typically found around junctions during peak traffic flows. Meanwhile URT complaints, with their reduced severity, are often found to be the initial response towards the introduction of detrimental socio-environmental influences. Hence, the excessive sharing of boundaries and reduced proximity between created TPM₁₀ emissions and outer city URTI health fronts

As with the other collective and individually explored respiratory conditions, LRTI health fronts were not unanimously shown to correspond in a significant manner to community contributions in TPM₁₀ (O_H $P_{\downarrow} > 0.05$). This should be of little surprise when considering that spatially detached peripheral communities are thought to provide the majority of environmental burdens felt across inner-city locales; which contradict the lower magnitude health gradients, described above, occurring amongst first-order neighbours positioned away from central locales. Interestingly, the transitions in community created TPM₁₀ emissions would appear to explain the weaker health fronts not sufficiently captured by the residentially experienced TPM₁₀ dataset, due to the overshadowing recordings across inner-city locales.

Boundaries Overlapped (G,H) H= Created TPM₁₀ Emissions	Statistic	Observed (meters)	Expected (meters)	P↑	P↓
(G) J00-99 Admissions Per 1,000 Children	O_G	512	790 (±278)	0.86	0.14
	O_H	827	889 (±296)	0.54	0.45
	O_{GH}	669	840 (±235)	0.76	0.24
	O_S (count)	9	3 (±2)	0.03*	0.99
(G) J00-06 Admissions Per 1,000 Children	O_G	248	798 (±298)	0.99	0.01*
	O_H	729	1018 (±333)	0.83	0.17
	O_{GH}	489	908 (±256)	0.98	0.02*
	O_S (count)	11	3 (±2)	0.01*	0.99
(G) J20-22 Admissions Per 1,000 Children	O_G	410	802 (±300)	0.96	0.04*
	O_H	673	910 (±288)	0.79	0.21
	O_{GH}	541	856 (±235)	0.95	0.05*
	O_S (count)	8	4 (±2)	0.06	0.97

* P≤0.05

TABLE 7.2: Univariate boundary overlap analysis of community created transport emission levels (from private modes) and children’s respiratory outcomes

Following on from these outputs, overlap statistics were then implemented as a means of statistically summarising the spatial interactions and associations, between community levels of created TPM₁₀ emissions and individual socio-environmental factors of interest (Appendix F2). Whilst, spatial transitions in residentially experienced TPM₁₀ emission levels were themselves, identified to universally occur within close proximity to those from highly polluting

communities ($O_G=293m$, $P\downarrow\leq 0.01$), no such global associations were reciprocated by reverse positioned boundary tests ($O_{GH}, O_H P>0.05$). Despite this, it is highly likely that a range of more subtle residentially experienced TPM_{10} transitions are in existence near to those communities whom sufficiently contribute, as indicate by the inverted association with minor health gradients. Plausible reasoning for their concealment shows this as a consequence of the steep localised pollutant gradients occurring across central locales, which have overshadowed any minor transitions in pollutant exposures across other parts of the city.

In accordance to the cluster analysis and contour plots, the overlap statistics identified boundaries in deprivation as sharing a significant number of elements with those marking community created TPM_{10} outputs ($O_S P\uparrow=0.05$, Appendix F2). Furthermore, zones of rapid change in socioeconomic status were designated to occur within a proximal 305m from a polluting community boundary ($O_G P\downarrow\leq 0.01$), with an average distance of 375m also separating opposing boundary locations ($O_{GH} P\downarrow\leq 0.01$). In combining such information with the conclusions of the preceding chapters, one may understand that the Leicester's major respiratory health fronts, typically located within the inner-city, are considerably defined by the contributions of pollutants emitted from the motor-vehicle.

Furthermore, the spatial peaks in residentially experience levels of TPM_{10} are shown to be produced primarily by sizeable pockets of affluent communities, whom shift their burdens onto deprived communities located nearby; but far enough away to not feel the full force of these affects themselves. Furthermore, those residents residing along the fringes of this affluent zone typically provide only moderate burdens to those nearby inner-city communities, whilst those residing within the distant and most affluent communities (at the heart of this pocket) are found to emit the highest amounts of TPM_{10} and experience the smallest impacts. It would therefore appear that health, social and environmental gradients truly operate in an aligned manner within an intra-urban setting.

In exploring whether lifestyle choices determine the thought process behind a communities environmental contribution (Appendix F2), one may observe that spatial transitions in relation to smoking prevalence comprehensively occur within close proximity to those boundaries in community created levels of TPM_{10} from personal transportation modes ($O_G=461m$, $P\downarrow=0.05$). In addition, the boundary analysis outputs identify these two factors as sharing 9 additional boundary elements than expected by chance ($O_S P\uparrow=0.04$), further cementing such ties. As highlighted by the contour plots (Figure 7.3), boundaries typically distinguish low levels off smoking within those communities whom residentially experience low levels of TPM_{10} emissions, whilst heavily contributing towards the cities wider TPM_{10} emissions problems. Although these communities are self-conscious in mitigating the level of

risk placed on their children (i.e. passive smoking), they appear unaware or simply uninterested in how they shift their transport burdens onto others.

In terms of boundaries in obesity, one may once again observe that those most obese emit the fewest pollutant from personal transportation measures (Figures 6.2, 7.4). In particular, the highly polluting affluent pocket inclusive of Knighton and sections of Castle are cordoned off by a fragmented expanse of boundaries in obesity. Statistically such observations are confirmed by universally close proximity of obesity boundaries to those outlining community created TPM₁₀ emissions ($O_G = 332m$, $P_{\downarrow} \leq 0.01$), in addition to a common positioning of an additional 10 boundaries than expected by chance ($O_S P_{\uparrow} = 0.04$) (Appendix F2). In confliction to these observations, transitions in community created emissions were recorded to occur far away from obesity boundaries ($O_H = 1294m$, $P_{\uparrow} = 0.04$). However, upon inspecting the spatial occurrences of both boundary sets, such disassociations have arose not through a fundamental disagreement of the previous statements. Simply put, boundary formation for obesity was less sensitive to its moderate transitions across northern Leicester, which would have coincided boundaries in community created emissions (Figures 6.2, 7.4).

Through investigating the spatial influence of ethnic profiles (Appendix F2), it was uncovered that spatial transitions in 'White British' residency were located in significantly distant locations of the city, when compared to boundaries in levels of community created TPM₁₀ ($O_G = 1292m$, $P_{\uparrow} = 0.04$), and vice versa ($O_H = 1071m$, $P_{\uparrow} = 0.04$). To a certain extent this confirms that the ethnic minority is not unanimously responsible for the majority of TPM₁₀ burdens, as if this was the case, one would expect a mutual emergence of high polluting communities with increased residency. Rather the polluting communities, typically well within this 'White British' domain, have been shown to represent affluent communities that are positively integrating with 'Indian' residents. Similarly, boundaries in 'Indian' residency were located in significantly distant locations of the city to transitions in community created TPM₁₀ ($O_G = 1195m$, $P_{\uparrow} = 0.05$), and vice versa ($O_H = 1204m$, $P_{\uparrow} = 0.03$). This is because 'Indian' residents are primarily located within the eastern sector of the city, where personal TPM₁₀ contributions are low.

In contrast, spatial transitions in 'White Non-British' residency were found to be universally positioned within reach of polluting communities ($O_G = 340m$, $P_{\downarrow} \leq 0.01$), in addition to sharing an additional five boundary elements that expected by chance ($O_S P_{\uparrow} = 0.05$). As previously discussed, a substantial section of 'White Non-British' residents are found to peak around the polluting and affluent communities of Western Park; replicating the successful integration of some 'Indian' residents with those of the ethnic majority. On a final note of interest, one should note that communities whom emit substantial proportions of TPM₁₀ are

typically located within distant communities to those somewhat exclusively housing children of ‘Afro-Caribbean’ ethnicity ($O_H=1690m$, $P\uparrow=0.04$). This would coincide with the earlier concept, which understood that whilst some environmental burdens of polluting (and usually peripheral) communities are unfairly passed onto their immediate neighbours, the most unjust burdens tend to be drawn to, and accumulate around central locales housing those socially vulnerable communities.

In pursuing the investigation of recurrent themes within this research project, boundary overlap procedures were subsequently applied to explore the connections between the ‘double-burden’ of deprivation and TPM_{10} exposure, with community created transport emission levels (from private modes). Table 7.3, informs the reader that a strong universal association exists between both factors, with polluting communities typically shifting their environmental burdens onto communities separated by a small distance threshold ($O_G=276m$, $P\downarrow\leq 0.01$). This buffer zone of approximately 300m between a common creation source and an accumulation of transport emissions, is further strengthened by outputs quantifying the reverse ($O_H=376m$, $P\downarrow=0.02$) and collective boundary ($OGH=326m$, $P\downarrow=0.01$) relations.

Boundaries Overlapped (G,H) H= Created TPM_{10} Emissions	Statistic	Observed (meters)	Expected (meters)	P\uparrow	P\downarrow
(G) Carstairs Index & Experienced TPM_{10} Emissions	O_G	276	802 (± 266)	0.99	0.01*
	O_H	376	830 (± 278)	0.98	0.02*
	O_{GH}	326	816 (± 223)	1.00	0.00*
	O_S (count)	15	4 (± 2)	0.00*	1.00

* $P\leq 0.05$

TABLE 7.3: Bivariate boundary overlap analysis of community created transport emission levels (from private modes) with deprivation and residential TPM_{10} exposures

Through the inclusion of a third social or lifestyle factor to this ‘double burden’ relationship (use of the trivariate boundaries presented within Chapter 6), one should note that transitions in ‘White British’, ‘White Non-British’ and ‘Indian’ children substantially weaken the uncovered bonds of the double burden (Appendix F3); further signalling their disassociation of experiencing a combination of these undesirable impacts. Whilst, the inclusion of obesity prevalence displaced the existence of a universal connection between boundaries in emission creation levels and transitions of the ‘double burden’ (O_H $P\downarrow > 0.05$); those of the trivariate set were found to comprehensively reside within a reduced distance to these polluting communities ($O_G=156m$, $P\downarrow\leq 0.01$). This reduction in distance has occurred through a strengthening of markers around those most affluent and polluting areas (i.e. Knighton and Western Park), whom also exhibit low levels of obesity. In general terms, those most obese

were previously identified as emitting the fewest pollutant from personal transportation measures, typically residing within the most deprived communities. However, low levels of obesity are also recorded across inner-city communities, where the 'double burden' is most rampant. Such complex undertones offer reasoning for the lack relationship in the opposing direction.

On a concluding note, those trivariate boundaries including either the 'Other South Asian' or 'Afro-Caribbean' ethnic minority group as the final component, barely deviate from the spatially strong affiliations recorded between magnitudes of emission production and the 'double burden' (Table 7.3, Appendix F3). Out of these two ethnic minorities, persons of 'Afro-Caribbean' origins would appear to feel the highest magnitude of the environmental aspects of this double burden, on average residing 25m closer to boundaries in emission creation than 'Other South Asian' residents ($O_G P \downarrow < 0.05$). Such observations are in-line with the conclusions drawn from Chapter 6.

7.5. A FAIR DISTRIBUTION OF POLLUTER RESPONSIBILITIES

From the preceding sections of this chapter, it has become apparent that several of the most affluent and environmentally least burdened of Leicester's communities, are responsible for the release of large quantities of pollutants from personal modes of transportation. In contrast, those socially vulnerable communities, whilst occasionally contributing to their own environmental degradation, consistently experience a disproportionate level of environmental burdens. In conclusion, one may observe that an environmentally unjust situation currently prevails across the city of Leicester. However, simply pointing the finger at those affluent communities does not allow for a fair assessment of the current state of environmental affairs, without accounting for a number of other circumstances. For instance, the level of employment will determine some variations in pollutant contributions purely through an increase in required trips. Under these circumstances, it would be socially unfair to place a raised environmental accountability onto these communities, as in many ways these inhabitants are already paying societal contributions (collected via taxation), which benefit those in vulnerable situations. Rather it is of interest to place environmental accountability on excessive travel distances and the use of certain transportation measures, which should be viewed after existing societal contributions are accounted for.

To explore whether a fair distribution of polluter responsibilities exists, this section initially presents a series of contour plots, describing how a communities socio-environmental

characteristics may influence their overall mobility and uptake of particular transportation modes, after adjusting for levels of employment. All of this was conducted in fashion, which provides a greater understanding of the favoured modes of transportation used by Leicester's communities across different spectrums of the previously discussed 'double burden' of interest. Contour plots of employment levels appear to follow anticipated distributions (Appendix F4), with those socially and environmentally favourable communities containing the largest economically active populations (>70%). Therefore, it is of little surprise, that this lower left quadrant contributes a high proportion of TPM₁₀ emissions levels experienced by residents across the wider urban area of Leicester (Appendix F4). In contrast, the top right quadrant identified the communities represented here to by enlarge only display employment levels of <40% amongst persons aged 16-74 years (a figure well below the citywide average of 55.99%). Typically the communities positioned here on the socio-environmental spectrum are observed to emit low levels of TPM₁₀ (<0.015t/yr.), yet are burdened by high levels of environmental burdens. Upon examining those deprived (Rank >0) and affluent (Rank <0) communities as two individual components, one may observe that those communities with higher employment rates tend to contribute high levels of TPM₁₀, whilst experiencing reduced levels of residential exposures.

From previous plots one may recall a small pocket of communities contained within the upper right quadrant, indicating where the impact of the 'double-burden' was most felt (Appendix F1). Children housed here had severely reduced respiratory functions (≥80 J00-99 admissions per 1,000), most likely induced by a combination of deprivation levels 5 ranks above the citywide average and residential TPM₁₀ exposures <2.0t/yr. Unlike surrounding communities, employment levels here are found to persist around 40-45%, rather than falling below 40% (Appendix F4). Consequently, these communities would appear to experience increased levels of mobility and favour personal forms of transportation (Figure 7.5), which are thought to be of a poor condition due to their financial constraints. The moderate contributions from this fleet are likely the reason behind driving the respiratory health effects of these communities from high to severe. Therefore the implementation of a small but targeted strategy providing assistance with vehicle maintenance and or encouraging alternative transportation modes could have a noticeable impact.

In fairly examining the polluter responsibilities (Figure 7.5), one may observe that employed persons from affluent communities are likely to commute greater distances to work compared to their socially disadvantaged counterparts. In general, those deemed of affluence travelled >15km per day on their return commute to work, whereas the workforce from deprived communities only travelled reduced 6-15km. This follows the concept that districts of

work are located close to deprived communities, and as such affluent communities have further to travel, which encourages the use of more environmentally unfriendly modes of transportation. In addition, a common district of destination is likely to create an accumulation of pollutants, which may be further added to by the shorter trips of those less privileged residents living within the nearby vicinity.

In viewing the graphical representation of distances travelled per employed person (PEP) via their preferred mode of transportation, one may observe a very low use of 'green' transportation modes (on foot, bicycle or carpool) by those communities in the bottom left quadrant of the 'double burden' relationship (Figure 7.5). Here, typically only 4-16% of the total distance travelled PEP was completed via a 'green' form of transport. It would appear that 'green' transport use dramatically increases with respect to residentially experienced levels of TPM_{10} , regardless of social status. In-fact once a residential TPM_{10} level of $\geq 2.5t/yr.$ is reached, $>28\%$ of residents are found to travel by a 'green' mode of transport. Although one should note that levels also increase with deprivation, though not to be of personal choice but out of necessity.

It has already been acknowledged that an increase in physical exercise across polluted neighbourhoods, whilst keeping additional vehicles off the road, may not actually decrease respiratory cases due to increased periods of exposure. Nevertheless, one may observe that communities feeling the brunt of the 'double burden' share broadly similar socio-environmental attributes, yet have a much lower use of 'green' transport (16-28%). Here it has been shown that an increased use of poor vehicle stock, increased levels of respiratory admissions from high to severe (Figure 7.5). Therefore, the encouragement of green modes may be viewed as a means of mitigation (akin to those communities with comparable attributes) rather than an out and out solution. Even if such actions were in operation, further measures are still required to limit external communities from shifting their share of pollutants onto these inhabitants.

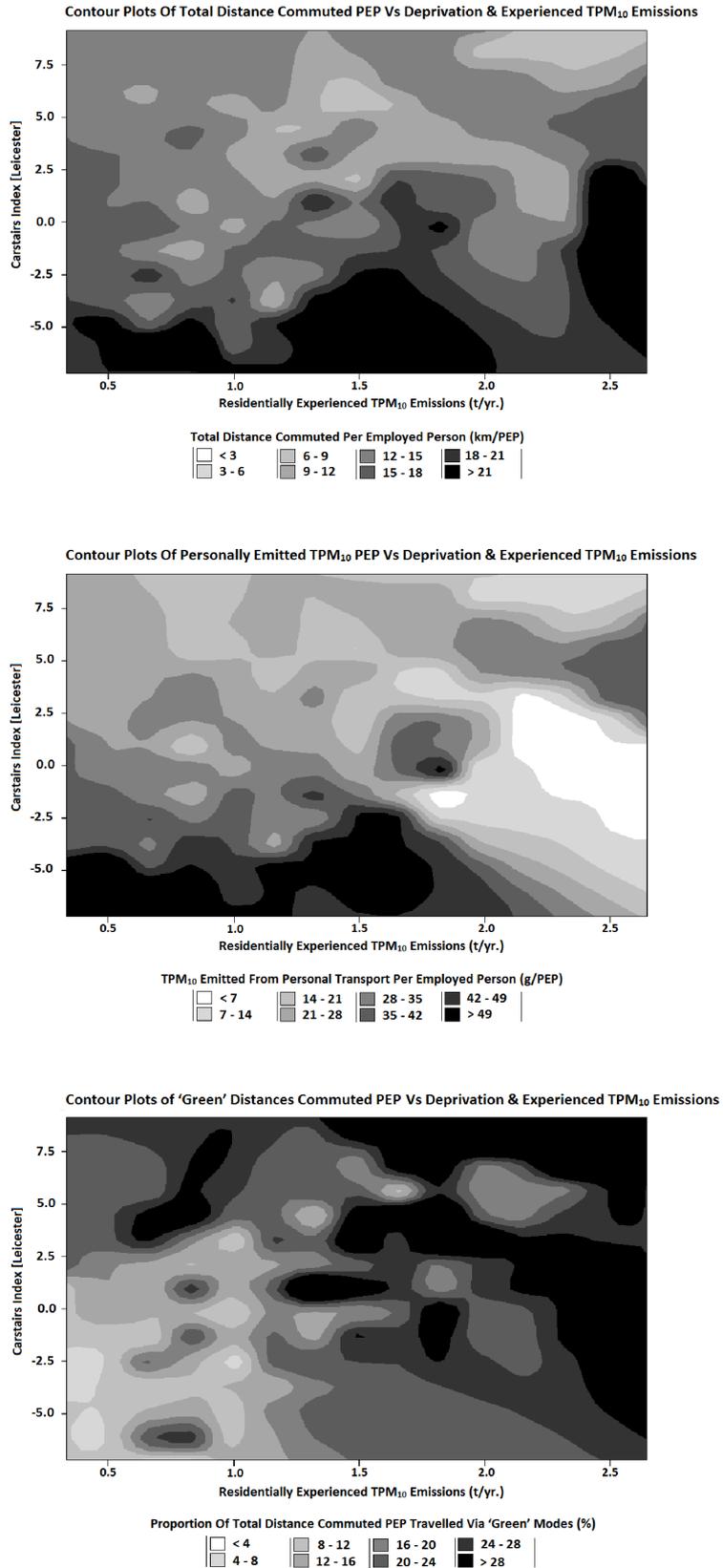


FIGURE 7.5: Contour plots, exploring Leicester’s ‘Double Burden’ in relation to levels of personal emission creation and commute distances completed by a range of transportation measures, after adjusting for the number of persons employed within each community

Upon inspecting the contour plot of communally emitted TPM₁₀ PEP (from personal transportation modes), one should observe that the broad structures contained within the graphical representation excluding workforce counts, remain largely unchanged (Appendix F4, Figure 7.5). Critically, the lower left quadrant still contains communities that excessively contribute to the cities TPM₁₀ problem from personal modes of transportation, and should thus remain the focus of the solution. In truth, the only major change to be observed between the two plots centre's around these highly polluting communities, which now appear to extend into those extremely affluent communities experiencing moderate environmental burdens (approximately 1.5t/yr.) compared to the expected LLSOA TPM₁₀ level (1.04t/yr.). Within this zone of extension, communities are found to move from the third highest polluting interval of the contour plot (when not accounting for population counts) to that of the most polluting category. As with other affluent areas, elevated emission contributions are not thought to be caused by vehicle condition, but rather by excessive commute distances.

As previously discussed, those communities within this extension typically house ≥80% children of 'White British' origins, and as such are less ethnic integrated than most polluting areas. However, unlike other highly polluting affluent communities, these communities, to a certain respect, are already paying for aspects of their environmental contributions through moderate residential exposures. It is from this information, that the ethnic majority group could not be directly blamed for the city's environmental burdens, with class acting as the ruling factor. Meanwhile, the top right quadrant still contained deprived inhabitants whom contributed little in proportion to their experienced environmental burdens. However, taking into account personal travel emission outputs per worker did clarify and considerably extend the volume of socially and environmentally deprived communities, contributing towards the environmental burden confronting them. Whilst pollution contributions remained at the fourth highest interval across both contour schemes, the area recording those severest respiratory conditions was now found to be fully incorporate by the pollution plots.

In addition, little change was observed across the lower right quadrant, which theoretically characterises an affluent populace exposed to a sizeable magnitude of environmental burdens (as only 4 LLSOAs loosely abide to such trends). In this form the quadrant is essentially a hypothetical reactionary scenario of affluent communities, if they were not to pass on their burdens to those external and often deprived locales (i.e. a just situation). Under this scenario communities are observed to personally emit low levels of TPM₁₀, despite the fact that a large subset of this distribution was shown to commute considerable distances per worker (>21km/PEP), on a daily basis. However, one should note that this subset corresponds to a peak in the uptake of green transportation modes (>28%),

showing that it is perhaps possible to reduce personal emission contributions across communities whom tend to commute across large distances. This approval of 'green' transport, would likely occurred in recognition of their residential exposure to high pollutant levels (or the visual impact of vehicles), thus raising the environmental awareness of these inhabitants.

Whilst larger trips remain undertaken by public transportation, as approximately two thirds of the distance commuted is still eaten up by mechanical measures, it is likely that 'green' modes are favoured for those frequent, shorter trips. However, this increased use of public transport, raises questions relating back to those high residential exposure levels, with larger vehicles travelling close to residential areas on a more frequent basis, if of a certain age are likely to cause more harm than good. Thus highlighting the delicate balance faced by planners in aiming to providing a high quantity of public transportation whilst maintaining the quality of their fleet. It should also be recalled, that suburban communities around the expected socioeconomic baseline are most likely to travel via bus, yet they were found to reside within low pollution zones; likely caused by the open nature of such areas. It would be likely that these hypothetical affluent communities are also to be found in suburban areas, thus avoiding an intense accumulation of pollutants through the urban canyon effect. Therefore under such a scenario environmental conditions at their place of residence would likely improve, however their movements may still impact others dependent on the maintenance of the public transport fleet.

After summarising a range of practical as well as theoretical community mobility and personal emission distribution structures in relation to socio-environmental influences (in a manner deemed 'fair' to societal contributions), a series of Univariate Local Moran's I statistics were subsequently applied to locate where the key uptake of individual forms of transportation has occurred (Appendix's F5, F6). Through taking into account a range of transport measures, one should also understand (in a rather crude manner) the extent to which communities with a high uptake of personal transport modes contribute towards the overall environmental burden. For instance, these same communities may also have a high use of public transport, identifying themselves as inconveniencing external communities through a spectrum of mechanical measures. If this is indeed the case, then the extent to which environmental disparities exist within Leicester, occur across a far wider range than explored here. Another extreme scenario is that those poorer communities actually have a high use of public transport, and in a certain respect are involved at a higher level in fabricating their own environmental problems. If this scenario is found to be accurate, it is not truly the fault of

vulnerable communities, with the real payment lying with the supplier of such services, for the provision of an environmentally inadequate fleet to the consumer.

Collectively across Leicester, LLSOAs were found to on average exhibit employment levels of 55.99% for persons aged 16-74 years. The proportion of an areas workforce typically commuting to work via Car/Van, Bus or 'Green' modes of transport were respectively recorded at 44.41%, 15.63% and 28.09% [Green: Walking=16.34%, Cycling =4.03%, Carpool=7.72%]. Spatial plots of favoured mobility forms reveal personal transport use to be particularly high around those affluent areas, which were found to emit the highest quantities of personally created transport emission levels (Appendix F5). This statement confirms that the majority of detrimental emission outputs are not a result of vehicle age, but rather vehicle use. In-fact levels of Car use for commutes to and from work were recorded to exist at 57.99% in Knighton and 58.68% within Humberstone, both of which were characterised by the respectively high employment levels of 63.69% and 64.39%. In stark contrast, the uptake of public transport would appear to be particularly low across the LLSOA communities of Knighton (10.91%), as confirmed via cluster analysis outputs (Appendix F5). Whereas, residents within Humberstone use of public transportation (14.75%) appeared low yet only just beneath the citywide rate, and as such remained undetected by the cluster analysis outputs to be of particular significance. This would suggest that an element of class may be involved in relation to public transport use, an issue which would be of interest for urban planners to address.

Meanwhile, personal transport levels were recorded to exist at a low level around central locales, with the ward of Spinney Hills recording Car/Van use at only 36.79%. In addition, Bus use was also observed at a low level (13.00%), indicating that this community has a limited use of mechanical forms of transportation in their daily commutes (Appendix F5). However, across these areas residentially experienced TPM_{10} is typically recorded as high (1.54t/yr.), in line with the communities deprived nature as recorded by the Carstairs Index (4.20) and low employment levels (43.67%). As many of the workers are likely to have recently re-entered the job market or reside in low skilled placements, it would appear that 'Green' transport measures (38.43%) are favoured most likely out of necessity rather than choice. As confirmed by 28.59% of the workforce commuting to work on foot, which is deemed the most economically sensible measure. In contrast, the overall uptake of 'Green' measures is considerably reduced amongst those affluent communities contained within the wards of Knighton (20.23%) and Humberstone (17.18%).

If one is to refer back to the Bivariate Moran's I analysis comparing a locations created to those experienced TPM_{10} emissions of its neighbouring communities (Figure 7.2), one may recall that select inner city communities were accountable for a proportion of their

environmental burdens. Across these 6 High-High inner-city LLSOAs average levels of car use were recorded at 37.30%, with such residents characterised to experience mild levels of deprivation (0.19), high levels of TPM₁₀ (1.82t/yr.) and high J00-99 hospitalisations amongst children (65.19 per 1,000). In contrast, the 16 Low-High inner-city LLSOAs who pollute little, have a slightly lower uptake of car use (32.26%) perhaps as a result of their increased deprivation (3.78). Whilst remaining geographically positioned on the trail of a substantial proportion of trips to central business districts, their own travel actions may be observed to ease those persistently high levels of experienced TPM₁₀ (1.75t/yr.) and J00-99 hospitalisation rates (51.94 per 1,000). One should note that levels of 'Green' transport measures are popular across both High-High (40.27%) and Low-High (43.07%) inner-city communities, whereas the uptake of public transportation for both remains respectively low at 12.76% and 15.02%; thus highlighting the important of community contributions from personal transportation modes across vulnerable communities.

However, levels of employment were shown to be considerably elevated within High-High (55.34%) compared to those Low-High (41.29%) inner city communities, in part explaining the dissimilarities between their typical LLSOA recorded total daily commute distances (H-H = 12,609km; H-L = 6,184km), of which around half was completed via Car by both community types (H-H = 64.17%; H-L = 55.26%). Hot-Hot communities are thus shown to travel around double the distance by car to that of their inner-city counterparts, which seems excessive when allowing for shifts in employment levels. Upon factoring in levels of employment, one may still observe drastic differences between the distances commuted in total (H-H = 18.11km/PEP; H-L = 13.26km/PEP) and individually by Car (H-H = 11.52km/PEP; H-L = 7.31km/PEP). In-fact residents from High-High inner-city locales were even deemed to commute over a greater distance in total and by Car than those of the city average, which were respectively documented at 14.79km/PEP and 9.95km/PEP.

These findings tie in with two prior thought processes, the first of which appears to confirm that the travel actions of certain inner-city communities pushes them over a health threshold, resulting in severe rather than high respiratory burdens. Such health burdens are thought to materialise through an increase in the use and distance travelled by an inexpensive and thus poorer quality personal transport fleet, with reduced emission standards. Secondly, increased mobility appears to have a role in improving social status (even across the lower end of the spectrum as documented here), with private modes allowing for more direct, flexible and perhaps rapid movements across the city, thus opening up new job opportunities but at the cost of environmental outcomes. However, whilst affluent communities should be viewed as having a moral obligation for mitigating the effects of their transport emissions, is it correct

to place such obligations on deprived yet socially rising communities (whom in the grander scale contribute relatively little)? In some aspects this is similar to the dilemma faced at a global scale, involving the moral obligations of developed nations to fiscally and thus environmentally assist (e.g. carbon production, industrial operations) rather than prevent the social climb of other nations, whom are presently experiencing their own industrial revolutions. A balance must thus be found between distinct stages of development and environmental responsibility.

Maintaining focus on Leicester's bivariate TPM₁₀ emissions inner-city clusters (Figure 7.2), one should note a universally high participation in 'Green' forms of transportation exists (H-H = 4.47km/PEP; H-L = 3.78km/PEP) compared to the typical rate of uptake (2.73km/PEP). Here, these hotspot communities even appear greener than their neighbours do, but any increased levels of physical activity across such highly polluted residential areas may also inadvertently diminish respiratory functionality. In exploring the uptake of other mechanical forms, one may observe that distance commuted by bus (H-H = 1.91km/PEP; H-L = 2.05km/PEP) hovers between low to near that of the expected standardised citywide rate (1.94km/PEP).

Such a prosaic use of public services is rather surprising, considering that the central bus depot and thus the core of the transport network is located inside of the inner-city ring road, at the heart of these communities. Consequently these locales would experience an accumulation of public transport pollutants (in addition to the high volumes of personal transport emissions), yet at present such services are not extensively used by local inhabitants, perhaps caused by a tailoring of services for the greater influx rather than outflux of passenger trips during key periods of the day. Furthermore, incoming routes would tend to start from residential and terminate in employment zones, and thus the reverse route would be of limited use for those inner-city residents looking for a direct conduit to more distant commercial/industrial premises. Perhaps further encouragement and or a greater set of public services is required to remove the necessity of some inner-city communities from adding additional vehicles to the roads, as the framework for public services is already in existence here.

Upon exploring the citywide uptake of Buses (Appendix F5), which act as the major and somewhat exclusive public transport service operating across Leicester, one may observe that a main cluster of high use exists across 5 LLSOAs around the cities southern peripheral ward of Eyres Monsell. These communities were characterised as housing predominantly 'White British' children (87.36%), whom experience low TPM₁₀ levels (0.96t/yr.), and moderate intensities of deprivation as indicated by Carstairs Index rank (1.90) and employment levels

(48.66%). Here the uptake of Bus services amongst those employed was on average recorded at 21.70% (+6.07% on citywide estimates), while the uptake of Car/Van (42.81%) and 'Green' (26.22%) transportation modes were close to the city average. Their dependency on mechanical modes may be further observed by particularly low rates of foot travel (11.95%), recorded 4.39% beneath citywide uptake estimates. This lack of physical transportation is perhaps further represented by the communities elevated levels of obesity (29.82%) and smoking (45.14%) prevalence's during adulthood.

To add additional context, at present these LLSOAs travel 8.89km/PEP by bus, 7.06km/PEP by car and 1.67km/PEP via 'Green' modes of transport, compared to their respective city average levels of 1.94km/PEP, 9.95km/PEP and 2.73km/PEP. Furthermore, one should note that these mechanically reliant LLSOA communities typically commute a rather modest total daily distance (6,704km), which upon factoring in levels of employment equates to 13.22km/PEP, a figure 1.57km/PEP beneath the city average. This questions the need for mechanical transportation forms by such communities, considering the reduced commute distances and reduced physical health of some inhabitants.

One should recall that these same communities represent a set of suburban inhabitants whom appeared on the verge of choosing to travel via Car or Bus, and when personal transportation was favoured by nearby communities emission rates appeared particularly high (as financial restrictions were thought to result in poor vehicle stock). Therefore a balance needs to be found between encouraging physical transportation modes without encouraging a shift from public to personal transportation measures. Interestingly, the frequent passage of large public vehicles throughout these suburban locations has not caused a substantial rise in TPM_{10} emissions, in a way confirming that it is right for this study to place focus upon those contributions from personal transportation modes. However, a common centrally focused bus destination, questions whether a larger and more important impact occurs across inner city communities, which remain largely unexplored by this study.

The next stage of this section follows on from the above findings, cartographically displaying a spatially complete range of personal, public, 'green' and total distances travelled PEP, in order to allow for a fairly critique of transportation measures based upon societal contributions (Figure 7.6, Appendix F7). For distributions of Car/Van commute distances PEP, 13 highly polluting hotspots were recorded to be of interest under this balanced analysis. In particular, a cluster comprising of 9 LLSOAs may be observed to remain focused around Knighton, reconfirming the excessive personal environmental contributions of these residents. Although after accounting for workforce levels a number of eastern LLSOA communities were noted to disappear under this fairer assessment of emissions from personal transportation

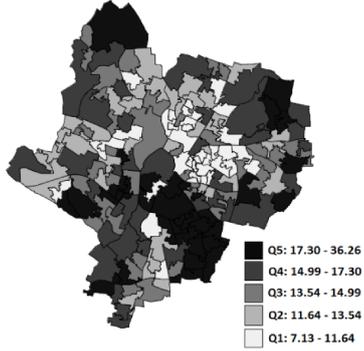
modes. Nevertheless, some aspects of Humberstone were still located to be of significance as was a minor localisation around Beaumont Leys.

Characteristically these 13 hotspot communities travelled a total daily distance of 22.27km/PEP, of which 16.11km was undertaken via Car/Van, 2.08km by Bus and 3.92km by 'Green' modes. Such a total travel distance equates to almost double the distance travelled by a typical LLSOA community, and whilst distances of personal transportation rose proportionately, those completed via Bus and 'Green' modes were not scaled up to an acceptable level. As previously established these communities are of an affluent nature (Rank of -4.42), whom residentially experience very few TPM₁₀ emissions (0.82t/yr.) yet contribute quite heavily from personal modes of transportation (0.035t/yr.); in-fact 8 out of the 13LLSOAs are contained within the upper 10% most privately polluting private communities of the city.

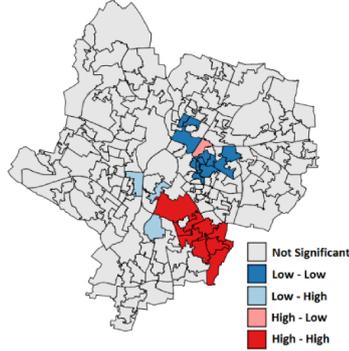
Meanwhile, 11 LLSOAs recording low levels of Car/Van travel PEP were found to remain around eastern areas of the inner and outer city centre, under this fairer assessment (Figure 7.6). However a number of focal inner-city communities had disappeared, indicating that they were starting to favour personal transport use. In these disappearing cases, emission outputs were now representative of citywide values (despite vehicle uptakes generally remaining moderate-low), as increased mobility sometimes allowed for more distant employment opportunities, which have become more noticeable by prevailing financial restrictions limiting residents to poorer quality vehicle stock. Characteristically the 11 cold-spot communities of significance, travelled a total daily distance of 10.23km/PEP, of which 5.86km was undertaken via Car/Van, 1.71km by Bus and 2.46km by 'Green' modes.

From the comparison of cold and hot-spots it would appear that Car/Van use is heavily linked towards an excessive use of travel, whereas the uptake of other forms of mechanical transport do not appear linked to increased mobility. Cartographic plots further confirm this, showing that the magnitude of uptake and distance travelled via Bus is evenly distributed throughout the city (Figure 7.6). On a final note relating to Car/Van travel, one may observe that a high outlier exists within the aforementioned central cold-spot, however when compared to values across the wider city travel is not deemed excessive (+0.09km/PEP above the expected).

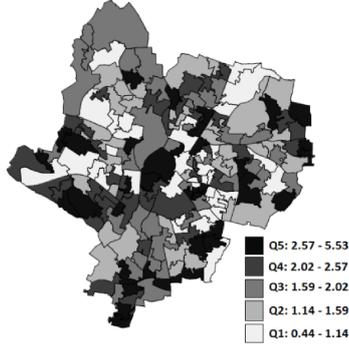
Total Distance Commuted Per Employed Person (km/PEP)



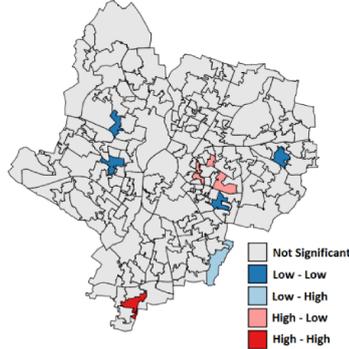
Univariate Local Moran's I: Total Distance Commuted (Km/PEP) [R² = 0.40]



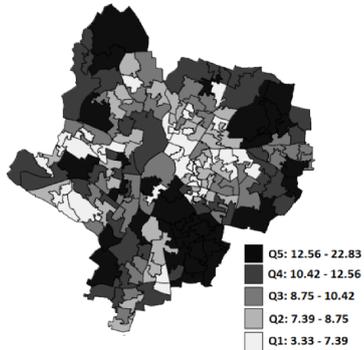
Distance Commuted Via Public Transport Per Employed Person (km/PEP)



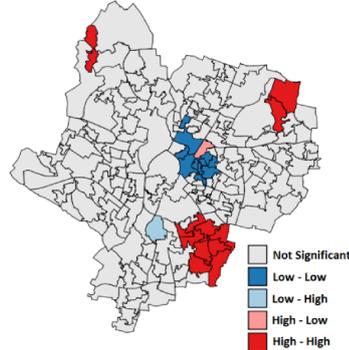
Univariate Local Moran's I: Public Transport Commute (km/PEP) [R² = 0.05]



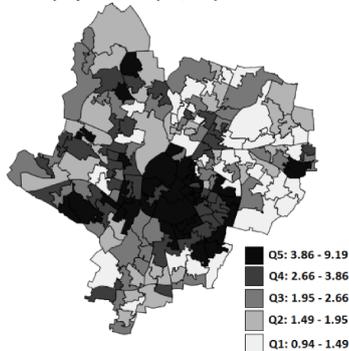
Distance Commuted Via Personal Transport Per Employed Person (km/PEP)



Univariate Local Moran's I: Personal Transport Commute (km/PEP) [R² = 0.50]



Distance Commuted Via 'Green' Modes Per Employed Person (km/PEP)



Univariate Local Moran's I: 'Green' Mode Commute (km/PEP) [R² = 0.28]

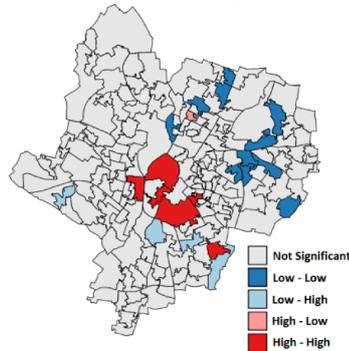


FIGURE 7.6: Local Moran's I cluster and outlier analysis, describing the spatial structures of those commute distances travelled per employed person (km/PEP) in total and by individual transportation forms

Interestingly for distributions of commute distances PEP completed via Bus, a single Low-High outlier is observed to exist along the most peripheral edge of Knighton (Figure 7.6). Here, created TPM₁₀ emissions are recorded at 0.040t/yr. (upper 5% polluters) whereas residentially experienced levels exist at an exceptionally low 0.78t/yr. Furthermore, commute distances undertaken via Bus (0.43km/PEP) and 'Green' (1.25km/PEP) modes are deemed somewhat insignificant. Meanwhile its five neighbouring communities are also typically associated with high levels of emission creation (0.036t/yr.), while also experiencing low residential emission (0.86t/yr.). Importantly, one may note that these communities are also more open to transportation via Bus (3.65km/PEP) and 'Green' (5.58km/PEP) modes. It would therefore appear that the 10% increase in personally created transported emissions of this outlier has directly relates to a decline in public and 'Green' transportation modes. Furthermore, environmental disparities are shown to even exist between affluent communities, albeit at a minor level. The focus of any measures should remain targeting at those poorest and socially vulnerable communities.

Distances commuted via 'Green' transportation measures PEP, were observed to exist at distinctly low levels across eastern Leicester (Figure 7.6). In particular, residents from the ward of Humberstone were recorded to only commute 1.54km/PEP via such modes, which is an insignificant fraction of their overall commute distance of 17.55km/PEP. Whilst foot travel is not a feasibly possibility for travelling over these large commute distances, cycling offers some potential. However a more reasonable form of travel in communities travelling across large distances where personal vehicles are favoured over public transport, involves carpooling. It would be advised that future carpooling schemes target those most affluent communities, whom current that have a low uptake of public services perhaps in part founded by negative stigmatisms. Here carpooling could have a reasonable effect on the number of vehicles entering the road, if effectively implemented.

At present carpooling only accounts for 7.72% of trips undertaken by a typical LLSOA within Leicester, therefore there is significant room for improvements. In constructing a ratio of the travel distances undertaken via Car/Van (km/PEP) Vs. Carpooling (km/PEP), it is possible to obtain a standardised ratio of implementation and to view the possible gains to be obtained. Across Leicester, a typical LLSOA was observed to record a distance of 8.33km/PEP travelled via Car/Van for each 1km/PEP completed via carpooling. Interestingly this ratio was found to increase across those communities with the greatest level of personal transportation use, as shown by LLSOA communities exclusively contained within Knighton (12.94km: 1km) and Humberstone (15.76km: 1km). In contrast, levels of carpooling increased across those

most deprived communities conducting the fewest and smallest trips via Car/Van, as observed by Stoneygate's ratio of 6.64km: 1km.

In conclusion, public transport appears evenly distributed across Leicester at a moderate-low level of uptake, with no evidence existing for assigning an excessive use of such services to any particular community. Furthermore, where some suburban communities favour public transport, road-transport emissions remain low. As such, there is no direct evidence that the frequency of Buses passing near to residential areas act as a key driving force behind a city's environmental issues. However one should note that such areas represent open environments, and that perhaps a different set of conclusions may be drawn from confined inner city areas, especially where accumulation of terminating services occur. Here, public transportation contributions are thought to be exclusively high, combining with those of personal modes whom are also thought to heavily contribute here, resulting in an environmental effect of volatile proportions.

Whilst the majority of travel distance remains completed via personal transport, communities favouring this mode of transportation, characteristically of an affluent nature, share high disassociations with an uptake of public travel services. Affluent communities therefore do not inconveniencing external communities through a spectrum of mechanical measures. Yet, those poorest inner-city communities are not observed to have a raised level of public transport use, and are thus not involved in fabricating their own environmental problems from other unexplored mechanical modes. However, a low uptake of public services here is perhaps an issue of concern, as some inner-city communities have started to favour the use of inexpensive and often poor private vehicles, with this newfound mobility seemingly resulting in increased social standing, but at the cost of environmental attributes. To tackle such issues, perhaps these central communities require further incentive to use public modes, rather than adding vehicles onto the road network where services already exist. In addition, the likely negative stigma of public services amongst affluent communities also requires further investigating, yet a more likely and immediate response for mitigating their environmental contributions involves the use of carpooling. Here, the convenience and luxury of personal transportation is likely to be favoured, and could mark a substantial reduction in the volume of vehicles which enter inner-city areas.

7.6. LESSONS FROM EXTERNAL IMPLEMENTATIONS OF ZONE BASED POLICY

Affluent communities represent those most mobile residents within the City of Leicester. Of concern are the high levels of disassociation between affluent residents and 'green' forms of transport, including the uptake of public travel services, which potentially offer a greener alternative to personal modes of motorised for long-distance urban travel. Yet airing on the side of caution, the encouragement of public transport is not necessarily advisable, especially looking at the predicament that the proactive, similarly sized City of Oxford currently faces.

For over 30 years, Oxford has been a pioneer in the promotion of bus use in the UK, implementing the country's first park-and-ride scheme in 1973. Most recently, the high-profile local environment policy of the 1998 'Bus Quality Partnership' (BQP), resulted in the retrofitting of particulate traps to around half of the city's fleet, so that vehicles on high frequency routes met Euro II Standards (Oxford City Council, 2006). Yet in-line with the national picture, the major source of transport emissions in Oxford originate from diesel powered heavy vehicles (64% NOx) despite only accounting for 18% of traffic movements, as unlike the modernising car fleet, public services with subpar emission standards are infrequently updated (Oxford City Council, 2006). In 2001, Oxford City Council designated an AQMA, covering part of the city centre where the predicted annual mean objective for nitrogen dioxide of $40\mu\text{g}/\text{m}^3$ would not be met by the target date of December 2005. It was recognized that an average reduction in NOx emissions by 68% was required in order to meet the air quality objective, although the amount varied significantly from street to street (32-90%) as a result of differing transport trends (Oxford City Council 2006).

After a series of in-depth consultation with local service providers and various governmental bodies (e.g. DEFRA, Primary Health Trust) a series of realistic solutions became integrated into the Oxford Transport Strategy (OTS), prioritising air quality as a key agenda within the development of the city. Rarely is such a proactive and integrated stance taken at a local level within the UK. Three schemes were found to provide moderate benefits, with improvements to traffic signal phasing minimising congestion around hotspots, to cut citywide NOx levels by 12% (Oxford City Council 2006). Secondly, a 'Bus Gate Enforcement' restricting traffic from entering the High street between 07.30 and 16.30, although not formally enforced, resulted in a 20% reduction of NOx across inner-city streets (Oxford City Council 2006). However, strategies such as this do not directly reduce the amount of pollutants emitted, as they only act to spread such contaminants over a wider area. Nevertheless, the implementation of a similar scheme across central Leicester could be quite effective, perhaps encouraging the use of existing park and ride infrastructures amongst peripheral communities entering central locales. Here environmental balances would be addressed, as vehicles

entering the inner-city reduce whilst the number of trips within peripheral locales increases. Thirdly, whilst the BQP intended to upgrade the public fleet to a minimum of Euro II standards, this only consisted of a voluntary and hence partially fulfilled agreement with two main bus operators, which was only partially fulfilled. However, if this deal could be fully implemented and extended to all operators, a 20% citywide reduction in NO_x was deemed attainable (Oxford City Council 2006).

A range of other more generic solutions were also proposed, include the encouragement of carpools, work place travel plans, cycling and walking schemes, which collectively achieved only 1% reductions (Oxford City Council 2006). Thus casting doubt on whether existing protocols for such schemes could actually encourage the uptake of more sustainable personal transportation modes by Leicester's highly mobile communities. Perhaps a more targeted approach is required in which policy is more locally tailored to those characteristics and needs of such residents. Whilst increased roadside emission testing also provided minimal reductions (0.5%), it did appear a useful public exercise as a statement of Oxford City Councils intent to tackle environmental issues. Controversially, even a £20 fixed penalty for not idling ones engine once stationary were proposed, to achieve a modest 1% reduction in emissions (Oxford City Council 2006). However, enforcement was deemed unrealistic and new revenues of income could be viewed negatively, thus highlighting the delicate balance planner's face. Ambitiously, the Action Plan also investigated scrappage schemes of those worst emitting vehicles (Pre-Euro), however this was considered unviable unless financial incentives were provided at a national level.

Interestingly, NO_x levels across Oxford's central AQMA were recorded to fall 4% year-on-year from 1995-2000, this trend was surprisingly replaced by a 1.3% annual rises across 2000-2004, at the time the aforementioned schemes were implemented (Oxford City Council 2006). In-fact, the retrofitting of particulate traps across part of the bus fleet was shown accountable for inadvertently increasing NO_x levels, a measure the council considered acceptable, with the health benefits from reduced particulates outweighing those from an increase in NO_x (Oxford City Council 2006). At the time, a trial of Selective Catalytic Reduction (SCR) devices fitted to a local bus were found to reduce NO_x emissions by 70% (equivalent to Euro IV standards), however the cost of this technology in its current format was deemed unfeasible for a broader uptake (Oxford City Council 2006). Speed restrictions across central Oxford for public safety, reducing traffic speeds from 30 to 20 mph, were also thought to have increased NO_x emissions by 27% (Oxford City Council 2006). Thus highlighting how vulnerable air quality action plans are to financial constraints and the necessity of more immediately visible safety policies.

Recently the implementation of Low Emission Zone's (LEZ) have been seen as one of the more promising options to introduce greater numbers of cleaner vehicles and reduce the numbers of older more polluting vehicles on the road networks of capital cities (Tonne et al 2008, Cesaroni et al 2012). Whilst a theoretical application of comparable road user charging schemes has been seen to provide appropriate results across the considerably smaller UK municipal centre of Leeds (Mitchell 2005, Namdeo & Mitchell 2008), in practice the overall outcome of such measures remain unclear. For instance, is the draw of smaller commercial centres sufficient to retain business and a viable flow of consumers, if such a charge was in operation at a local rather than regional or national scale? Perhaps it would be more viable for a city the size of Leicester to adopt a style of LEZ, known as 'Environmental Zones', which have been in operational force across the Swedish city centres of Stockholm, Gothenburg and Malmo since 1996. These zones aim to improve air quality and reducing noise through requiring all diesel engine heavy-duty vehicles >3.5 tonnes to meet the Euro I emission standard. Older vehicles retrofitted with a certified emissions control device or new engine are also allowed to operate in the zone. In contrast to London's LEZ, such schemes are passive in nature (low cost) with zone enforcement of older vehicles existing through a permit system (windscreen stickers), with vehicles driving illegally subject to a fee enforced by police authorities. Furthermore, the self-governance of such zones would appear a success with compliance rates in Stockholm, Goteborg and Malmö in 1997 (94%) and 2004 (96%) recorded at a similar level (Goteborg's Stad Trafikkontoret, 2006).

The Goteborg Environmental Zone covers approximately 15km², containing 100,000 inhabitants whom live and work within the area. In 2004, the actual distribution of fuel types for Buses were recorded at 71.5% diesel and 28.5% gas, whereas without the Environmental Zone rates were predicted to respectively exist at 95.7% and 4.3% (Goteborg's Stad Trafikkontoret, 2006). This shows that operators were not only meeting emission standards, but also actively engaging in the uptake of greener vehicle stock, to future-proof themselves for stricter criteria. Goteborg's Environmental Zone has seen the greatest impact on particulate emissions, in terms of percentage reduction (-33.2%), with moderate reductions also occurring in relation to NO_x (-7.8%) (Goteborg's Stad Trafikkontoret, 2006). This is of huge importance, as the health effects of particles are probably the major significant health effect if all emission components are taken into consideration. Similar reductions in for particulates in the form of PM_{2.5} (-33%) and NO₂ (-5%) have been recorded when comparing 2001 levels to theoretical models where an Environment Zone is non-existent within the City of Stockholm (Rapaport 2002). As such, the Environmental Zone may be clearly viewed as an effective measure to force new technology on the market, encouraging the purchase of cleaner or

upgrading of vehicles. A particular advantage to such schemes is that a large portion of the operation cost is placed upon the polluter, aka the bus operators, and as such, Environmental Zone's follow the Polluter Pays Principle.

In order to provide a traffic management scheme across Leicester, which reduces the level of air pollutants and adheres to the PPP, one should collectively address both personal and public contributions. A central 'Bus Gate Enforcement' zone would likely curb the magnitude of environmental burdens placed on vulnerable inner-city residents, from peripheral communities with high levels of personal mobility, as existing park and ride infrastructures become more widely used. However unless the entire city was also governed by a scheme similar to that of the Environmental Zones (perhaps with stricter emission standards), environmental situations for those most vulnerable may remain if not worsen with the excessive use of poor public transport stock. Under this scheme, the regulation of the PPP is almost exclusively in the hands of an intangible entity (the bus operators), rather than an actual community of people. Whilst this may be easier to regulate, it presents a worrying scenario, as any sense of responsibility may be lost within the system. However, this is likely a catch-22 situation, in that adequate retrofitting solutions and new vehicle stock may only become economically viable, once those services become adequately used. As such, both schemes require a high level of integration to provide optimal outputs. Relating back to previous discussions, the completion of the outer-city ring road should be viewed as a necessity in the prevention of traffic entering central locales regardless of whether these schemes come into fruition, although it would assist with the schemes enforcement.

7.6. CONCLUSIONS

Whilst Polluter-Pays Principles are traditionally theorised at an international level, this chapter sought to develop upon a localised implementation of such principles through exploring a collection of spatially detailed intra-urban communities, within the context of social, environmental and health outcomes. Although, Mitchell & Dorling (2003) successfully formulated such a procedure, their enquiry was limited to only aspects of deprivation and pollution, and the spatial resolution of their nationwide study failed to adequately outline intra-urban interactions. In particular, intra-urban areas are thought to represent some of the most extreme disparities in socio-environmental attributes, and as such it is conceivable that their responses were previously smoothed out by this broader spatial analysis.

To address such issues, annual LLSOA estimates of road-transport PM_{10} (TPM_{10}) emissions created from individual communities throughout Leicester, were derived from combining vehicle fleet composition counts with workforce trips, assumed to represent the significant proportion of population movements. The Local Moran's I test, identified a moderate and positive level of spatial structuring to exist across Leicester ($R^2 = 0.44$) in terms of community emission contributions. Overall, these outputs revealed the city's greatest polluters to reside predominantly within affluent communities located along the city's periphery, whereas those creating the least emissions resided in central locations, experiencing a range of socio-environmental health burdens. Whilst some inner-city communities moderately contributed towards their environmental demise, these contributions were substantially outweighed by those made from external communities, whom appear to avoid the social, environment and physical cost of their actions. In its current state, the city's traffic management strategy seemingly operates in an ethically unjust manner.

Contour plots, simultaneously examining created and residentially experienced TPM_{10} emissions against a third attribute, confirmed children's respiratory cases to rise in relation to residential pollutant levels. However, the effects of this generally materialised in a more meaningful manner across those communities whom emit lower levels of pollutants from personal modes of transportation. Children with severely reduced respiratory health tended to be housed within inner-city communities contributing modest amounts of transport related pollutants. In a just situation their outputs would result in only moderate health implications, however at present these residents are also unfairly plagued by the contributions of external communities, whom health wise pay very little.

Overlap statistics cross-examining transitions in community created transport emissions (from private modes) with children's collective or specific respiratory conditions, revealed an immediate spatial association between both elements ($O_S P \uparrow < 0.05$). These shared boundaries depict an inverse relationship between TPM_{10} production and moderate-mild respiratory outcomes across first-order communities, located away from the previously highlighted central zone of concern. In contrast, the limited collection of mutually corresponding inner-city boundaries, would infer that these residents are unable to successfully shift their burdens onto neighbouring communities to the same extent. Whilst the pure volume of vehicles commuting from Leicester's affluent peripheral communities are shown to impact all en-route, the full force of such burdens in relation to children's URT and LRT health were respectively recorded to occur between 248-489m and 410-541m outside of these polluter pockets ($O_G, O_{GH} P \downarrow < 0.05$). Such measurements approximately mark the reaccumulation of vehicles originating from Knighton (collection of peripheral communities

most at fault) on approach to common central destinations of commerce, located near those socially and environmentally disadvantaged communities. As discussed previously, respiratory symptoms of reduced severity appear to be the initial response towards the introduction of environmental influences.

Typically, 'White British' families resided within communities experiencing moderate to low residential TPM_{10} exposures. However, 'White British' families are not fundamentally accountable for Leicester's environmentally unjust predicament, as only a small segment of this societal group highly pollute and residentially experience few discharges. In-fact, overlap statistics recorded spatial transitions in 'White British' residency to be located across significantly distant locations of the city, when compared to boundaries in levels of community created TPM_{10} ($O_G=1292m$, $P\uparrow=0.04$), and vice versa ($O_H=1071m$, $P\uparrow=0.04$). To a certain extent this confirms that the ethnic majority is not unanimously responsible for the city's environmental burdens, as if this was the case, one would expect a sharp mutual emergence of both conditions.

It would appear that it is not race, but deprivation, which is the key driving factor behind the creation of personal TPM_{10} emissions; although elements of race, appear involved in determining affluence. For instance, Leicester's most affluent 10% of communities, predominantly located around the wards of Knighton and Humberstone, personally created a hefty 0.035t/yr. of TPM_{10} , yet residentially experiencing only 0.85t/yr. of TPM_{10} emissions. Whilst, the composition of children from such communities remains predominantly of 'White British' origin (70.34%), these areas are also representative of elements of successful integration with sections of the 'Indian' community (17.30%). Contour plots of deprivation versus pollutant scenarios reinforce this point, revealing a structure of strong social banding to exist throughout the City of Leicester; whereby affluent residents generally contributed the highest levels of private transport emissions whilst residing in areas experiencing low levels of road-transport emissions. Yet, simply pointing the finger at those communities is not a particularly fair assessment of the current state of environmental affairs. For instance, it was deemed socially unfair to raise the environmental accountability of those communities with high employment rates, where travel becomes a necessity. In many ways these inhabitants already pay societal contributions (collected via taxation), which benefit those in vulnerable situations. Rather it became of interest to place environmental accountability on excessive travel and the use of certain transportation measures, after existing societal contributions are accounted for. Even after adjusting for levels of employment, persons from affluent communities appeared more likely to commute greater distances to work on their daily return commute (>15km/PEP) compared to their socially disadvantaged counterparts (0-15km/PEP).

This increased travel distance of affluent communities encouraged the use of more environmentally unfriendly modes of transportation (96-84% km/PEP). Furthermore, such trips seemingly terminated at common destinations, causing an accumulation of pollutants potentially exacerbated by the shorter trips of those less privileged residents living within the nearby vicinity. Meanwhile, increased residential awareness towards an areas environmental predicament, was shown by an uptake of 'green' transportation measures across polluted communities (>28% km/PEP), regardless of social status.

Although one should note that levels of 'green' transportation also increased with deprivation, though not to be of personal choice but out of necessity. Whilst the encouragement of physical forms of travel is seen to reduce obesity, for central communities these benefits are likely offset by movements across polluted districts. Interestingly, communities feeling the brunt of the 'double burden' have a reduced use of 'green' transport (16-28% km/PEP), offset by the acquisition of low-grade vehicles thought accountable for exacerbating such burdens. Therefore, the encouragement of 'green' modes should be viewed as a means of mitigation rather than an out-and-out solution. Yet, even if such actions were in operation, further measures are still required to limit external communities from shifting their share of pollutants onto these inhabitants.

Through exploring the wider spectrum of transport measures in use across Leicester, it became possible to understand (in a rather crude manner) the extent to which communities with high levels of personal transport modes contributed towards the overall environmental burden. Public transport appeared evenly distributed throughout the city at a moderate-low level of uptake, with no evidence existing for assigning an excessive use of such services to any particular community. Furthermore, where some suburban communities residential favoured public transport, road-transport emissions remained low. As such, there is no direct evidence that the frequency of Buses passing near to residential areas act as a key driving force behind a city's environmental issues. However, the confined nature of the inner-city acting to contain pollutants (urban canyon effect), combined with the pure volume of public services terminating at a central hub, is likely to be problematic. Here, exclusively high public and private transportation contributions are thought to combine, resulting in an environmental effect of volatile proportions. As such, future research should aim at conducting a wider transportation enquiry, inclusive of the travel contributions from public services, whose environmental accountability is with the individual service providers.

Whilst the majority of travel distance remains completed via personal transport, communities favouring this mode of transportation, characteristically of an affluent nature, share high disassociations with an uptake of public travel services. Affluent communities

therefore do not inconveniencing external communities through a spectrum of mechanical measures. In addition, those poorest inner-city communities are not observed to have a raised level of public transport use, and are thus not involved in fabricating their own environmental problems from other unexplored mechanical modes. However, a low uptake of public services here is perhaps an issue of concern, as some inner-city communities have started to favour the use of inexpensive and often poor private vehicles; with this newfound mobility seemingly resulting in increased social standing, but at the cost of environmental attributes. To tackle such issues, perhaps these central communities require further incentive to use public modes, rather than adding vehicles to the road network. However any measures should not be conducted in a manner which impedes the social climb of such communities, with public transportation services in their current format potentially offering little towards the access of new job markets (i.e. out of town industrial/distribution centres).

Similarly, action is required across several suburban deprived and physically unhealthy 'White British' communities, whom may be easily swayed between public or private transportation modes (typically the later action occurs with increased social standing). However, the small commute distances and reduced physical health of these inhabitants, questions their need for mechanical transportation forms. Therefore, in certain cases a balance needs to be found between encouraging physical transportation modes, without prompting a shift from public to personal transportation modes. In addition, the likely negative stigma of public services amongst affluent communities requires further investigation. For these communities, a more likely and immediate response for mitigating their environmental contributions involves the use of carpooling. Here, the convenience and luxury of personal transportation is likely to be favoured, and could mark a substantial reduction in the volume of vehicles which enter inner-city areas. However, at present, the uptake of carpooling appears most favourable across those most deprived communities conducting the fewest and smallest trips via personal modes, and thus offers few benefits.

The use of contour plots and LISA to model the intra-urban PPP have respectively allowed for the exploration of global structures and detection of individual communities at the heart of the local environmental debate. The wider implementation of these practices will benefit the development of future local transport policy. Through focusing the consultation process, 'green' initiatives may be tailored towards the needs of local populaces, potentially increasing the success of such initiatives where prior uptake has been low.

CONCLUSIONS & FUTURE RESEARCH

OVERVIEW

The overall aim of this thesis was to gain a greater understanding and insight of the respiratory health burdens attributed to transport derived air pollutants, across diverging sub-city communities within an Environmental Justice (EJ) context. In reviewing the literature, conventional modelling strategies of a temporal nature were recognised as an effective means of deriving a citywide risk from pollutant exposure; yet were considered wholly inadequate in the wider evaluation of health equity, in which social and physical environmental stimuli are considered locally entwined. As such, emerging modelling techniques incorporating the spatial structures of urban environments were implemented (after undergoing a process of extensive statistical verification), to establish the magnitude to which a 'triple jeopardy' of social, health and environmental inequalities actually exist. In focusing on the prototypical multicultural UK City of Leicester, a substantial ethnic mix was explored across a setting deemed to experience conceivably minimal localised socio-ethnic disparities. It is believed that the holistic nature of the presented spatial models is capable of providing a more comprehensive overview of and insight into this topic than has previously been offered.

In this chapter, the conclusions from the thesis are detailed relating back to the initial research objectives set in chapter 1. Ultimately, suggestions for further research are made to conclude the thesis. It should be emphasised that in spite of the fact that the models are purely discussed in a socio-environmental context, validation procedures highlight a potential use in many other contexts examining events of a spatial nature; perhaps uncovering further ties previously left unexplored by temporal techniques.

8.1. PRIMARY CONCLUSIONS

The modelling and prediction of health disparities with respect to pollutants emitted from road-transportation across the urban environment is a formidable task, concerning a series of spatially entwined social and physical stimuli. Thus, a requisite for the development, validation and implementation of spatially inclusive methods to evaluate multiple rather than single component burdens has long been emphasised. However, prior to exploring the extent to which socio-environmental influences collectively impede health, a sense of focus was traditionally placed on solely quantifying the influence of localised variations in road-transport on health during childhood (chapter 1.3):

- I. **To consider spatial variations in respiratory health, establishing their affiliations with airborne pollutants emitted from mobile sources, across the City of Leicester's high-resolution Lower Level Super Output Area (LLSOA) census blocks.**

AIM I: HIGHLIGHTS

- *Localised Indices of Spatial Association (LISA) identified a significant clustering of children's respiratory hospitalisations across inner city locales ($P \leq 0.05$), with rates 30-270% above the citywide average*
- *A ring road encircles inner-city residential areas, producing annual TPM_{10} emission levels 75% above the citywide average*
- *Bivariate correlation measures identified moderate global and first-order (inner-city) associations between children's respiratory hospitalisations and TPM_{10} , where $P \leq 0.05$*
- *Heightened respiratory risks exist within close proximity to zones of rapid change in TPM_{10} (283m, $P \leq 0.01$), providing a European exposure-proximity threshold in agreement with American literature*
- *Local regression models accounting for spatial relationship structures indicate that TPM_{10} influences 475 children's respiratory admissions per annum across Leicester (18.96% of the total burden)*

In the understanding of this question, one may initially refer back to objective 1 (chapter 1.4) involving the use of exploratory data analysis techniques to geographically determine and quantify the magnitude of correlation between road transport emissions and NHS hospital records. In chapter 4, a spatial composite index known as the Global Moran's I coefficient (Moran 1948) was applied to measure the extent of overall clustering of the aforementioned parameters. Here, a Global Moran's I value of 0.39 was recorded for children's overall respiratory hospitalisation rates during 2000-09 under a first-order weighting scheme, identifying moderate citywide levels of spatial correlation between directly adjacent LLSOA communities ($P < 0.01$). For PM_{10} road transport emissions (TPM_{10}) a looser sense of spatial

dependency was observed, as shown by a first-order value of 0.83 moderately deteriorating to a value of 0.28 for observations separated by a fourth-order distance ($P < 0.01$).

Localised Indices of Spatial Association (LISA) in the form of the Local Moran's I coefficient (Anselin 1995), subsequently dissected these global coefficient values allowing for a geographic exploration of variation across first-order observations. The Local Moran's I statistical test identified the existence of a significantly high clustering of average annual respiratory hospital admissions across inner city children. Observed hospitalisation rates within this zone of high clustering range from 49.39 to 147.83 admissions per 1,000 children, which is considerably higher than Leicester UAs average rate of 39.43 admissions. Furthermore, this highlighted area of interest was noted to contain the city's inner ring road and several key arterial roads linked with the city's outer ring road, and national road network. Residents within these respective inner city clusters were identified to annually experience an extra 0.78 t/yr. of TPM_{10} .

Traditional bivariate correlation measures, in the form of Pearson's R, confirmed the existence of an important and linear correlation between children's respiratory hospitalisations and TPM_{10} emissions (0.37, $P < 0.01$). Bivariate Local Moran's I statistic conducted in a manner, which held children's J00-99 admissions at ego locations (i) whilst placing the influence of TPM_{10} at first-order LLSOAs (j), also demonstrated this positive relationship to continue into neighbouring locales. Here, substantial positive correlations between respiratory symptoms and TPM_{10} emission levels appear to almost exclusively inhabit inner city LLSOAs. Meanwhile, low levels of respiratory incident were accompanied by reduced TPM_{10} emissions across Leicester's eastern periphery, which harbours the missing section of the city's outer ring road. As such traffic is diverted towards the inner-city, and these eastern communities are thus viewed as passing on their share of residentially expected environmental burdens.

Objective 6 (chapter 1.4) continues with this theme of spatial pattern recognition, in seeking to describe and analyse the existence of geographic boundaries between transport emissions and hospital admissions; involving a tangent of boundary detection procedures (Womble 1951) known as polygon wombling (Jacquez & Greiling 2003). Health boundaries are of intrinsic medical interest, in that they reflect the geographic extent and intensity of underlying physical and or social processes, identifying populations whom are most likely to be at risk from subsequent fluctuations. Traditionally, the spatial extent of health impacts associated with road-transportation has been explored through the quantification of effects within artificial buffers, defined by subjective distances from specified major road links; a process which has established a critical threshold of 100-400m in relation to particulate matter

concentrations (Zhou et al 2007). In constructing naturally occurring health and emission boundaries across Leicester (unrestricted to specific road-links), chapter 6 has shown unbiased markers of extreme environmental pollutant levels to construct children's J00-99 health fronts 283m away from these sources of emission ($O_G P \downarrow < 0.01$). An exposure-proximity threshold which appears to verify those critical benchmarks set by traditional measures, thus encouraging the wider uptake of boundary statistics which to-date have received few demographic applications.

In seeking to account for these seemingly spatial dependent relations between respiratory health and airborne pollutants emitted from mobile sources, a series of quantitative models were constructed to provide a dose-response evaluation. Initially this was conducted in line with objective 3 (chapter 1.4), as presented in chapter 4's construction of spatially suitable global regression models. Here, traditional global multivariate regression results indicated each tonne of residentially experienced TPM_{10} emissions within Leicester to be associated with an annual increase of 7.78 J00-99 hospital admissions per 1,000 children (19.66% of the overall respiratory burden, $p < 0.05$), after controlling for socioeconomic characteristics. Conservative multilevel models, addressing certain dataset issues of spatial nonstationarity through the incorporation of a generalised set of spatial structures in disease tolerance (5-quantiles), similarly identified a 1 t/yr. increment of TPM_{10} to annually account for an additional 8.75 J00-99 hospital admissions per 1,000 children ($P \leq 0.05$). However after removing an outlying city centre LLSOA of interest, global admission rates associated with TPM_{10} emissions were observed to fall to 2.50 hospitalisations per 1,000 children ($P \leq 0.05$). Thus highlighting the magnitude of extremely localised spatial dependence found within an urban setting, of which inner-city children would appear to experience an overwhelming detrimental impact caused by TPM_{10} emissions.

As such, the use of localised regression techniques for the integration and exploration of multiple previously unidentified non-stationary relationships are preferential evaluator mechanisms (objective 4, chapter 1.4). Geographically Weighted Regression (GWR) modelling practices indicate residentially experienced TPM_{10} emissions after adjustment for social covariates, to influence 178-545 children's respiratory admissions per annum (7.09-21.74% of all children's respiratory admissions) across Leicester during 2000-2009. The GWR model outputs also allowed for the creation of an annual road-transport emissions dose-response relationship from localised regression models with significance at the 95% confidence interval:

$$J00-99 \text{ cases per } 1,000 \text{ children} = -15.691 + (10.394 * TPM_{10}) + (11.223 * TPM_{10}^2)$$

Using this trend captured from a 60NN GWR model, LLSOA TPM₁₀ emission levels were reported to average 8.79 admissions per 1,000 children, influencing 475 children's respiratory admissions per annum across Leicester (18.96% of the total J00-99 respiratory burden). The threshold for the occurrence of hospitalisation was identified to occur within areas experiencing annual TPM₁₀ emission rates above 0.81 tonnes (P<0.05). Furthermore, it is calculated that a 5% reduction in residentially experienced LLSOA TPM₁₀ emissions across Leicester would amount to 110 fewer children's respiratory admissions per annum (P<0.05), reducing the total amount of respiratory hospital incidents by 4.39%. To the authors knowledge this is the first time a dose-response relationship has been specifically associated with road-transport emissions.

The second aim of this research project is closely related, and as such shares many of the objectives discussed in the section above:

- II. To investigate how spatial variations in social-ethnic status relate to and interact with airborne pollutants emitted from mobile sources, across the model British multicultural City of Leicester. Thus, understanding the failure of temporal models in capturing Pearce et al's (2010) 'triple jeopardy', within a setting where environmental injustices are considered minimalistic.**

AIM II: HIGHLIGHTS

- *Inner city children experience disproportionately large socio-environmental respiratory burdens in line with Pearce et al's (2010) 'triple jeopardy'*
- *Inner city neighbourhoods tend to house greater numbers of children from ethnic minorities, questioning the environmental equity of Leicester*
- *LISA identified a mutual clustering of deprivation and TPM₁₀ emissions across inner city locales, indicating that a double-burden collectively influences a child's respiratory status (P≤0.05)*
- *Respiratory risks universally appear within close proximity to zones of rapid change in deprivation and TPM₁₀, recording an exposure-threshold of 334m (P≤0.01)*
- *Local regression models measured LLSOA community levels of deprivation to annually influence +17.96% of citywide J00-99 cases*
- *It is theorised that exposure to detrimental socio-environmental factors may initiate URTI episodes, with sustained exposure causing an immunosuppressive response, encouraging the onset of viral infection manifesting as LRTI episodes*

In the understanding of this question, one may initially refer back to objective 2 (chapter 1.4), which seeks to determine whether ethnic minorities and/or deprived communities reside

within locations experiencing reduced levels of air quality. Similar to objective 1 this was to be achieved through an exploratory spatial data analysis, primarily contained within chapter 4.

Here, Global Moran's I statistics identified substantial levels of spatial autocorrelation amongst first-order observations (<450m separation) of deprivation (0.56) and levels of 'White British' children (0.82), where $P < 0.01$. Akin to TPM_{10} emissions, a looser sense of spatial dependency existed than to what was presented by hospitalisation rates which are exclusively of a first-order phenomenon; with moderate magnitudes of spatial correlation respectively persisting across third and fourth-order observations for measures of deprivation (0.22) and 'White British' children (0.40), where $P < 0.01$. In dissecting these spatial composite indices, Local Moran's I coefficients founded upon a first-order row standardised weighting system, notably identified similar patterns of clustering for levels of residentially experienced TPM_{10} emissions and Carstairs Index measures of deprivation across inner city locales; highlighting the potential avenue for a double burden of social and environmental issues collectively influencing a person's wellbeing. The case for environmental equity may be further brought into question upon examining the distribution of Leicester's White British ethnic group, which is identified to heavily populate the city's southern and western peripheries. Significantly low levels of clustering for persons of White British origin, within and adjacent to the east of the city centre thus instigate a potential scenario in which ethnic minority groups bear a disproportionate burden of environmental and social problems.

LLSOAs contained within the inner city respiratory hot-spot were observed to experience annual average J00-99 admission rates of 80.65 cases per 1,000 children, whereas communities positioned within cold-spots reported an average rate 1.9 times smaller. Substantial socio-environmental differences appear to occur between the inner-city hot-spots and those external cold-spots, with residents from the respiratory pocket of concern experiencing Carstairs Index deprivation values 4.24 ranks higher, in addition to being exposed to an extra 1.06 t/yr. of TPM_{10} emissions than their cold-spot counterparts. Interestingly, J00-99 hotspot communities respectively housed 6.90% and 11.33% more children of 'Other South Asian' and 'Afro-Caribbean' ethnicity than their cold-spot counterparts. Meanwhile, 32.78% more children of Indian ethnicity were found to occupy Leicester's respiratory cold-spots. Here, It is plausible that the lifestyle choices of this particular minority group uniquely prevents the onset of poor health, and, or that such social groups have the knowledge to access relevant public services to mitigate the severity and extent of such complaints.

As with TPM_{10} , traditional statistical bivariate correlation measures (Pearson's R) confirmed the existence of an important and linear correlation between children's respiratory hospitalisations and deprivation (0.40) or 'Afro-Caribbean' residency (0.39), where $P < 0.01$.

Moderate levels of negative non-linear correlation (Spearman's Rho) were also observed to occur between J00-99 admissions and levels of 'Indian' children (-0.38, $p < 0.01$), reconfirming the beneficial influence associated with 'Indian' lifestyle choices. Furthermore, Carstairs Index values of deprivation across Leicester were observed to yield significant linear correlations of interest with TPM_{10} emissions (0.40), and community levels of 'Afro-Caribbean' children (0.30). Linear correlations were also observed between levels of TPM_{10} emissions and residents of 'Afro-Caribbean' (0.42) and 'Other South Asian' (0.43) ethnicities. Such correlation statistics broadly compliment the aforementioned Local Moran's I outputs, appearing in agreement with Pearce et al's (2010) 'triple jeopardy' of social, health and environmental inequalities. Bivariate Local Moran's I statistics comparing children's respiratory admissions to social markers across first-order neighbouring locales, confirm that these elements of spatial correlation are upheld for deprivation (0.22), 'Indian' (-0.17) and 'Afro-Caribbean' (0.21) ethnicities, where $P < 0.05$.

Upon establishing that deprived communities and select minority groups reside within locations experiencing reduced levels of air quality, objective 6 (chapter 1.4) looked to quantify the magnitude in which these spatial relations operate. Within chapter 6, extreme shifts in the proportion of 'Afro-Caribbean' children housed at the LLSOA level were identified to occur within close proximity to boundaries in children's overall respiratory health ($O_G = 296m$, $P \downarrow < 0.01$). Meanwhile, overlap statistics for respiratory health boundaries alongside 'Indian' ethnicity, 'Other South Asian' ethnicity and deprivation measures, revealed corresponding areas of rapid change to be of limited significance. Interestingly, substantial spatial shifts in 'Afro-Caribbean' residency were identified to reduce the distance between TPM_{10} boundaries and respiratory incidents by 38m ($P \downarrow < 0.01$), thus implying that these two independent variables operate across spatially similar neighbourhoods.

Whilst bivariate boundaries of TPM_{10} emissions and 'Afro-Caribbean' lifestyle choices were observed to instigate the bulk of respiratory admission cases across inner-city locales, J00-99 health fronts were not uniformly located within proximal distances to such features (O_H $P \downarrow > 0.1$), indicating that additional influences have a role in determining the city's wider respiratory issues. In combining surfaces of deprivation with TPM_{10} emissions, one would appear to be able to adequately capture respiratory health fronts caused by such a double burden ($O_H = 334m$, $P \downarrow = 0.01$). Yet bivariate boundaries in deprivation and TPM_{10} were found to not universally trigger a J00-99 health front (O_G , O_{GH} $P \downarrow > 0.05$), a phenomenon possibly introduced by an over-smoothed TPM_{10} dataset being unable to restrict deprivation driven boundary placements. Nevertheless, in constructing trivariate boundary sets containing readings of TPM_{10} , deprivation and either 'Afro-Caribbean' or 'Other South Asian' residency

levels as their final component, a close visual resemblance of respiratory health boundaries was obtained ($O_H \leq 343m$, $O_S \geq 10$ BEs).

The next stage of analysis falls in-line with objective 3 (chapter 1.4), and refers back to chapter 4's series of quantitative models which provide appropriate global dose-response relations for social as well as environmental stimuli. Here, traditional multivariate OLS linear regression procedures revealed deprivation to be annually associated with 558 respiratory children's cases (+22.29%), and TPM_{10} emissions with 378 cases (+15.10%). Meanwhile levels of 'Afro-Caribbean' and 'Other South Asian' residency respectively influenced 106 (+4.23%) and 142 (+5.67%) cases ($P < 0.05$). In contrast, levels of 'Indian' residency were associated with substantial respiratory benefits, resulting in 498 fewer children's annual J00-99 admissions (-19.89%). A traditional multivariate analysis thus provides strong evidence to support the concept that an accumulation in respiratory risk is distributed disproportionately with respect to socioeconomic status (SES), specific ethnic minorities, and environmental exposures within Leicester UA's 0-15 year age group.

Multilevel models accounting for quantile variations in respiratory outcomes prior to the examination of socio-environmental stimuli, provided extremely conservative estimates which were only able to confirm a strong prevailing signal with respect to TPM_{10} . However, upon the removal of the focal LLSOA of interest, signals for TPM_{10} , deprivation, Indian residency and 'Afro-Caribbean' residency were observed to remerge, albeit at the respectively diminished levels of 5.26% ($P < 0.05$), 3.30 ($P < 0.1$), -3.50% ($P < 0.05$) and 4.7% ($P < 0.1$) of citywide J00-99 cases. Still such values should not be taken at face value, but rather as signals of the key underlying mechanisms operating across the city; in the face of multilevel models which have discounted a high level of explanatory variation from such processes (trend validation modelling).

From here, localised regression techniques were applied in order to collectively integrate and explore the presence of multiple non-stationary socio-environmental relationships on respiratory outcomes (objective 4, chapter 1.4). For the key socio-environmental factors of interest, GWR modelling practices indicated LLSOA community levels of deprivation as annually accountable for +17.96% (17.93-21.41%) of citywide J00-99 cases, TPM_{10} for +15.66% (7.09-21.74%), 'Other South Asian' residency for +5.69% (5.41-5.93%), 'Afro-Caribbean' residency for +2.40 (1.32-2.88%), and 'Indian' residency for -14.98% (-16.70 to -14.98%). GWR model outputs for TPM_{10} significantly confirm that the brunt of their associated health impacts are focused upon inner-city residents ($P < 0.05$). Likewise, the upper parameter estimates for deprivation are found to coexist across inner-city locales. These findings clearly indicate that a 'double burden' of environmental exposure and deprivation operates across

inner-city communities, to collectively impede respiratory health during childhood. Still, it would appear that this mutual relationship may have an element of exclusivity to inner city locales, as a second deprivation cluster of concern is observed along Leicester's southern periphery where relatively low TPM₁₀ levels are reported.

Whilst 'Afro-Caribbean' families tending to reside within inner-city locales, partially contained within the respiratory zone of concern, it would appear that Afro-Caribbean lifestyle choices here actually have a low influence on respiratory outcomes. Whether the lifestyle choices of 'Afro-Caribbean' residents within inner-city locales actually mitigates other detrimental influences remains questionable, as all three local models providing insignificant coefficients within such areas. Furthermore such beneficial outputs fly in the face of citywide trends, with 'Afro-Caribbean' residence levels associated with reduced respiratory health across Leicester's north-easterly sector ($P < 0.05$). One possibility is that social groups may be forced to positively alter their traditional lifestyle choices, when exposed to an excessive combination of social-environmental burdens which provide recognisable health impacts. Alternatively the unfavourable lifestyles of 'Afro-Caribbean' residents within inner-city locales may have been overshadowed by the far greater health impacts brought about by the 'double burden' deprivation and air pollutant exposure. In either case it would appear that this ethnic minority group unfairly experiences a 'triple jeopardy' of social, health and environmental inequalities.

Perhaps most interesting are the significantly modelled hospital burdens attributed to the lifestyle choices of 'Other South Asian' residents, within the western and northern districts of Leicester heavily populated by 'White British' residents. Here, respiratory hospitalisations would appear to prevail, in spite of the social mobility, which has enabled later generations of the 'Other South Asian' group to locate away from problematic inner-city areas. The causes of which may be due to persevered lifestyle choices, which potentially break down to an insufficient understanding of how to accessing basic public services. Alternatively, if families have recently relocated away from inner-city areas, children's health gradient constraints are likely to prevail, with social mobility only allowing one to moderate prior health issues (Blane et al 1999). In stark contrast to the cities other minority groups, GWR model estimates confirm that levels of 'Indian' residency substantially decrease the likelihood of children's respiratory admissions occurring. Furthermore, cartographic plots indicate that the lifestyle choices of 'Indian' residents actively reduces the number of respiratory hospitalisations across the inner-city respiratory hot-spot, acting in a manner which mitigates the spatial spread of such symptoms to the wider city centre communities.

Upon establishing these foundations, objective 5 (chapter 1.4) sought to determine whether spatial relationships exist between specific relatively minor and severe respiratory conditions, and if so, what is the extent to which socio-environmental mechanisms play in the decline of a child's respiratory health. Pearson's R procedures identified a substantial degree of linear association (0.80) to exist exclusively between relatively severe (J20-22) and mild (J00-06) respiratory infections ($P < 0.01$); the two conditions respectively involved within 41.65% and 23.66% of children's respiratory hospitalisations. Local Moran's I statistics also detected major hot-spots within common inner-city localities, and some common cold-spots towards Leicester's northern and eastern peripheries ($P < 0.05$)

From here, GWR models disseminated localised J00-06 and J20-22 admissions to specific socio-environmental factors, to determine whether common mechanisms are accountable for a decline in children's respiratory health. Pearson's R tests identified a substantial degree of linear association between modelled upper respiratory and lower respiratory tract infection (URTI, LRTI) hospitalisations caused by TPM_{10} emissions (0.85), deprivation (0.76), and community levels of Indian (0.72) children. Bivariate Local Moran's I tests also spatially identified TPM_{10} emissions and levels of deprivation as likely candidates responsible for a communities deteriorating respiratory health. Exacerbations of J00-06 and J20-20 admissions associated with these two factors appeared to solely affect inner city communities, confirming the previously reported 'triple jeopardy' of social, health and environmental inequalities within Leicester. Lifestyle choices, such as those seen by Indian residents, were also shown to mitigate the influence of detrimental socio-environmental factors, and in part reduce the potential spatial extent of such respiratory issues around other centric localities.

Interestingly, seasonal decompositions of children's URTI's show a 50-50 split across hot and cold seasons within Leicester from 2000-09, whereas LRTI's prevail during the cold season (77.0%) when viral activity is most abundant. Thus, it is theorised that exposure to detrimental socio-environmental factors may initiate URTI episodes, with prolonging recovery times likely occurring from sustained exposures. If a sufficient level of recovery is not reached in time for the cold season, then the child may become host to a viral infection exacerbating previous respiratory complaints, potentially resulting in lower respiratory tract conditions of greater severity. To date, such immunosuppressive responses have been widely reported within laboratory conditions (Becker & Soukup 1999, Gilmour et al 2001), with limited supportive evidence existing across real-world settings (Karr et al 2009), until now.

Furthermore, the findings of this investigation appear to confirm an extensive body of anecdotal research (Fry & Sandler 1993), which has previously suggested a connection

between certain socio-environmental influences and cases of 'Catarrhal Child Syndrome' (CCS); the common childhood condition collectively describing the frequent reoccurrence of acute respiratory infections, only partially linked to viral activity. It is believed that the identified socio-environmental mechanisms behind CCS have previously eluded health experts, as spatial modelling techniques appear to be a necessary force in filtering geographical noise from the signal.

Finally, the third aim of this research project is focused around the concept of a localised polluter pays principle, and whether such principles are already in existence or in requirement (chapter 1.3):

- III. To dynamically assess the mobile polluting potentials of sub-city population groups, in order to ascertain whether those contributing towards the environmental degradation of the city, experience proportional environmental, social and or health burdens.**

AIM III: Highlights

- *LISA measured moderate and positive level of spatial structuring across Leicester (R^2 0.44) with regards to community TPM_{10} contributions from personal transport ($P \leq 0.05$)*
- *The cities greatest polluters reside within affluent communities along the cities periphery and are capable of shifting their personal TPM_{10} contributions onto others*
- *Those communities creating the least emissions resided in central locations unfairly plagued by a range of social, environmental and health burdens*
- *Increased levels of personal mobility occur if inner-city communities purchase low-grade vehicle stock, to the further detriment of their environmental positioning*
- *Intra-urban modelling of the PPP allows for the detection of individual communities at the heart of environmental debate, with targeted consultation tailoring 'green' initiatives towards the needs of local populaces*

In accordance to objective 7 (chapter 1.4), community specific TPM_{10} contributions involving the use of personal transportation were defined in a dynamic fashion, through the use of workforce origin-destination matrices in combination with DVLA citations of vehicle ownership. From here, it became possible to evaluate whether transport related pollutant levels are caused by local communities or through the movement of external social groups residing within another sector of the city (objective 8). Within chapter 7, Local Moran's I statistics identified moderate and positive level of spatial structuring to exist across Leicester (R^2 0.44) with regards to community emission contributions. Overall, these outputs revealed

the cities greatest polluters to predominantly reside within affluent communities located along the cities periphery, whereas those creating the least emissions resided in central locations, experiencing a range of socio-environmental health burdens. Whilst some inner-city communities moderately contributed towards their environmental demise, these contributions were substantially outweighed by those made from external communities, whom appear to avoid the social, environment and physical cost of their actions. In its current state, the city's traffic management strategy seemingly operates in an ethically unjust manner.

Contour plots, simultaneously examining created and residentially experienced TPM_{10} emissions against a third attribute, confirmed children's respiratory cases to rise in relation to residential pollutant levels. However, the effects of this generally appeared to be felt in a more meaningful manner by those communities whom emit lower levels of pollutants from personal modes of transportation. Furthermore, children with severely reduced respiratory health tended to be housed within inner-city communities contributing modest amounts of transport related pollutants. In a just situation their outputs would result in only moderate health implications, however at present these residents are also unfairly plagued by the contributions of external communities, whom health wise pay very little. Interestingly it would appear that it is not race, but deprivation which is the key driving factor behind the creation on personal TPM_{10} emissions; although elements of race would appear involved in determining affluence. Whilst Leicester's most affluent 10% of communities, predominantly house White British' families (70.34%), these areas are also representative of elements of successful integration with sections of the 'Indian' community (17.30%).

Even after adjusting for levels of employment, persons from affluent communities appeared more likely to commute greater distances to work on their daily return commute (>15km/PEP) compared to their socially disadvantaged counterparts (0-15km/PEP). This increased travel distance of affluent communities was found to encourage the use of more environmentally unfriendly modes of transportation (84-96% km/PEP). Meanwhile, increased residential awareness towards an areas environmental predicament, was shown by an uptake of 'green' transportation measures across polluted communities (>28% km/PEP), regardless of social status. Whilst the encouragement of physical forms of travel is seen to reduce obesity, for central communities these benefits are likely offset by movements across polluted districts. Interestingly, communities feeling the brunt of the 'double burden' have a reduced use of 'green' transport (16-28% km/PEP), offset by the acquisition of low grade vehicles, thought accountable for exacerbating such burdens. Here, the encouragement of 'green' transportation modes is as a means of mitigation rather than an out and out solution. Yet,

even if such actions were in operation, further measures are still required to limit external communities from shifting their share of pollutants onto these inhabitants.

Upon defining the extent to which such environmental inequalities exist, objective 9 (chapter 1.4) sought to provide a series of localised initiatives and more generalised transport policies to readdress those imbalances. For instance, a low uptake of public services across inner-city locales is an issue of concern, especially where such communities have started to favour the use of inexpensive private vehicles. Interestingly this newfound mobility appears associated with an increased level of social standing, to the further detriment of their local environment. Within the city's traffic management plan, it is of future interest to consult inner-city communities in a strategic manner, to devise local schemes that improve and or encouraging the use of public transportation. However, any measures should not impede the social climb of such communities, considering that public transportation services in their current centrally focused format, potentially offer little towards the access of new job markets (i.e. out of town industrial/distribution centres). Therefore, the consultation process should also explore whether it is economically feasible to provide assistance towards the maintenance of vehicle stock within deprived inner-city communities. At the other end of the social spectrum, a negative stigma towards the use of public services appears to present itself within those affluent and subsequently most mobile communities. For these communities, a more likely and immediate response for mitigating their environmental contributions involves the use of carpooling. Here, the convenience and luxury of personal transportation is likely to be favoured over public services, and could mark a substantial reduction in the volume of vehicles which enter inner-city areas if correctly tailored to specific local communities.

In terms of short-term infrastructure development, a series of bottlenecks have been observed in relation to the missing eastern section of the outer ring-road, which coincidentally falls within close proximity to some of those most affluent areas. If completed not only would one expect problematic areas associated with congestion to be eliminated, traffic would also be discouraged from entering the inner city, potentially mitigating the environmental burdens felt by those most vulnerable communities. In the long term it is suggested that a central 'Bus Gate Enforcement' zone would likely curb the magnitude of environmental burdens placed on vulnerable inner-city residents, from peripheral communities with high levels of personal mobility, as existing park and ride infrastructures become more widely used. However unless the entire city was also governed by a scheme similar to that of the Scandinavian Environmental Zones (perhaps with stricter emission standards), the environmental situations for those most vulnerable may remain, if not worsen with the excessive use of poor public

transport stock; thus demonstrating the complexity of issues currently faced by local transport management.

8.2. ECOLOGICAL CONSIDERATIONS

In evaluating spatially aggregated health and socio-environmental exposure datasets, lies the potential for ecological fallacy, by assuming that associations observed at the community level universally hold for individuals residing within such areas. Whilst ecological studies cannot be used as substitutes for individual correlation studies, this does not indicate that ecological studies are etiologically useless, rather ecological variables are to be viewed as a necessity in the examination of structural, contextual, and sociological influences of disease development (Schwartz 1994). Thus, ecological studies are useful epidemiologic tools for public health surveillance where careful interpretation of their limitations and results has occurred. As such, ecological studies often assist in the generation of hypotheses but rarely provide a strong test of a causal hypothesis. In particular, ecological limitations are well documented throughout the research project, concerning the use of healthy lifestyle choices constructed from modelled outputs, and how inferences for ethnic minority derive from a small proportion of community residents.

Still, the research conducted within the project has successfully highlighted how structures of socio-environmental phenomenon individually and cooperatively influence respiratory outcomes, the latter of which is much harder to evaluate without the use of spatial modelling techniques for ecological data. In addition, through establishing critical distance-response thresholds, this research provides an intuitive foundation for future investigations on an individualistic basis targeting those considered most at risk. However, realistically health datum is unlikely to be made available at an individual level due to confidentiality clauses, and even if this was available, such outputs would still remain restricted by the resolution of socio-environmental databases. Here, the use of ecological datasets will have continued relevance within health surveillance, however in the future if highly localised health databases became available, it would become possible to verify structural findings between communities and neighbourhoods to minimise fallacy.

8.3. FURTHER RESEARCH

Whilst the ever evolving connections between social, environmental and health disparities experienced during childhood makes research into this field an on-going pursuit, eight specific future areas of research are highlighted that have arisen as a direct result of the current research. Initially, four improvements were suggested with respect to the general limitations found within the overall epidemiological evidence.

In the air quality literature it is widely documented that the fundamental strength of any epidemiological evidence, is that it evaluates health outcomes for real people, whom are living in normal environments, exposed to natural magnitudes of exposure. Yet, unfavourably there are at least four fundamental limitations inherent to the use of observational studies, where people reside in uncontrolled environments exposed to fluctuating doses of environmental stimuli. Firstly, the epidemiological evidence provides limited information with respect to those underlying biological mechanisms, despite biological plausibility existing within the historically coherent cascade of respiratory evidence. Still, the epidemiological evidence is clearly limited on this subject, with an element of reliance placed on laboratory and panel based investigations. As such, linkages between acute and chronic effects in terms of biological mechanisms have remained unclear. To an extent, this project has partially satisfied such limitations, through identifying common pathways between minor and severe respiratory outcomes recorded in the form of hospital admissions. Yet, the pathway between everyday respiratory health issues and hospital visitations remains largely unexplored. [1] Future research should therefore aim to address the early stages of respiratory deterioration, involving the association between diagnosed respiratory issues at a primary care level and hospital visitations during childhood.

A second basic limitation relates to coherence between ambient and personal exposures. As previously discussed, in correlating measures of pollution with census data, it was assumed that an individual's exposure occurred entirely within the relevant LLSOA. This is an assumption that future work should address with the aim of getting a better understanding of exposure. Still, children like the elderly favourably spend a majority of time close to their place of residence, with external exposures almost exclusively occurring at schools within the nearby vicinity. Here broadly similar exposures are expected to occur, but in reality, these micro-transitions in pollutants remain unaccounted for. To an extent, this is where anonymised census blocks may appear beneficial over precisely mapped patient outcomes, as geographically averaged outputs are less prone to errors in exposure caused by an individual's movements. As with the majority of preceding investigations, outdoor exposures were assumed representative of those values recorded indoors, and although children spent a

considerable amount of time outdoors this remains a secondary micro-environment of exposure to consider. However, to an extent the burdens found at home would relate to the quality of housing stock, and are thus likely quantified by the Carstairs Index of deprivation. Furthermore, accurate measurements of personal exposure to air pollutants for population-based studies are viewed as impractical and, for some applications unnecessary, considering that public policy and abatement strategies focus purely on ambient concentrations. Still, unlike the adult population, children's movements and thus additional exposures appear simpler, in that they are restricted to a singular common environment. **[2]** It is recommended that future investigations seek to implement broadly geo-link communities and attended schools, with pollutant magnitudes recorded there correcting residential exposures.

A third basic limitation of epidemiological studies involves the difficulty of disentangling independent effects or potential interactions between highly correlated risk factors. To a certain extent, the use of spatial techniques goes a long way in answering this question, through the incorporation or removal of influences based on spatial proximity, something which has been previously ignored by temporal techniques. Furthermore, all socio-environmental and health factors investigated here shown sense of spatial dependency, which when evaluated provided a clear image of the intertwined socio-environmental processes. **[3]** Still, future research should look towards the use of spatial-temporal models, to also account for temporal fluctuations. However, their application is currently limited by the frequency in which census data is recorded, in addition to changing the way such criteria are recorded over time; thus a stricter standardisation of datasets is required. Interestingly, a number of mid census datasets are starting to emerging, which could better facilitate such approaches in the future, potentially allowing for the correction of annual fluctuations.

A fourth basic limitation of epidemiological studies is the inability to explore in full, the relative health impacts of the various constituents of particulate pollution. Whilst biological evidence has traditionally suggested that combustion-source particulate pollution has a larger impact on respiratory health than comparable exposure to non-combustion related particles, rarely have the individual components been explored in a real-world setting; with the available monitoring equipment often unable to differentiating source specific components. Within this research, the use of emission inventories offers a partial solution, filtering out non-toxic background sources. Still, the 1x1km model resolution of this dataset unfavourably restricts the full inclusion of the various intra-urban microenvironments, yet on-the-other-hand beneficially provides a national coverage (model transferability). **[4]** For further validation, future research intends to construct a series of detailed local models, capturing micro-traffic outputs and atmospheric dispersion parameters. Even though

particulate dispersion is conceivably minimal for a landlocked city, and is thus unlikely to have significantly altered exposure gradients across LLSOA census blocks.

[5] In terms of external developments, this research demonstrates the valuable application of spatial modelling practices within the field of environmental epidemiology. Although we are aware of the health effects from PM_{10} and $PM_{2.5}$, a substantial amount of laboratory-based research has moved towards the health effects of nanoparticles at a cellular level. Urban monitoring networks are only just starting to accurately record levels of $PM_{2.5}$, with the widespread measurement of the extremely localised spatiotemporal ultrafine fraction likely appearing in the somewhat distant future. Until then a series of spatial techniques are ready and waiting to investigate this phenomenon at a demographic level.

With respect to the specific findings of the research project, three new areas of future research were proposed:

[6] Whilst effective dose-response relations were constructed across a range of intra-urban TPM_{10} emission levels after accommodating for spatial and social confounders, the precision of the constructed stimulus-response models remain debatable. For instance, with deprivation a rather complex relation was uncovered linking various health pathways to three specific subdivisions of the phenomenon; requiring further validation across wider range urban settings. Also for the various ethnic groups, bar the Indian populace, stimulus-response models only provide meaningful results where these groups for <10% of a community's residents. It is suggested that this methodology is rolled out to wider range of UK cities, using either a single model or meta-analysis approach, to better define such trends. Thus, the research presented here should be viewed as a preliminary strategy, displaying the potential application and value of spatial modelling practices.

[7] In spite of the frequency of 'Catarrhal Child Syndrome', its causes have previously remained uncertain, with anecdotal evidence suggesting the involvement of underlying social and genetic factors alongside viral activity. In exploring a spectrum of respiratory admission cases, this project has indicated that certain socio-environmental stimuli are accountable for an initial decline in children's respiratory health, to create a state favouring the onset of viral activity, which may further deteriorate respiratory health.. To-date, only one other epidemiological study exists, specifically exploring those interactions between exposure burdens and viral activity (Karr et al 2009). As such, additional research is recommended to confirm such findings.

[8] Finally, future research should follow on from those initial outputs provided by the localised Polluter Pays Principle analysis of Leicester. Primarily, this new research should explore the economic viability of the three proposed transport schemes including the: Completion of the cities outer-ring road, a Bus Gate Enforcement zone, and Environmental Zone. This research should also look at the short-term local transport schemes discussed here, in terms of formulating cost effective measures for those influential geographic communities. In the past carpooling has been by enlarge a unsuccessful measure, however through tailoring these measures to the needs of local communities, a beneficial response is more likely to be achieved. In addition, the overall methodology for this tangent of the research project may be further improved through the incorporation of emissions from public transport, to explore the full spectrum of environmental contributions made by each community.

REFERENCES

- AEA TECHNOLOGY, & DEFRA. (2010). *National Atmospheric Emissions Inventory: UK emission mapping by UNECE sectors (1x1km PM₁₀ emissions)*. Available via: <http://naei.defra.gov.uk/data/> [Accessed: January 2010]
- ALLOWAY, BJ., & AYRES, D.C. (1997). *Chemical principles of environmental pollution, 2nd edition*. London, UK: Blackie Academic & Professional (An Imprint of Chapman & Hall)
- AMARILLO, AC., & CARRERAS, HA. (2012). The effect of airborne particles and weather conditions on pediatric respiratory infections in Cordoba, Argentina. *Environmental Pollution*. **170**, pp.217-221
- ANDERSON, HR., BAILEY, PA., COOPER, JS., & PALMER, JC. (1981). Influence of morbidity, illness label, and social, family, and health service factors on drug treatment of childhood asthma. *The Lancet*. **318**(8254), pp.1030-1032
- ANDERSON, HR., ATKINSON, RW., PEACOCK, JL., MARSTON, L., & KONSTANTINOU, K. (2004). *Meta-analysis of time-series studies and panel studies of particulate matter (PM) and ozone (O₃): Report of a WHO Task Group*. Copenhagen, DEN: World Health Organization Regional Office for Europe:
- ANDERSSON, M., MODIG, L., HEDMAN, L., FORSBERG, B., & RONMARK, E. (2011). Heavy vehicle traffic is related to wheeze among schoolchildren: A population-based study in an area with low traffic flows. *Environmental Health*, **10**(91)
- ANSELIN, L. (1993). Exploratory Spatial Data Analysis and Geographic Information Systems. In: *DOSES/Eurostat Workshop on New Tools for Spatial Analysis*, November 18-20th, 1993, Lisbon, Portugal,
- ANSELIN, L. (1995). Local Indicators of Spatial Association: LISA. *Geographical Analysis*. **27**(2), pp.93-115
- ARBIA, G., & FINGLETON, B. (2008). New spatial econometric techniques and applications in regional science. *Papers in Regional Science*. **87**(3), pp.311-317
- ATKINSON, RW., ANDERSON, HR., SUNYER, J., AYRES, J., BACCINI, M., VONK, JM., BOUMGHAR, A., FORASTIERE, F., FORSBERG, B., TOULOUMI, G., SCHWARTZ, J., & KATSOUYANNI, K. (2001). Acute effects of particulate air pollution on respiratory admissions: Results from APHEA 2. *American Journal of Respiratory and Critical Care Medicine*. **164**(10), pp.1860-1866
- ATKINSON, RW., ANDERSON, HR., SUNYER, J., AYRES, J., BACCINI, M., VONK, JM., BOUMGHAR, A., FORASTIERE, F., FORSBERG, B., TOULOUMI, G., SCHWARTZ, J., & KATSOUYANNI, K. (2003). Acute effects of particulate air pollution on respiratory admissions. In: *Revised Analyses of Time-Series Studies of Air Pollution and Health. Special Report 14*. Boston, USA: Health Effects Institute
- AUDIT COMMISSION. (2010). *Improving data quality in the NHS: Annual report on the PbR assurance programme*. Available via: <http://www.auditcommission.gov.uk/2010/08/improving-data-quality-in-the-nhs-2010/> [Accessed: September 2011]

- REFERENCES -

- AVOL, EL., LINN, WS., SHAMOO, DA., VENET, TG., & HACKNEY JD. (1983). Acute Respiratory Effects of Los Angeles Smog in Continuously Exercising Adults. *Journal of the Air Pollution Control Association*. **33**(11), pp.1055-1060
- BAE, CHC. (1997). The equity impacts of Los Angeles' air quality policies. *Environment and Planning A*. **29**(9), pp.1563-1584
- BARBUJANI, G., ODEN, NL., & SOKAL, RR. (1989). Detecting regions of abrupt change in maps of biological variables. *Systematic Zoology*. **38**(4), pp.376-389
- BARCELO, MA., SAEZ, M., & SAURINA, C. (2009). Spatial variability in mortality inequalities, socioeconomic deprivation, and air pollution in small areas of the Barcelona Metropolitan Region, Spain. *Science of the Total Environment*. **407**(21), pp.5501-5523
- BARNETT, AG., WILLIAMS, GM., SCHWARTZ, J., NELLER, AH., BEST, TL., PETROESCHEVSKY, AL., & SIMPSON, RW. (2005). Air pollution and child respiratory health: A case-crossover study in Australia and New Zealand. *American Journal of Respiratory and Critical Care Medicine*. **171**(11), pp.1272-1278
- BATES, DV. (1999). Introduction. In: S.T. Holgate, J.M. Samet, H.S. Koren, R.L. Maynard (Ed.) *Air pollution and health*. London: Academic Press, pp.1-4
- BECKER, S., & SOUKUP, JM. (1999). Exposure to urban air particulates alters the macrophage-mediated inflammatory response to respiratory viral infection. *Journal of Toxicology and Environmental Health, Part A*. **57**(7), pp.445-457
- BENNETT, WD., & ZEMAN, KL. (1998). Deposition of fine particles in children spontaneously breathing at rest. *Inhalation Toxicology*. **10**(9), pp.831-842
- BESAG, J., & NEWELL, J. (1991). The detection of clusters in rare diseases. *Journal of the Royal Statistical Society, Series A*. **154**, pp.143-155
- BETTER HEALTH. (2011). *Tobacco use among minority ethnic populations and cessation interventions*. Race Equality Foundation. Available via: www.better-health.org.uk/sites/default/files/briefings/downloads/health-brief22_0.pdf[Accessed: May 2012]
- BEUTHER, DA., & SUTHERLAND, ER. (2007). Overweight, obesity, and incident asthma: A meta-analysis of prospective epidemiologic studies. *American Journal of Respiratory and Critical Care Medicine*, **175**(7), pp.661-666
- BIOMEDWARE. (2011). *BoundarySeer 1.3.13*, Ann Arbor, USA
- BIOMEDWARE. (2012). *SpaceStat Version 3.5.6*, Ann Arbor, USA
- BIVAND, R., & YU, D. (2012.) SPGWR: Geographically weighted regression. [R] Package Version 0.6-14. Vienna, AUT: [R] Foundation for Statistical Computing
- BLAIR, J., JOHNSON, K., & CARRUTHERS, D. (2004). *Source Apportionment for London using ADMS-Urban*. Cambridge Environmental Research Consultants Ltd (CERC), FM489/R3/04

- BLANE, D., HARDING, S., & ROSATO, M. (1999). Does social mobility affect the size of the socioeconomic mortality differential? Evidence from the Office for National Statistics Longitudinal Study. *Journal of the Royal Statistical Society, Series A (Statistics in Society)*. **162**(1), pp.59-70
- BONNEY, R., & LE-GOFF, W. (2007). Leicester's cultural diversity in the context of the British debate on multiculturalism. *International Journal of Diversity in Organisations*. **6**
- BOUBEL, RW., FOX, DL., TURNER, BD., & STERN, AC. (1994). *Fundamentals of air pollution, 3rd edition*. London, UK: Elsevier Academic Press
- BOULEY, G., AZOULAY-DUPUIS, E., & GAUDEBOUT, C. (1986). Impaired acquired resistance of mice to klebsiella pneumoniae infection induced by acute NO₂ exposure. *Environmental Research*. **41**(2), pp.497-504
- BOWEN, W. (2002). An analytical review of environmental justice research: What do we really know? *Environmental Management*. **29**(1), pp.3-15
- BOYCE, JK. (2007). Is inequality bad for the environment? In: R.C. Wilkinson, W.R. Freudenburg (Ed.) *Equity and the Environment [Research in Social Problems and Public Policy, Volume 15]*. Bingley, UK: Emerald Group Publishing Ltd, pp.267-288
- BRAUER, M., HOEK, G., VAN-VLIET, P., MELIEFSTE, K., FISCHER, PH., WIJGA, A., KOOPMAN, LP., NEIJENS, HJ., GERRITSEN, J., KERKHOF, M., HEINRICH, J., BELLANDER, T., & BRUNEKREEF, B. (2002). Air pollution from traffic and the development of respiratory infections and asthmatic and allergic symptoms in children. *American Journal of Respiratory and Critical Care Medicine*. **166**(8), pp.1092-1098
- BRIGGS, DJ., DE HOOGH, C., GULLIVER, J., WILLS, J., ELLIOTT, P., KINGHAM, S., & SMALLBONE, K. (2000). A regression-based method for mapping traffic-related air pollution: Application and testing in four contrasting urban environments. *Science of the Total Environment*. **253**(1-3), pp.151-167
- BROOK, RD., & RAJAGOPALAN, S. (2009). Particulate matter, air pollution, and blood pressure. *Journal of the American Society of Hypertension*. **3**(5), pp.332-350
- BROWN, H., & PRESCOTT, R. (2006). *Applied mixed models in medicine, 2nd edition*. Chichester, UK: John Wiley & Sons Ltd
- BROWN, JS., BATESON, TF., & MCDONNELL, WF. (2008). Effects of exposure to 0.06 ppm Ozone on FEV₁ in humans: A secondary analysis of existing data. *Environmental Health Perspectives*. **116**(8), pp.1023-1026
- BRUNEKREEF, B., DOCKERY, DW., & KRZYZANOWSKI, M. (1995). Epidemiologic studies on short-term effects of low levels of major ambient air pollution components. *Environmental Health Perspectives*. **103**(2), pp.3-13
- BRUNEKREEF, B., JANSSEN, NA., DE HARTOG, J., HARSSEMA, H., KNAPE, M., VAN-VLIET, P. (1997). Air pollution from truck traffic and lung function in children living near motorways. *Epidemiology*. **8**(3), pp.298-303

- REFERENCES -

- BUCKLEY, RD., & LOOSLE, CG. (1969) Effects of nitrogen dioxide inhalation on germfree mouse lung. *Archives of Environmental Health*. **18**(4), pp.588-595
- BURRA, T., JERRETT, M., BURNETT, R., & ANDERSON, M. (2002). Conceptual and practical issues in the detection of local disease clusters: A study of mortality in Hamilton, Ontario. *The Canadian Geographer*, **6**(2), pp.160-171
- CAMPBELL, SE., CAMPBELL, MK., GRIMSHAW, JM., & WALKER, AE. (2001). A systematic review of discharge coding accuracy. *Journal of Public Health*. **23**(3), pp.205-211
- CAPPUCCIO, FP., COOK, DG., ATKINSON, RW., & WICKS, PD. (1998). The Wandsworth Heart and Stroke Study: A population-based survey of cardiovascular risk factors in different ethnic groups. *Nutrition Metabolism and Cardiovascular Diseases*, **8**(6), pp.371-385
- CARSON, RL. (1963). *Silent Spring*. New York, USA: Houghton Mifflin Company (40th Anniversary Edition Reprint, 2002)
- CARSTAIRS, V., & MORRIS, R. (1991). *Deprivation and health in Scotland*. Aberdeen, UK: Aberdeen University Press
- CASTRANOVA, V., VALLYATHAN, V., RAMSEY, DM., MCLAURIN, JL., PACK, D., LEONARD, S., BARGER, MW., MA, JYC, DALAL, NS., & TEASS, A. (1997). Augmentation of pulmonary reactions to quartz inhalation by trace amounts of iron-containing particles. *Environmental Health Perspectives*. **105**(5), pp.1319-1324
- CESARONI, G., BOOGAARD, H., JONKERS, S., PORTA, D., BADALONI, C., CATTANI, G., FORASTIERE, F., & HOEK, G. (2012). Health benefits of traffic-related air pollution reduction in different socioeconomic groups: The effect of low-emission zoning in Rome. *Occupational and Environmental Medicine*. **69**(2), pp.133-139
- CHAKRABORTY, J. (2009). Automobiles, air toxics, and adverse health risks: Environmental inequities in Tampa Bay, Florida. *Annals of the Association of American Geographers*. **99**(4), pp.674-697
- CHANG, J., DELFINO, RJ., GILLEN, D., TJOA, T., NICKERSON, B., & COOPER, D. (2009). Repeated respiratory hospital encounters among children with asthma and residential proximity to traffic. *Occupational and Environmental Medicine*. **66**(2), pp.90-98
- CHEN, YC., DONG, GH., LIN, KC., & LEE, YL. (2013). Gender difference of childhood overweight and obesity in predicting the risk of incident asthma: A systematic review and meta-analysis. *Obesity Reviews*, **14**(3), pp.222-231
- CHU, C-M., TSAI, B-W., & CHANG, K-T. (2009). Integrating Decision Tree and Spatial Cluster Analysis for Landslide Susceptibility Zonation. *World Academy of Science, Engineering and Technology*. **59**, pp.479-483
- CICCONE, G., FORASTIERE, F., AGABITI, N., BIGGERI, A., BISANTI, L., CHELLINI, E., CORBO, G., DELL'ORCO, V., DALMASSO, P., VOLANTE, TF., GALASSI, C., PIFFER, S., RENZONI, E., RUSCONI, F., SESTINI, P., & VIEGI, G. (1998). Road traffic and adverse respiratory effects in children. *Occupational and Environmental Medicine*. **55**(11), pp.771-778

- CODY, RP., WEISEL, CP., BIRNBAUM, G., & LIOY, PJ. (1992). The effect of ozone associated with summertime photochemical smog on the frequency of asthma visits to hospital emergency departments. *Environmental Research*. **58**(2), pp.184-194
- COLLS, J. (2002). *Air Pollution*, 2nd edition. London, UK: Taylor & Francis Group
- COLVILE, RN., HUTCHINSON, EJ., MINDELL, JS., & WARREN, RF. (2001). The transport sector as a source of air pollution. *Atmospheric Environment*. **35**(9), pp.1537-1565
- COMMITTEE ON THE MEDICAL EFFECTS OF AIR POLLUTANTS. (COMEAP 1998). *Quantification of the effects of air pollution on health in the United Kingdom*. London, UK: Department of Health, Her Majesty's Stationary Office
- COMMITTEE ON THE MEDICAL EFFECTS OF AIR POLLUTANTS. (COMEAP 2006) *Cardiovascular disease and air pollution*. London, UK: Department of Health, Her Majesty's Stationary Office
- COMMITTEE ON THE MEDICAL EFFECTS OF AIR POLLUTANTS. (COMEAP 2009). *Long-Term Exposure to Air Pollution: Effect on Mortality*. London, UK: Department of Health, Her Majesty's Stationary Office
- COMMITTEE ON THE MEDICAL EFFECTS OF AIR POLLUTANTS. (COMEAP 2010). *The Mortality Effects of Long-Term Exposure to Particulate Air Pollution in the United Kingdom*. London, UK: Department of Health, Her Majesty's Stationary Office
- CENTRE ON MIGRATION, POLICY & SOCIETY. (COMPAS 2006). *Refugees and other new migrants: A review of the evidence on successful approaches to integration*. London, UK: Home Office, Her Majesty's Stationary Office
- CONSUMERS ASSOCIATION. (2013). *Buying a car, petrol or diesel?*. London: Which Ltd. Available via: <http://which.co.uk/cars/choosing-a-car/buying-a-car/petrol-or-diesel/> [Accessed: January 2013]
- COOPER, AR., PAGE, AS., WHEELER, BW., HILLSDON, M., GRIEW, P., & JAGO, R. (2010). Patterns of GPS measured time outdoors after school and objective physical activity in English children: The PEACH project. *International Journal of Behavioural Nutrition and Physical Activity*. **7**(31)
- CROUSE, DL., ROSS, NA., & GOLDBERG, MS. (2009). Double burden of deprivation and high concentrations of ambient air pollution at the neighbourhood scale in Montreal, Canada. *Social Science & Medicine*. **69**(6), pp.971-981
- CUMMING, G., & SEMPLE, SJ. (1980). *Disorders of the respiratory system*, 2nd edition. Oxford, UK: Blackwell Scientific Publication
- CUTTER, SL. (1995). Race, class and environmental justice. *Progress in Human Geography*. **19**(1), pp.111-122
- DEBENHAM, J., CLARKE, G., & STILLWELL, J. (2001). Deriving supply-side variables to extend geodemographic classification. In: *12th European Colloquium on Quantitative and Theoretical Geography*, September 7-11th, 2001, St-Valery-en-Caux, France

- REFERENCES -

- DEPARTMENT FOR COMMUNITIES & LOCAL GOVERNMENT. (DCLG 2006). *Improving Opportunity, Strengthening Society: Race Equality in Public Services*. Coventry, UK: Institute of Community Cohesion
- DEPARTMENT FOR ENVIRONMENT, FOOD AND RURAL AFFAIRS. (DEFRA 2001). *The air quality strategy for England, Scotland, Wales and Northern Ireland: Working together for clean air [CM4548]*. London, UK: Department of Health, Her Majesty's Stationary Office
- DEPARTMENT FOR ENVIRONMENT, FOOD AND RURAL AFFAIRS. (DEFRA 2006). *Air quality and social deprivation in the UK: An environmental inequalities analysis*. Didcot, UK: AEA Technology
- DEPARTMENT FOR ENVIRONMENT, FOOD AND RURAL AFFAIRS. (DEFRA 2007). *The Air Quality Strategy for England, Scotland, Wales and Northern Ireland: Volume 2 [CM4548]*. London, UK: Department of Health, Her Majesty's Stationary Office
- DEGUEN, S., & ZMIROU-NAVIER, D. (2010). Social inequalities resulting from health risks related to ambient air quality: A European review. *European Journal of Public Health*. **20**(1), pp.27–35
- DEPARTMENT FOR TRANSPORT. (DfT 1997). *National road traffic forecasts, Great Britain*. Available via: www.dft.gov.uk/pgr/economics/ntm/ntmdatasources/nrtf1997/ [Accessed: September 2009]
- DEPARTMENT FOR TRANSPORT. (DfT 2004). *The future of transport: A network for 2030*. Available via: www.dft.gov.uk/about/strategy/whitepapers/previous/fot/ [Accessed: September 2009]
- DEPARTMENT FOR TRANSPORT. (DfT 2009). Trip End Model Presentation Program (TEMPro) Version 6.1.
- DEPARTMENT FOR TRANSPORT. (DfT 2010). *National Travel Survey 2009: Travel by age and gender*. Available via: www.dft.gov.uk/statistics/releases/national-travel-survey-2010 [Accessed: January 2011]
- DEPARTMENT FOR TRANSPORT. (DfT 2013). *National Travel Survey 2012: Statistical Release*. Available via: <https://www.gov.uk/government/publications/national-travel-survey-2012> [Accessed: August 2013]
- DOCKERY, DW., POPE, CA., XU, X., SPENGLER, JD., WARE, JH., FAY, ME., FERRIS, BG., & SPEIZER, FE. (1993). An association between air pollution and mortality in six U.S. cities. *The New England Journal Of Medicine*. **329**(24), pp.1753-1759
- DOCKERY, DW. (2001). Epidemiologic Evidence of Cardiovascular Effects of Particulate Air Pollution. *Environmental Health Perspectives*. **109**(4), pp.483-486
- DOMINICI, F., MCDERMOTT, A., ZEGER, SL., & SAMET, JM. (2002). On the use of generalized additive models in time-series studies of air pollution and health. *American Journal of Epidemiology*. **156**(3), pp.193-203

- DOMINICI, F., MCDERMOTT, A., DANIELS, M., ZEGER, S.L., & SAMET, J.M. (2003). Mortality among residents of 90 cities. In: *Revised Analyses of Time-Series Studies of Air Pollution and Health: Special Report 14*. Boston, USA: Health Effects Institute
- DONALDSON, K., LI, X.Y., & MACNEE, W. (1998). Ultrafine particle mediated lung injury. *Journal of Aerosol Science*. **29**(5-6), pp.553-560
- DONALDSON, K., & MACNEE, W. (1998). The mechanisms of lung injury caused by PM₁₀. In: R.E Hester & R.M Harrison (Ed.) *Air pollution and health*. Letchworth, UK: The Royal Society of Chemistry
- DONALDSON, K., & MACNEE, W. (2001). Potential mechanisms of adverse pulmonary and cardiovascular effects of particulate air pollution (PM₁₀). *International Journal of Hygiene and Environmental Health*. **203**(5-6), pp.411-415
- DONATI, D., CELLESI, C., ROSSOLINI, A., LORUSSO, V., MOSCHETTINI, D., AMATO, T., & VALENSIN, P.E. (1998). Serological diagnosis of respiratory viral infections: A five-year study of hospitalised patients. *New Microbiologica*. **21**(4): pp.365-374
- DUHME, H., WEILAND, S.K., KEIL, U., KRAEMER, B., SCHMID, M., STENDER, M., & CHAMBLESS, L. (1996). The association between self-reported symptoms of asthma and allergic rhinitis and self-reported traffic density on street of residence in adolescents. *Epidemiology*. **7**(6), pp.578-582
- DURAN-TAULERIA, E., RONA, R.J., CHINN, S., & BURNEY, P. (1996). Influence of ethnic group on asthma treatment in children in 1990-91: National cross sectional study. *BMJ*. **313**(7050), pp.148-152
- DUVAL, S., & TWEEDIE, R. (2000). Trim and fill: A simple funnel-plot-based method of testing and adjusting for publication bias in meta-analysis. *Biometrics*. **56**(2), pp.455-463
- EUROPEAN COMMISSION. (EC 1980). *Council Directive 80/779/EEC. On air quality limit values and guide values for sulphur dioxide and suspended particulates*. Available via: <http://eur-lex.europa.eu/LexUriServ/LexUriServ.do?uri=OJ:L:1980:229:0030:0048:EN:PDF> [Accessed: May 2010]
- EUROPEAN COMMISSION. (EC 1985a). *Council Directive 85/203/EEC. On air quality standards for nitrogen dioxide*. Available via :<http://eurlex.europa.eu/LexUriServ/LexUriServ.do?uri=OJ:L:1985:087:0001:0007:EN:PDF> [Accessed: May 2010]
- EUROPEAN COMMISSION. (EC 1985b). *Council Directive 85/210/EEC. On the approximation of the laws of the Member States concerning the lead content of petrol*. Available via:<http://eur-lex.europa.eu/LexUriServ/LexUriServ.do?uri=OJ:L:1985:096:0025:0029:EN:PDF> [Accessed: May 2010]
- EUROPEAN COMMISSION. (EC 1999). *Council Directive 1999/30/EC. Relating to limit values for sulphur dioxide, nitrogen dioxide and oxides of nitrogen, particulate matter and lead in ambient air [First Daughter Directive]*. Available via: <http://eur-lex.europa.eu/LexUriServ/LexUriServ.do?uri=OJ:L:1999:163:0041:0060:EN:PDF> [Accessed: May 2010]

- REFERENCES -

- EUROPEAN COMMISSION. (EC 2005). *Environment fact sheet: Moving towards clean air for Europe*. Available via: <http://ec.europa.eu/environment/pubs/pdf/factsheets/air> [Accessed: May 2010]
- EUROPEAN COMMISSION. (EC 2006). *Development of a methodology to assess population exposed to high levels of noise and air pollution close to major transport infrastructure*. London, UK: Entec Limited
- EUROPEAN COMMISSION. (EC 2008). *Directive 2008/50/EC. On ambient air quality and cleaner air for Europe*. Available via: <http://eurlex.europa.eu/LexUriServ/LexUriServ.do?uri=OJ:L:2008:152:0001:0044:EN:PDF> [Accessed: May 2010]
- EUROPEAN COMMISSION. (EC 2012). *Environment: many Europeans still exposed to harmful air pollutants. Press Release IP/12/1002*. Available via: http://europa.eu/rapid/press-release_IP-12-1002_en.htm [Accessed: January 2013]
- EDINA DIGIMAP. (2012). *Ordnance Survey 1:50,000 Landranger Map: Leicestershire*. EDINA Data Library Services, University of Edinburgh. Available via: <http://edina.ac.uk/digimap> [Accessed: March 2012]
- EGGER, M., DAVEY-SMITH, G., SCHNEIDER, M., & MINDER, C. (1997). Bias in meta-analysis detected by a simple, graphical test. *BMJ*. **315**(7109), pp.629–634
- EHRlich, R., SILVERSTEIN, E., MAIGETTER, R., & FENTERS, JD. (1975). Immunologic response in vaccinated mice during long-term exposure to nitrogen dioxide. *Environmental Research*. **10**(2), pp.217-223
- EQUALITY AND HUMAN RIGHTS COMMISSION. (EHRC 2011). *How fair is Britain? Equality, Human Rights and Good Relations in 2010: The First Triennial Review*. Available via: www.equalityhumanrights.com/uploaded_files/triennial_review/how_fair_is_britain_-_complete_report.pdf [Accessed: December 2011]
- ENVIRONMENT AGENCY. (2004). Position Statement: addressing environmental inequalities. Available via: http://www.environmentagency.gov.uk/static/documents/Research/ca221final_888457.pdf [Accessed: December 2010]
- ENVIRONMENTAL AUDIT COMMITTEE. (2010). *Air Quality: Volume I*. House of Commons, London: The Stationery Office Limited, HC 229-I
- ENVIRONMENTAL SYSTEMS RESEARCH INSTITUTE. (ESRI 2009). *ArcGIS Desktop: Release 9.3.1*. Redlands, USA
- EXPERT PANEL ON AIR QUALITY STANDARDS. (EPAQS 1995). *Particles*. London, UK: DEFRA Publications
- FARHAT, SCL., PAULO, RLP., SHIMODA, TM., CONCEICAO, GMS., LIN, CA., BRAGA, ALF., WARTH, MNP., & SALDIVA, PHN. (2005). Effect of air pollution on pediatric respiratory emergency room visits and hospital admissions. *Brazilian Journal of Medical and Biological Research*. **38**(2), pp.227-235

- FEITELSON, E. (2002). Introducing environmental equity dimensions into the sustainable transport discourse: Issues and pitfalls. *Transportation Research Part D*. **7**(2), pp.99-118
- FITCH, JM. (1960). In Defence of the City. *Proceedings of the Academy of Political Science*, **27**(1), pp.2-11
- FORTIN, MJ., & DRAPEAU, P. (1995). Delineation of ecological boundaries: Comparison of approaches and significance tests. *OIKOS*, **72**(3), pp.323-332
- FOTHERINGHAM, AS., CHARLTON, ME. & BRUNSDON, C. (1998). Geographically weighted regression: A natural evolution of the expansion method for spatial data analysis. *Environment and Planning A*. **30**(11), pp.1905-1927
- FOTHERINGHAM, AS., BRUNSDON, C., & CHARLTON, ME. (2002). *Geographically weighted regression: The analysis of spatially varying relationships*. Chichester, UK: Wiley
- FRUIN, S., WESTERDAHL, D., SAX, T., SIOUTAS, C., & FINE, PM. (2008). Measurements and predictors of on-road ultrafine particle concentrations and associated pollutants in Los Angeles. *Atmospheric Environment*. **42**(2), pp.207-219
- FRY, J. (1966). Profiles of disease: A study in the natural history of common diseases. London, UK: E.& S. Livingstone Ltd
- FRY, J., & SANDLER, G. (1993). Common diseases: Their nature, prevalence and care / John Fry and Gerald Sandler. London, UK: Kluwer Academic Publishers
- FUJII, T., HOGG, JC., KEICHO, N., VINCENT, R., VAN-EEDEN SF., & HAYASHI, S. (2003). Adenoviral E1A modulates inflammatory mediator expression by lung epithelial cells exposed to PM₁₀. *American Journal of Physiology - Lung Cellular and Molecular Physiology*. **284**(2), L.290-L.297
- FUSCO, D., FORASTIERE, F., MICHELOZZI, P., SPADEA, T., OSTRO, B., ARCA, M., & PERUCCI, CA. (2001). Air pollution and hospital admissions for respiratory conditions in Rome, Italy. *European Respiratory Journal*. **17**(6), pp.1143-1150
- GARSHICK, E., LADEN, F., HART, JE., & CARON, A. (2003). Residence near a major road and respiratory symptoms in US veterans. *Epidemiology*. **14**(6), pp.728-736
- GAUDERMAN, WJ., AVOL, E., LURMANN, F., KUENZLI, N., GILLILAND, F., PETERS, J., & MCCONNELL, R. (2005). Childhood asthma and exposure to traffic and nitrogen dioxide. *Epidemiology*. **16**(6), pp.737-743
- GAUDERMAN, WJ., VORA, H., MCCONNELL, R., BERHANE, K., GILLILAND, F., THOMAS, D., LURMANN, F., AVOL, E., KUNZLI, N., JERRETT, M., PETERS, J. (2007). Effect of exposure to traffic on lung development from 10 to 18 years of age: A cohort study. *The Lancet*. **369**(9561), pp.571-577
- GEHRING, U., CYRYS, J., SEDLMEIR, G., BRUNEKREEFZ, B., BELLANDER, T., FISCHER, P., BAUER, CP., REINHARDT, D., WICHMANN, HE., & HEINRICH, J. (2002). Traffic-related air pollution and respiratory health during the first 2 years of life. *European Respiratory Journal*. **19**(4), pp.690-698

- GETIS, A., & ORD, JK. (1992). The analysis of spatial association by use of distance statistics. *Geographical Analysis*. **24**, pp.189–206
- GIELEN, MH., VAN-DER-ZEE, SC., VAN-WIJNEN, JH., VAN-STEEN, CJ., & BRUNEKREEF, B. (1997). Acute effects of summer air pollution on respiratory health of asthmatic children. *American Journal of Respiratory and Critical Care Medicine*. **155**(6), pp.2105-2108
- GILBERT, A., & CHAKRABORTY, J. (2011). Using geographically weighted regression for environmental justice analysis: Cumulative cancer risks from air toxics in Florida. *Social Science Research*. **40**(1), pp.273-286
- GILMOUR, PS., BROWN, DM., LINDSAY, TG., BESWICK, PH., MACNEE, W., & DONALDSON, K. (1996). Adverse health effects of PM₁₀ particles: Involvement of iron in generation of hydroxyl radical. *Occupational and Environmental Medicine*. **53**(12), pp.817-822
- GILMOUR, PS., RAHMAN, I., HAYASHI, S., HOGG, JC., DONALDSON, K., & MACNEE, W. (2001). Adenoviral E1A primes alveolar epithelial cells to PM₁₀-induced transcription of interleukin-8. *American Journal of Physiology - Lung Cellular and Molecular Physiology*. **281**(3), L.598-L.606
- GRANTZ, DA., & SHRESTHA, A. (2005). Ozone reduces crop yields and alters competition with weeds such as yellow nutsedge. *California Agriculture*. **59**(2), pp.137-143
- GRASSIAN, VH., O'SHAUGHNESSY, PT., ADAMCAKOVA-DODD, A., PETTIBONE, JM., & THORNE, PS. (2007). Inhalation Exposure Study of Titanium Dioxide Nanoparticles with a Primary Particle Size of 2-5nm. *Environmental Health Perspectives*. **115**(3), pp.397-402
- GREAT BRITAIN. (1845). *Railways Clauses Consolidation Act 1845: Chapter 20, Victoria*. Available via: <http://legislation.gov.uk/ukpga/Vict/8-9/20/enacted> [Accessed: July 2012]
- GREAT BRITAIN. (1847). *Towns Improvement Clauses Act 1847: Chapter 34 (10 & 11), Victoria*. Available via: <http://legislation.gov.uk/ukpga/Vict/10-11/34> [Accessed: July 2012]
- GREAT BRITAIN. (1956). *Clean Air Act 1956: Chapter 52, Elizabeth II*. Available via: <http://legislation.gov.uk/ukpga/Eliz2/4-5/52/contents/enacted> [Accessed: July 2012]
- GREGORY, IN. (2009). Comparisons between geographies of mortality and deprivation from the 1900's and 2001: Spatial analysis of census and mortality statistics. *British Medical Journal*. **339**(7722), pp.676-679
- GRIFFIN, RD. (2006). *Principles of air quality management: Second edition*. Boca Raton, USA: CRC Press, Taylor & Francis Group
- GRIGG, J. (2009). Particulate Matter Exposure in Children. *Proceedings of the American Thoracic Society*. **6**(7), pp.564-569
- GOLD, DR., DAMOKOSH, AI., POPE, CA., DOCKERY, DW., MCDONNELL, WF., SERRANO, P., RETAMA, A., CASTILLEJOS, M. (1999). Particulate and ozone pollutant effects on the respiratory function of children in southwest Mexico City. *Epidemiology*. **10**(1), pp.8-16

- GOOGLE MAPS UK. (2012) *Highways Agency Live traffic map of Leicester*. Available via: <http://maps.google.co.uk/> [Accessed: September 2012]
- GOOVAERTS, P. (1997). *Geostatistics for natural resources evaluation*. Applied Geostatistics Series. New York, USA: Oxford University Press
- GOOVAERTS, P., AVRUSKIN, G., MELIKER, J., SLOTNICK, M., JACQUEZ, G., & NRIAGU, J. (2005). Geostatistical modelling of the spatial variability of arsenic in groundwater of southeast Michigan. *Water Resources research*. **41**(7)
- GORDON, SB., & READ, RC. (2002). Macrophage defences against respiratory tract infections. *British Medical Bulletin*. **61**, pp.45-61
- GOTEBORG'S STAD TRAFIKKONTORET. (2006). Assessment of the Environmental Zone in Goteborg: A report for the Traffic & Public Transport Authority of the City of Goteborg. Available via: www2.trafikkontoret.goteborg.se/resourcelibrary/Utv%C3%A4rdering%20MZ%2010%20%C3%A5r%20ENG.pdf [Accessed: November 2012]
- HAHON, N., BOOTH, JA., GREEN, F., & LEWIS, TR. (1985). Influenza virus infection in mice after exposure to coal dust and diesel engine emissions. *Environmental Research*. **37**(1), pp.44-60
- HAJAT, S., HAINES, A., GOUBET, S., ATKINSON, R., & ANDERSON, H. (1999). Association of air pollution with daily GP consultations for asthma and other lower respiratory conditions in London. *Thorax*. **54**(7), pp.597–605
- HAJAT, S., ANDERSON, H., ATKINSON, R., HAINES, A., & SEATON, A. (2002). Effects of air pollution on general practitioner consultations for upper respiratory diseases in London. *Occupational and Environmental Medicine*. **59**(5), pp.294-299
- HALL, JV., WINER, AM., KLEINMAN, MT., LURMANN, FW., BRAJER, V., & COLOME, SD. (1992). Valuing the Health Benefits of Clean Air. *Science: New Series*. **255**(5046), pp.812-817
- HALL, KR. (2008). Comparing geographic boundaries in songbird demography data with vegetation boundaries: A new approach to evaluating habitat quality. *Environmental and Ecological Statistics*. **15**(4), pp.491–521
- HAMILTON, RF., WU, N., PORTER, D., BUFORD, M., WOLFARTH, M., & HOLIAN, A. (2009). Particle length-dependent titanium dioxide nanomaterials toxicity and bioactivity. *Particle and Fibre Toxicology*. **6**(35)
- HARRISON, RM. (2001). Air pollution: Sources, concentrations and measurements. In: *Harrison R.M (Ed.) Pollution: Causes, effects and control, Fourth edition*. The Royal Society of Chemistry: Cambridge, UK
- HARRISON, RM., JONES, AM., & LAWRENCE, RG. (2004). Major component composition of PM₁₀ and PM_{2.5} from roadside and urban background sites. *Atmospheric Environment*. **38**(27), pp.4531-4538
- Health Effects Institute. (HEI 2000). *Reanalysis of the Harvard Six Cities Study and the American Cancer Society Study of Particulate Air Pollution and Mortality*. Special Report 12

- HEALTH EFFECTS INSTITUTE. (HEI 2009). *Traffic-Related Air Pollution: A Critical Review of the Literature on Emissions, Exposure, and Health Effects*. Special Report 17
- HERNANDEZ-CADENA, L., TELLEZ-ROJO, MM., SANIN-AGUIRRE, LH., LACASANA-NAVARRO, M., CAMPOS, A., & ROMIEU, I. (2000). Relationship between emergency consultations for respiratory diseases and air pollution in Juarez City, Chihuahua. *Salud Pública de México*. **42**(4), pp.288-297
- HERNANDEZ-CADENA, L., BARRAZA-VILLARREAL, A., RAMIREZ-AGUILAR, M., MORENO-MACIAS, H., MILLER, P., CARBAJAL-ARROYO, LA., & ROMIEU, I. (2007). Infant morbidity caused by respiratory diseases and its relation with air pollution in Juarez City, Chihuahua, Mexico. *Salud Pública de México*. **49**(1), pp.27-36
- HERSHENBERG, JL. (2001). *Pursuing environmental justice through the courts: An overview of the process and why it has failed*. Institute for Policy & Economic Development, Technical reports paper 12, University of Texas. Accessed via: http://digitalcommons.utep.edu/iped_techrep/12 [Accessed: January 2013]
- HERTEL, O., JENSEN, SS., ANDERSEN, HV., PALMGREN, F., WAHLIN, P., SKOV, H., NIELSEN, IV., SORENSEN, M., LOFT, S., & RAASCHOU-NIELSEN, O. (2001). Human exposure to traffic pollution. Experience from Danish studies. *Pure and Applied Chemistry*. **73**(1), pp.137-145
- HIPP, JR., & LAKON, CM. (2010). Social disparities in health: Disproportionate toxicity proximity in minority communities over a decade. *Health & Place*. **16**(4), pp.674-683
- HODGSON, FC., & TURNER J. (2003). Participation not consumption: The need for new participatory practices to address transport and social exclusion. *Transport Policy*, **10**(4), pp.265–272
- HOEK, G., & BRUNEKREEF, B. (1994). Effects of low-level winter air pollution concentrations on respiratory health of Dutch children. *Environmental Research*. **64**(2), pp.136-150
- HOPTON, J., & HUNT, S. (1996). The health effects of improvements to housing: A longitudinal study. *Housing Studies*. **11**(2), pp.271-286
- IBEAS, A., CORDERA, R., DELL'OLIO, L., COPPOLA, P., & DOMINGUEZ, A. (2012). Modelling transport and real-estate values interactions in urban systems. *Journal of Transport Geography*. **24**, pp.370-382
- IBM CORPORATION. (2010). *IBM SPSS Statistics for Windows, Version 19.0*. Armonk, USA
- ILSI RISK SCIENCE INSTITUTE. (2000). The relevance of the rat lung response to particle overload for human risk assessment: a workshop consensus report. *Inhalation Toxicology*. **12**(1-2), pp.1-17
- JAASKELAINEN, A., PUSSINEN, J., NUUTINEN, O., SCHWAB, U., PIRKOLA, J., KOLEHMAINEN, M., JARVELIN, MR., & LAITINEN, J. (2011). Intergenerational transmission of overweight among Finnish adolescents and their parents: A 16-year follow-up study. *International Journal of Obesity*, **35**(10), pp.1289-1294

- JACOBSON, MZ., COLELLA, WG., & GOLDEN, DM. (2005). Cleaning the Air and Improving Health with Hydrogen Fuel-Cell Vehicles. *Science*. **308**, pp.1901-1905
- JACQUEZ, GM. (1995). The map comparison problem: Tests for the overlap of geographical boundaries. *Statistics in Medicine*. **14**(21-22), pp.2343-2361
- JACQUEZ, GM., & GREILING, DA. (2003). Geographic boundaries in breast, lung and colorectal cancers in relation to exposure to air toxics in Long Island, New York. *International Journal of Health Geographic's*. **2**(4)
- JAKAB, GJ. (1987). Modulation of pulmonary defence mechanisms by acute exposures to nitrogen dioxide. *Environmental Research*. **42**(1), pp.215-228
- JALALUDIN, BB., CHEY, T., O'TOOLE, BI., SMITH, WT., CAPON, AG., & LEEDER, SR. (2000). Acute effects of low levels of ambient ozone on peak expiratory flow rate in a cohort of Australian children. *International Journal of Epidemiology*. **29**(3), pp.549-557
- JANSSEN, NAH., BRUNEKREEF, B., VAN-VLIET, P., AARTS, F., MELIEFSTE, K., HARSSEMA, H., & FISCHER, P. (2003). The relationship between air pollution from heavy traffic and allergic sensitization, bronchial hyper-responsiveness, and respiratory symptoms in Dutch schoolchildren. *Environmental Health Perspectives*. **111**(12), pp.1512-1518
- JARTTI, T., LEHTINEN, P., VUORINEN, T., OSTERBACK, R., VAN-DEN-HOOGEN, B., OSTERHAUS, AD., & RUUSKANEN, O. (2004) Respiratory picornaviruses and respiratory syncytial virus as causative agents of acute expiratory wheezing in children. *Emerging Infectious Diseases*. **10**(6), pp.1095-1101
- JASPERS, I., CIENCEWICKI, JM., ZHANG, W., BRIGHTON, LE., CARSON, JL., BECK, MA., & MADDEN, MC. (2005). Diesel exhaust enhances influenza virus infections in respiratory epithelial cells. *Toxicological Sciences*. **85**(2), pp.990-1002
- JEPHCOTE, C., & CHEN, H. (2012) Environmental injustices of children's exposure to air pollution from road-transport within the model British multicultural city of Leicester: 2000-09. *Science of the Total Environment*. **414**, pp.140-151
- JEPHCOTE, C., & CHEN, H. (2013). Geospatial analysis of naturally occurring boundaries in road-transport emissions and children's respiratory health across a demographically diverse cityscape. *Social Science & Medicine*, **82**, pp.87-99
- JOHNSTON, SL., PATTEMORE, PK., SANDERSON, G., SMITH, S., LAMPE, F., JOSEPHS, L., SYMINGTON, P., O'TOOLE, S., MYINT, SH., TYRRELL, DA., & HOLGATE, ST. (1995). Community study of role of viral infections in exacerbations of asthma in 9-11 year old children. *BMJ*. **310**(6989), pp.1225-1229
- JONES, LL., HASHIM, A., MCKEEVER, T., COOK, DG., BRITTON, J., & LEONARDI-BEE, J. (2011). Parental and household smoking and the increased risk of bronchitis, bronchiolitis and other lower respiratory infections in infancy: Systematic review and meta-analysis. *Respiratory Research*, **12**(5)
- JOUSIMIES-SOMER, HR., SAVOLAINEN, S., & YLIKOSKI, JS. (1989). Comparison of the nasal bacterial floras in two groups of healthy subjects and in patients with acute maxillary sinusitis. *Journal of Clinical Microbiology*. **27**(12), pp.2736-2743

- KARLSSON, HL., GUSTAFSSON, J., CRONHOLM, P., & MOLLER, L. (2009). Size-dependent toxicity of metal oxide particles: A comparison between nano and micrometer size. *Toxicology Letters*. **188**(2), pp.112-118
- KARR, CJ., RUDRA, CB., MILLER, KA., GOULD, TR., LARSON, T., SATHYANARAYANA, S., & KOENIG, JQ. (2009). Infant exposure to fine particulate matter and traffic and risk of hospitalization for RSV bronchiolitis in a region with lower ambient air pollution. *Environmental Research*. **109**(3), pp.321-327
- KEIL, U., WEILAND, SK., DUHME, H., & CHAMBLESS, L. (1996). The International Study of Asthma and Allergies in Childhood (ISAAC): objectives and methods; results from German ISAAC centres concerning traffic density and wheezing and allergic rhinitis. *Toxicology Letters*. **86**(2-3), pp.99-103
- KENYON, S., LYONS, G., & RAFFERTY, J. (2002). Transport and social exclusion: investigating the possibility of promoting inclusion through virtual mobility. *Journal of Transport Geography*, **10**(3), pp.207–219
- KIM, JJ., HUEN, K., ADAMS, S., SMORODINSKY, S., HOATS, A., MALIG, B., LIPSETT, M., & OSTRO, B. (2008). Residential traffic and children's respiratory health. *Environmental Health Perspectives*. **116**(9), pp.1274-1279
- KINGHAM, S., PEARCE, J., & ZAWAR-REZA, P. (2007). Driven to injustice? Environmental justice and vehicle pollution in Christchurch, New Zealand. *Transportation Research Part D*. **12**(4), pp.254-263
- KRIGE, DG. (1966). Two-dimensional weighted moving average trend surfaces for ore evaluation. *Journal of the South African Institute of Mining and Metallurgy*. **66**, pp.13-38
- KUEHNI, CE., STRIPPOLI, MF., LOW, N., BROOKEW, AM., & SILVERMAN, M. (2007). Wheeze and asthma prevalence and related health-service use in white and south Asian pre-schoolchildren in the United Kingdom. *Clinical and Experimental Allergy*. **37**(12), pp.1738-1746
- KUNZLI, N., KAISER, R., MEDINA, S., STUDNICKA, M., CHANEL, O., FILLIGER, P., HERRY, M., HORAK, F., PUYBONNIEUX-TEXIER, V., QUENEL, P., SCHNEIDER, J., SEETHALER, R., VERGNAUD, JC., & SOMMER, H. (2002). Public-health impact of outdoor and traffic-related air pollution: a European assessment. *The Lancet*. **356**(9232), pp.795-801
- KUNZLI, N., BRIDEVAUX, PO., LIU, LS., GARCIA-ESTEBAN, R., SCHINDLER, C., GERBASE, MW., SUNYER, J., KEIDEL, D., & ROCHAT, T. (2009). Traffic-related air pollution correlates with adult-onset asthma among never-smokers. *Thorax*. **64**(8), pp.664-670
- KUSEL, MM., DE-KLERK, NH., HOLT, PG., KEBADZE, T., JOHNSTON, SL., & SLY, PD. (2006). Role of respiratory viruses in acute upper and lower respiratory tract illness in the first year of life: A birth cohort study. *The Pediatric Infectious Disease Journal*. **25**(8), pp.680-686
- LAKSHMAN, R., MCCONVILLE, A., HOW, S., FLOWERS, J., WAREHAM, N., & COSFORD, P. (2010). Association between area-level socioeconomic deprivation and a cluster of behavioural risk factors: cross-sectional, population-based study. *Journal of Public Health*. **33**(2), pp.234-245

- LAMBERT, AL., MANGUM, JB., DELORME, MP. & EVERITT, JI. (2003). Ultrafine carbon black particles enhance respiratory syncytial virus-induced airway reactivity, pulmonary inflammation, and chemokine expression. *Toxicological Sciences*. **72**(2), pp.339-346
- LAWLOR, DA., EBRAHIM, S., & SMITH, GD. (2004). Association between self-reported childhood socioeconomic position and adult lung function: findings from the British Women's Heart and Health Study. *Thorax*. **59**, pp.199-203
- LE, TG., NGO, L., MEHTA, S., VAN-DZUNG, D., THACH, TQ., VU, XD., NGUYEN, DT., & COHEN, A. (2012). Effects of short-term exposure to air pollution on hospital admissions of young children for acute lower respiratory infections in Ho Chi Minh City, Vietnam. *Health Effects Institute*, **169**, pp.5-72
- LEICESTER CITY COUNCIL. (LCC 2011). *Leicester's Local Transport Plan 2011-2026: "Planning for people not cars", Part A - The Transport Strategy*. Leicester, UK: Regeneration, Highways and Transportation
- LESAGE, JP., & PACE, RK. (2010) *The biggest myth in spatial econometrics*. Social Science Research Network. Available via: http://papers.ssrn.com/sol3/papers.cfm?abstract_id=1725503 [Accessed: July 2011]
- LEUNG, Y., MEI, C., & ZHANG, W. (2000) Statistical tests for spatial nonstationarity based on the geographically weighted regression model. *Environment and Planning A*. **32**(1), pp.9-32
- LEFKOWITZ, SS., MCGRATH, JJ., LEFKOWITZ, DL. (1986). Effects of NO₂ on immune responses. *Journal of Toxicology and Environmental Health*. **17**(2-3), pp.241-248
- LI, N., SIOUTAS, C., CHO, A., SCHMITZ, D., MISRA, C., SEMPFF, J., WANG, M., OBERLEY, T., FROINES, J., & NEL, A. (2003). Ultrafine Particulate Pollutants Induce Oxidative Stress and Mitochondrial Damage. *Environmental Health Perspectives*. **111**(4), pp.455-460
- LI, S., BATTERMAN, S., WASILEVICH, E., ELASAAD, H., WAHL, R., & MUKHERJEE, B. (2011). Asthma exacerbation and proximity of residence to major roads: A population-based matched case-control study among the pediatric Medicaid population in Detroit, Michigan. *Environmental Health*. **10**(34)
- LIGHT, RJ., & PILLEMER, DB. (1984). *Summing up: The science of reviewing research*. Cambridge, UK: Harvard University Press
- LIN, S., MUNSIE, JP., HWANG, SA., FITZGERALD, E., & CAYO, MR. (2002). Childhood asthma hospitalization and residential exposure to state route traffic. *Environmental Research*. **88**(2), pp.73-81
- LIN, M., CHEN, Y., VILLENEUVE, PJ., BURNETT, RT., LEMYRE, L., HERTZMAN, C., MCGRAIL, KM., & KREWSKI, D. (2004). Gaseous Air Pollutants and Asthma Hospitalization of Children with Low Household Income in Vancouver, British Columbia, Canada. *American Journal of Epidemiology*. **159**(3), pp.294-303
- LIN, M., STIEB, DM., & CHEN, Y. (2005). Coarse Particulate Matter and Hospitalization for Respiratory Infections in Children Younger Than 15 Years in Toronto: A Case-Crossover Analysis. *Paediatrics*. **116**(2), pp.235-240

- LIPFERT, FW., WYZGA, RE., BATY, JD., & MILLER, JP. (2008). Vehicular traffic effects on survival within the Washington University-EPRI veterans cohort: New estimates and sensitivity studies. *Inhalation Toxicology*. **20**(10), pp.949-960
- LONGLEY, PA., & TOBON, C. (2004). Spatial Dependence and Heterogeneity in Patterns of Hardship: An Intra-Urban Analysis. *Annals of the Association of American Geographers*. **94**(3), pp.503-519
- LUKEN, RA. (2009). Equivocating on the Polluter-Pays Principle: The consequences for Pakistan. *Journal of Environmental Management*. **90**, pp.3479-3484
- LUPPI, B., PARISI, F., & RAJAGOPALAN, S. (2012). The rise and fall of the Polluter-Pays Principle in developing countries. *International Review of Law and Economics*. **32**, pp.135-144
- MAANTAY, J. (2007). Asthma and air pollution in the Bronx: Methodological and data considerations in using GIS for environmental justice and health research. *Health & Place*. **13**(1), pp.32-56
- MACINTYRE, EA., KARR, CJ., KOEHOORN, M., DEMERS, PA., TAMBURIC, L., LENCAR, C., BRAUER, M. (2011). Residential air pollution and otitis media during the first two years of life. *Epidemiology*. **22**(1), pp.81-89
- MACNEE, W., & DONALDSON, K. (2003). Mechanism of lung injury caused by PM₁₀ and ultrafine particles with special reference to COPD. *European respiratory Journal*. **21**(40), pp.47s-51s
- MAHESWARAN, R., & ELLIOTT, P. (2003). Stroke Mortality Associated With Living Near Main Roads in England and Wales. *Stroke*. **34**(12), pp.2776-2780
- MAKI, J., HIRANO, M., HOKA, S., KANAIDE, H., & HIRANO, K. (2010). Involvement of Reactive Oxygen Species in Thrombin-induced Pulmonary Vasoconstriction. *American Journal of Respiratory and Critical Care Medicine*. **182**(11), pp.1435-1444
- MANN, I. (2009). *A comparative study of the Polluter Pays Principle and its international normative effect on pollutive processes*. British Virginia Islands, Forbes Hare
- MATHIEU-NOLF, M. (2002). Poisons in the air: A cause of chronic disease in children. *Clinical Toxicology*. **40**(4), pp.483-491
- MAZUR, E. (2010). Acute pharyngotonsillitis: Current diagnosis and treatment. *New Medicine*. **4**, pp.117-129
- MCCONNELL, R., BERHANE, K., YAO, L., JERRETT, M., LURMANN, F., GILLILAND, F., KUNZLI, N., GAUDERMAN, J., AVOL, E., THOMAS, D., & PETERS, J. (2006). Traffic, susceptibility, and childhood asthma. *Environmental Health Perspectives*. **114**(5), pp.766-772
- MCLAREN, D., COTTRAY, O., TAYLOR, M., PIPES, S., & BULLOCK, S. (1999). Pollution injustice: The geographic relation between household income and polluting factories. London: Friends of the Earth Trust. Available via: http://foe.co.uk/resource/reports/income_pollution.html [Accessed: April 2011]

- MCLEOD, H., LANGFORD, I.H., JONES, A.P., STEDMAN, JR., DAY, R.J., LORENZONI, I., & BATEMAN, I.J. (2000). The relationship between socio-economic indicators and air pollution in England and Wales: Implications for environmental justice. *Regional Environmental Change*. **1**(2), pp.78-85
- MENNIS, J., & JORDAN, L. (2005). The Distribution of Environmental Equity: Exploring Spatial Nonstationarity in Multivariate Models of Air Toxic Releases. *Annals of the Association of American Geographers*. **95**(2), pp.249-268
- MENNIS, J. (2006). Mapping the results of geographically weighted regression. *The Cartographic Journal*. **43**(2), pp.171-179
- MERCER, S.W., & WATT, G.C. (2007). The inverse care law: Clinical primary care encounters in deprived and affluent areas of Scotland. *Annals of Family Medicine*. **5**(6), pp.503-510
- MERCER, S.W., CAWSTON, P.G., & BIKKER, A.P. (2007). Quality in general practice consultations; a qualitative study of the views of patients living in an area of high socio-economic deprivation in Scotland. *BMC Family Practice*. Vol.**8**(22)
- MICROSOFT CORPORATION. (2009). *Microsoft Office 2010*. Redmond, USA
- MIDDLETON, N., YIALLOUROS, P., NICOLAOU, N., KLEANTHOS, S., PIPIS, S., ZENIOU, M., DEMOKRITOU, P., & KOUTRAKIS, P. (2010). Residential exposure to motor vehicle emissions and the risk of wheezing among 7-8 year-old schoolchildren: A city-wide cross-sectional study in Nicosia, Cyprus. *Environmental Health*. **9**(28)
- MINITAB LTD. (2010). *Minitab Version 16.0.1*. Coventry, UK
- MITCHELL, A. (2004). *The ESRI guide to GIS analysis - Volume 2: Spatial measurements and statistics*. Redlands, USA: ESRI Press
- MITCHELL G. (2005). Forecasting environmental equity: Air quality responses to road user charging in Leeds, UK. *Journal of Environmental Management*. **77**(3), pp.212-226
- MITCHELL, G., & DORLING, D. (2003). An environmental justice analysis of British air quality. *Environment and Planning A*. **35**(5), pp.909-929
- MIYAKE, Y., TANAKA, K., FUJIWARA, H., MITANI, Y., IKEMI, H., SASAKI, S., OHYA, Y., & HIROTA, Y. (2010). Residential proximity to main roads during pregnancy and the risk of allergic disorders in Japanese infants: The Osaka Maternal and Child Health Study. *Pediatric Allergy and Immunology*. **21**(1), pp.22-28
- MORAN, P.A.P. (1948). The Interpretation of Statistical Maps. *Journal of the Royal Statistical Society. Series B (Methodological)*. **10**(2), pp.243-251
- MORELLO-FROSCH, R., PASTOR, M., & SADD, J. (2001). Environmental justice and southern California's "Riskscape": The distribution of air toxics exposures and health risks among diverse communities. *Urban Affairs Review*. **36**(4), pp.551-578
- MOUDGIL, H., & HONEYBOURNE, D. (1998). Differences in asthma management between white European and Indian subcontinent ethnic groups living in socioeconomically deprived areas in the Birmingham (UK) conurbation. *Thorax*. **53**(6), pp.490-494

- MOURA, M., JUNGER, WL., MENDONCA, GA., & LEON, AP. (2009). Air quality and emergency pediatric care for symptoms of bronchial obstruction categorized by age bracket in Rio de Janeiro, Brazil. *Cadernos de Saúde Pública*. **25**(3), pp.635-644
- MUSHTAQ, N., EZZATI, M., HALL, L., DICKSON, I., KIRWAN, M., PNG, KM., MUDWAY, IS., & GRIGG, J. (2011). Adhesion of *Streptococcus pneumoniae* to human airway epithelial cells exposed to urban particulate matter. *Journal of Allergy and Clinical Immunology*. **127**(5), pp.1236-1242
- NADEL, S. (2009). *Infectious diseases in the pediatric intensive care unit*. London, UK: Springer-Verlag Ltd
- NAESS, O., PIRO, FN., NAFSTAD, P., SMITH, GD., & LEYLAND, AH. (2007). Air pollution, social deprivation, and mortality: A multilevel cohort study. *Epidemiology*. **18**(6), pp.686-694
- NAMDEO, A., & BELL, MC. (2005). Characteristics and health implications of fine and coarse particulates at roadside, urban background and rural sites in UK. *Environment International*. **31**(4), pp.565-573
- NAMDEO, A., & STRINGER, C. (2008). Investigating the relationship between air pollution, health and social deprivation in Leeds, UK. *Environment International*. **34**(5), pp.585-591
- NEAS, LM., DOCKERY, DW., KOUTRAKIS, P., SPEIZER, FE. (1999). Fine particles and peak flow in children: Acidity versus mass. *Epidemiology*. **10**(5), pp.550-553
- NEMMAR, A., HOYLAERTS, MF., HOET, PH. & NEMERY, B. (2004). Possible mechanisms of the cardiovascular effects of inhaled particles: systemic translocation and prothrombotic effects. *Toxicology Letters*. **149**(1-3), pp.243-253
- NEWCOMB, P., & LI, J. (2008). Predicting admissions for childhood asthma based on proximity to major roadways. *Journal of Nursing Scholarship*. **40**(4), pp.319-325
- NICHOLS, LA. (1959). The Catarrhal Child. *Journal of the College of General Practitioners and Research Newsletter*. **2**(1), pp.43-52
- NIGHTINGALE, JA., MAGGS, R., CULLINAN, P., DONNELLY, LE., ROGERS, DF., KINNERSLEY, R., CHUNG, KF., BARNES, PJ., ASHMORE, M., & NEWMAN-TAYLOR, A. (2000). Airway inflammation after controlled exposure to diesel exhaust particulates. *American Journal of Respiratory and Critical Care Medicine*. **162**(1), pp.161-166
- NORMAN, P., BOYLE, P., & REES, P. (2005). Selective migration, health and deprivation: A longitudinal analysis. *Social Science & Medicine*. **60**(12), pp.2755-2771
- NORMAN, P., BOYLE, P., EXETER, D., FENG, Z., & POPHAM, F. (2011). Rising premature mortality in the UK's persistently deprived areas: Only a Scottish phenomenon? *Social Science & Medicine*. **73**(11), pp.1575-1584
- NTZIACHRISTOS, L., & SAMARAS, Z. (2000). *COPERT III Computer program to calculate emissions from road transport, Methodology and emission factors (Version 2.1)*. Copenhagen, DEN: European Environment Agency, Technical Report No. 49

- NTZIACHRISTOS, L., GIECHASKIEL, B., PISTIKOPOULOS, P., FYSIKAS, E., SAMARAS, Z. (2003). Particle emissions characteristics of different on-road vehicles. *SAE Transactions 2003-01-1888. Journal of Fuels and Lubricants*. **112**, pp.1568-1579
- OBERDORSTER, G. (1995). Lung particle overload: Implications for occupational exposures to particles. *Regulatory Toxicology and Pharmacology*. **21**(1), pp.123-135
- OBERDORSTER, G., STONE, V., & DONALDSON, K. (2007). Toxicology of nanoparticles: A historical perspective. *Nanotoxicology*. **1**(1), pp.2-25
- BERG, M., JAAKKOLA, MS., WOODWARD, A., PERUGA, A., & PRÜSS-USTUN, A. (2011). Worldwide burden of disease from exposure to second-hand smoke: a retrospective analysis of data from 192 countries. *The Lancet*, **377**(9760), pp.139-146
- O'DONNELL, DR., PARSLLOW, RC., & DRAPER, ES. (2010). Deprivation, ethnicity and prematurity in infant respiratory failure in PICU in the UK. *Acta Paediatrica*. **99**(8), pp.1186-1191
- OFFICE FOR NATIONAL STATISTICS. (ONS 2001a). *2001 Census: Digitised boundary data (English Unitary Authority Census Area Statistic Wards) Leicester Unitary Authority*. UKBORDERS, Census Geography Data Unit, MIMAS (University of Manchester)
- OFFICE FOR NATIONAL STATISTICS. (ONS 2001b). *2001 Census: Digitised boundary data (English Super Output Areas, Lower Layer) Leicester Unitary Authority*. UKBORDERS, Census Geography Data Unit, MIMAS (University of Manchester)
- OFFICE FOR NATIONAL STATISTICS. (ONS 2003). *UK Census 2001: Standard Area Statistics (England and Wales)*. CasWeb, Census Dissemination Unit, MIMAS (University of Manchester)
- OFFICE FOR NATIONAL STATISTICS. (ONS 2008a). *Index of Multiple Deprivation 2007: Measure of multiple deprivation at small area level made up of seven domains*. Newport, UK: Neighbourhood Statistical Releases, Department for Communities and Local Government
- OFFICE FOR NATIONAL STATISTICS. (ONS 2008b). *Healthy Lifestyle Behaviours: Model Based Estimates for Primary Care Organisations in England, 2003-2005*. Leeds, UK: National Centre for Social Research and The NHS Information Centre for health and social care
- OFFICE FOR NATIONAL STATISTICS. (ONS 2010). *Mid-Year Population Estimates for Lower Layer Super Output Areas in England and Wales by Broad Age Group and Sex: 2002-2008 (revised)*. Available via: <http://ons.gov.uk/ons/datasets-and-tables/index.html> [Accessed: December 2010]
- OFFICE FOR NATIONAL STATISTICS. (ONS 2011). *Methodology note on production of population estimates using a postcode best fit methodology*. Newport, UK: Small Area Population Estimates
- O'NEILL, MS., JERRETT, M., KAWACHI, I., LEVY, JI., COHEN, AJ., GOUVEIA, N., WILKINSON, P., FLETCHER, T., CIFUENTES, L., & SCHWARTZ, J. (2003). Health, Wealth, and Air Pollution: Advancing Theory and Methods. *Environmental Health Perspectives*. **111**(16), pp.1861-1870

- OOSTERLEE, A., DRIJVER, M., LEBRET, E., & BRUNEKREEF, B. (1996). Chronic respiratory symptoms in children and adults living along streets with high traffic density. *Occupational and Environmental Medicine*. **53**(4), pp.241-247
- OPENSHAW, S. (1984). *The modifiable areal unit problem*. London, UK: Institute of British Geographers, Concepts and techniques in modern geography, No.38
- ORD, JK., & GETIS, A. (1995). Local spatial autocorrelation statistics: distributional issues and an application. *Geographical Analysis*. **27**(4), pp.286-306
- ORGANISATION FOR ECONOMIC CO-OPERATION AND DEVELOPMENT. (OECD 1988). *Transport and the Environment*. Paris, FRA: OECD Publishing
- ORGANISATION FOR ECONOMIC CO-OPERATION AND DEVELOPMENT. (OECD 1992). *The Polluter-Pays Principle: OECD Analysis and Recommendations*. Paris, FRA: OECD Publishing. OCDE/GD(92)81
- ORGANISATION FOR ECONOMIC CO-OPERATION AND DEVELOPMENT. (OECD 2003). *OECD Economic Surveys 2002-2003: Portugal*. Paris, FRA: OECD Publishing
- OXFORD CITY COUNCIL. (2006). *Local Air Quality Management: Central Oxford Air Quality Action Plan*. Available via: www.oxford.gov.uk/Direct/64005AQAPFinalApril06.pdf [Accessed: November 2009]
- PEARCE, JR., RICHARDSON, EA., MITCHELL, RJ., & SHORTT, NK. (2010). Environmental justice and health: The implications of the socio-spatial distribution of multiple environmental deprivation for health inequalities in the United Kingdom. *Transactions of the Institute of British Geographers*. **35**(4), pp.522-539
- PEARSON K. (1985). Contributions to the mathematical theory of evolution II: Skew variation in homogeneous material. *Philosophical Transactions of the Royal Society of London A*. **186**(1895), pp.343-414
- PETERS, A., GOLDSTEIN, IF., BEYER, U., FRANKE, K., HEINRICH, J., DOCKERY, DW., SPENGLER, JD., & WICHMANN, HE. (1996). Acute Health Effects of Exposure to High Levels of Air Pollution in Eastern Europe. *American Journal of Epidemiology*. **144**(6), pp.570-581
- PETERS, A., DORING, A., WICHMANN, HE., & WOLFGANG, K. (1997). Increased plasma viscosity during an air pollution episode: A link to mortality? *The Lancet*. **349**(9065), pp.1582-1587
- PLATT, SD., MARTIN, CJ., HUNT, SM., & LEWIS, CW. (1989). Damp housing, mould growth, and symptomatic health state. *BMJ*. **298**(6689), pp.1673-1678
- POPE, CA., DOCKERY, DW., SPENGLER, JD., & RAIZENNE, ME. (1991). Respiratory health and PM₁₀ pollution: A daily time series analysis. *American Journal of Respiratory and Critical Care Medicine*. **144**(3), pp.668-674
- POPE, CA., & DOCKERY, DW. (1992). Acute health effects of PM₁₀ pollution on symptomatic and asymptomatic children. *American Review of Respiratory Disease*. **145**(5), pp.1123-1128

- POPE, CA., THUN, MJ., NAMBOODIRI, MM., DOCKERY, DW., EVANS, JS., SPEIZER, FE., HEATH, CW. (1995). Particulate air pollution as a predictor of mortality in a prospective study of U.S. adults. *American Journal of Respiratory and Critical Care Medicine*. **151**, pp.669-674
- POPE, AC. (2000). Epidemiology of Fine Particulate Air Pollution and Human Health: Biologic Mechanisms and who's at Risk? *Environmental Health Perspectives*. **108**(4), pp.713-723
- PSARRAS, S., PAPADOPOULOS, NG., & JOHNSTON, SL. (2004). Pathogenesis of respiratory syncytial virus bronchiolitis-related wheezing. *Paediatric Respiratory Reviews*. **5**(Sup. A), S179-S184
- PUJADES-RODRIGUEZ, M., LEWIS, S., MCKEEVER, T., BRITTON, J., & VENN, A. (2009). Effect of living close to a main road on asthma, allergy, lung function and chronic obstructive pulmonary disease. *Occupational and Environmental Medicine*. **66**(10), pp.679-684
- QUALITY OF URBAN AIR REVIEW GROUP. (QUARG 1996). *Airborne Particulate Matter in the United Kingdom*. London, UK: Her Majesty's Stationary Office
- RAPAPORT, E. (2002). The Stockholm environmental zone, a method to curb air pollution from bus and truck traffic. *Transportation Research Part D: Transport and Environment*. **7**(3), pp.213-224
- REEVES, SJ., SAUER, J., STEWART, R., GRANGER, A., & HOWARD, RJ. (2001). Increased first-contact rates for very-late-onset schizophrenia-like psychosis in African- and Caribbean-born elders. *The British Journal of Psychiatry*, **179**, pp.172-174
- RENWICK, LC., DONALDSON, K., & CLOUTER, A. (2001). Impairment of alveolar macrophage phagocytosis by ultrafine particles. *Toxicology and Applied Pharmacology*. **172**, pp.119-127
- RISOM, L., MOLLER, P., & LOFT, S. (2005). Oxidative stress-induced DNA damage by particulate air pollution. *Mutation Research*. **592**, pp.119-137
- RODGERS, JL., & NICEWANDER, WA. (1988). Thirteen ways to look at the correlation coefficient. *The American Statistician*. **42**(1), pp.59-66
- ROEMER, W., HOEK, G., & BRUNEKREEF, B. (1993). Effect of ambient winter air pollution on respiratory health of children with chronic respiratory symptoms. *American Review of Respiratory Disease*. **147**(1), pp.118-124
- ROEMER, W., HOEK, G., BRUNEKREEF, B., HALUSZKA, J., KALANDIDI, A., PEKKANEN, J. (1998). Daily variations in air pollution and respiratory health in a multicentre study: The PEACE project. *European Respiratory Journal*. **12**(6), pp.1354-1361
- ROEMER, W., HOEK, G., BRUNEKREEF, B. (2000). Pollution effects on asthmatic children in Europe, the PEACE study. *Clinical & Experimental Allergy*. **30**(8), pp.1067-1075

- ROMERO-PLACERES, M., MAS-BERMEJO, P., LACASANA-NAVARRO, M., ROJO-SOLIS, MMT., AGUILAR-VALDES, J., & ROMIEU, I. (2004). Air pollution, bronchial asthma, and acute respiratory infections in children less years of age [Havana City]. *Salud Publica De Mexico*. **46**(3), pp.222-233
- ROMIEU, I., MENESES, F., RUIZ, S., SIENRA, JJ., HUERTA, J., WHITE, MC., & ETZEL, RA. (1996). Effects of air pollution on the respiratory health of asthmatic children living in Mexico City. *American Journal of Respiratory and Critical Care Medicine*. **154**(2), pp.300-307
- ROYAL COMMISSION. (1971). Royal Commission on environmental pollution: First report. London, UK: Her Majesty's Stationary Office. Available via: <http://www.rcep.org.uk/reports/01-first%20report/1971-01firstreport.pdf> [Accessed: June 2011]
- RYAN, PH., LEMASTERS, G., BIAGINI, J., BERNSTEIN, D., GRINSHPUN, SA., SHUKLA, R., WILSON, K., VILLAREAL, M., BURKLE, J., & LOCKEY, J. (2005). Is it traffic type, volume, or distance? Wheezing in infants living near truck and bus traffic. *Journal of Allergy and Clinical Immunology*. **116**(2), pp.279-284
- SAELENS, BE., SALLIS, JF., FRANK, LD., COUCH, SC., ZHOU, C., COLBURN, T., CAIN, KL., CHAPMAN, J., GLANZ K. (2012). Obesogenic neighbourhood environments, child and parent obesity: 'The Neighbourhood Impact on Kids study'. *American Journal of Preventive Medicine*, **42**(5)
- SAXENA, S., ELIAHOO, J., & MAJEED, A. (2002). Socioeconomic and ethnic group differences in self-reported health status and use of health services by children and young people in England: cross sectional study. *BMJ*. **325**(7363), pp.520-525
- SCHEERS, H., MWALILI, SM., FAES, C., FIERENS, F., NEMERY, B., & NAWROT, TS. (2011). Does air pollution trigger infant mortality in Western Europe? A case-crossover study. *Environmental Health Perspectives*. **119**(7), pp.1017-1022
- SCHOETTLIN, CE., & LANDAU, E. (1961). Air Pollution and Asthmatic Attacks in the Los Angeles Area. *Public Health Reports*. **76**(6), pp.545-551
- SCHOLES, S., PICKERING, K., & RAYAT, P. (2007). Healthy Lifestyle Behaviours: Model Based Estimates for Middle Layer Super Output Areas and Local Authorities in England, 2003-2005: User Guide. Available via: <https://catalogue.ic.nhs.uk/publications/public-health/population/neig-la-mode-esti-heal-beha-03-05/neig-la-mode-esti-beha-03-05-gui.pdf> [Accessed: July 2013]
- SCHOLES, S., PICKERING, K., & DEVERILL, C. (2008a) Healthy Lifestyle Behaviours: Model Based Estimates for Middle Layer Super Output Areas and Local Authorities in England, 2003-2005: Stage 2 Report. Available via: <https://catalogue.ic.nhs.uk/publications/public-health/population/neig-la-mode-esti-heal-beha-03-05/neig-la-mode-esti-beha-03-05-rep-2.pdf> [Accessed: July 2013]
- SCHOLES, S., PICKERING, K., & DEVERILL, C. (2008b). Healthy Lifestyle Behaviours: Model Based Estimates for Local Authorities in England, 2003-2005: Stage 3 Report. Available via: <https://catalogue.ic.nhs.uk/publications/public-health/population/neig-la-mode-esti-heal-beha-03-05/neig-la-mode-estil-beha-03-05-rep-3.pdf> [Accessed: July 2013]

- SCHUENEMEYER, J., & DREW, L. (2011). *Statistics for Earth and Environmental Scientists*. New Jersey, USA: John Wiley & Sons Inc.
- SCHWARTZ, S. (1994) The fallacy of the ecological fallacy: The potential misuse of a concept and the consequences. *American Journal of Public Health*. **84**(5), pp.819-824
- SCHWARTZ, J. (2004). Air pollution and children's health. *Paediatrics*. **113**(4), pp.1037-1043
- SCOTTISH EXECUTIVE. (2004). *Investigating environmental justice in Scotland: Links between measures of environmental quality and social deprivation*. Scotland & Northern Ireland Forum for Environmental Research, Edinburgh, UK
- SEATON, A., SEATON, D., & LEITCH, AG. (1989). *Crofton and Douglas's respiratory diseases*. Oxford, UK: Blackwell Scientific
- SHAH, SM., & COOK, DG. (2008). Socio-economic determinants of casualty and NHS Direct use. *Journal of Public Health*. **30**(1), pp.75-81
- SHIMA, M., NITTA, Y., & ADACHI, M. (2003). Traffic-related air pollution and respiratory symptoms in children living along trunk roads in Chiba Prefecture, Japan. *Journal of Epidemiology*. **13**(2), pp.108-119
- SIMES, RJ. (1986). An improved Bonferroni procedure for multiple tests of significance. *Biometrika*. **73**(3), pp.751-754
- SIMPSON, L. (2002). Geography conversion tables: A framework for conversion of data between geographical units. *International Journal of Population Geography*. **8**(1), pp.69-82
- SIMPSON, L., & YU, A. (2003). Public access to conversion of data between geographies, with multiple look up tables derived from a postal directory. *Computers, Environment and Urban Systems*. **27**(3), p283-307
- SMITH, RL., DAVIS, JM., SACKS, J., SPECKMAN, P., & STYER, P. (2000). Regression models for air pollution and daily mortality: Analysis of data from Birmingham, Alabama. *Environmetrics*. **11**, pp.719-743
- SPANNHAKE, EW., REDDY, SP., JACOBY, DB., YU, XY., SAATIAN, B., & TIAN, J. (2002). Synergism between rhinovirus infection and oxidant pollutant exposure enhances airway epithelial cell cytokine production. *Environmental Health Perspectives*. **110**(7), pp.665-670
- SPEARMAN, C. (1904). The proof and measurement of association between two things. *The American journal of psychology*. **15**(1), pp.72-101
- SPENCER, N., LOGAN, S., SCHOLEY, S., & GENTLE, S. (1996). Deprivation and bronchiolitis. *Archives of Disease in Childhood*. **74**, pp.50-52
- STAFFORD, M., CUMMINS, S., ELLAWAY, A., SACKER, A., WIGGINS, RD. & MACINTYRE, S. (2007). Pathways to obesity: Identifying local, modifiable determinants of physical activity and diet. *Social Science & Medicine*. **65**(9), pp.1882-1897

- STEELE, RM., VAN-SLUIJS, E., SHARP, SJ., LANDSBAUGH, JR., EKELUND, U., & GRIFFIN, SJ. (2010). An investigation of patterns of children's sedentary and vigorous physical activity throughout the week. *International Journal of Behavioural Nutrition and Physical Activity*. **7**(88)
- STEERENBERG, PA., ZONNENBERG, JA., DORMANS, JA., JOON, PN., WOUTERS, IM., VAN-BREE, L., SCHEEPERS, PT., & VAN-LOVEREN, H. (1998). Diesel exhaust particles induced release of interleukin 6 and 8 by (primed) human bronchial epithelial cells (BEAS 2B) in vitro. *Experimental Lung Research*. **24**(1), pp.85-100
- STICK, S. (2000). The contribution of airway development to paediatric and adult lung disease. *Thorax*. **55**(7), pp.587-594
- STRAUSS, RS., & KNIGHT, J. (1999). Influence of the home environment on the development of obesity in children. *Pediatrics*, **103**(6)
- THE NATIONAL ARCHIVES. (TNA 2004). UK Public General Acts, Chapter 18: Traffic Management Act 2004. Available via: http://legislation.gov.uk/ukpga/2004/18/pdfs/ukpga_20040018_en.pdf [Accessed: August 2010]
- TOBLER, W.(1970). A computer movie simulating urban growth in the Detroit region. *Economic Geography*. **46**(2), pp.234-240
- TODAR, K. (2013). Todar's Online Textbook of Bacteriology. University of Wisconsin-Madison. Available via: <http://textbookofbacteriology.net/index.html> [Accessed: January 2013]
- TONNE, C., BEEVERS, S., ARMSTRONG, B., KELLY, F., & WILINSON, P. (2008). Air pollution and mortality benefits of the London Congestion Charge: spatial and socioeconomic inequalities. *Occupational and Environmental Medicine*. **65**(9), pp.620-627
- TRANSPORT RESEARCH LABORATORY. (TRL 2009). Road vehicle emission factors 2009. Available via: <https://gov.uk/government/publications/road-vehicle-emission-factors-2009> [Accessed: January 2010]
- TSAGATAKIS, I., BUSH, T., PASSANT, N., & BROPHY, N. (2010). *UK Emission Mapping Methodology 2008: A report of the National Atmospheric Emissions Inventory, AEAT/ENV/R/3105*. Harwell, UK: AEA Technology,
- TSAI, P.-J., LIN, M.-L., CHU, C.-M., & PERNG, C.-H. (2009). Spatial autocorrelation analysis of health care hotspots in Taiwan in 2006. *BMC Public Health*. **9**(464)
- TUKEY, JW. (1977). *Exploratory Data Analysis*. Reading, USA: Addison-Wesley Publishing
- UK METEOROLOGICAL OFFICE. (2006). *MIDAS Land Surface Stations data: 1853-2011*. NCAS British Atmospheric Data Centre. Available via: http://badc.nerc.ac.uk/view/badc.nerc.ac.uk_ATOM_dataent_ukmo-midas [Accessed: February 2011]
- URDAN, TC. (2005). *Statistics in Plain English*, 3rd edition. Mahwah, USA: Lawrence Erlbaum Associates, Inc.
- U.S. ENVIRONMENTAL PROTECTION AGENCY. (US EPA 1995). '*Environmental Justice Strategy*': Executive order, number 12898. Washington, USA: Government Printing Office

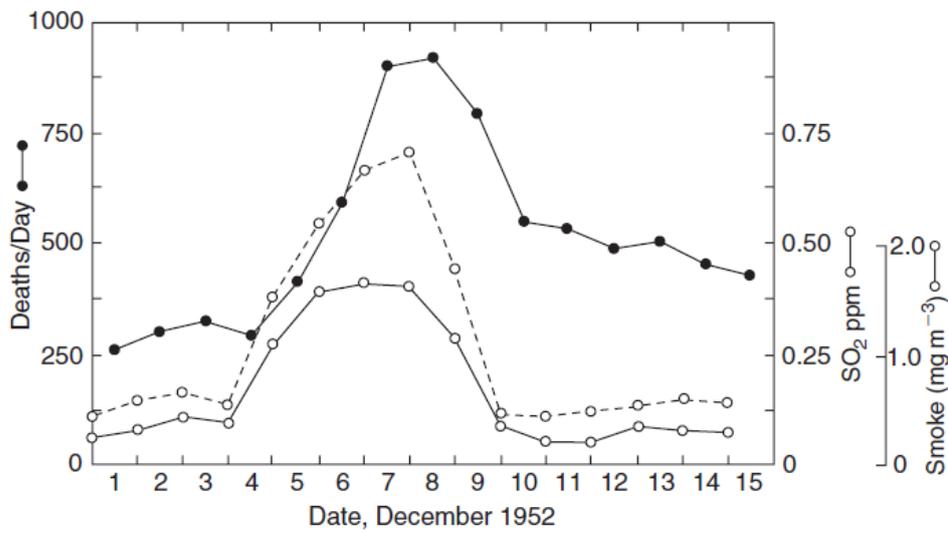
- U.S. ENVIRONMENTAL PROTECTION AGENCY. (US EPA 2013). *EPA and Environmental Justice*. Available via: <http://www.epa.gov/compliance/environmentaljustice/basics/index.html> [Accessed: July 2013]
- UTELL, MJ., & FRAMPTON, MW. (2000). Acute health effects of ambient air pollution: The ultrafine particle hypothesis. *Journal of Aerosol Medicine-Deposition Clearance and Effects in the Lung*. **13**(4), pp.355-359
- VAN-GROENIGEN, JW. (2000). The influence of variogram parameters on optimal sampling schemes for mapping by kriging. *Geoderma*. **97**(3-4), pp.223-236
- VEDAL, S., PETKAU, J., WHITE, R., & BLAIR, J. (1998). Acute effects of ambient inhalable particles in asthmatic and non-asthmatic children. *American Journal of Respiratory and Critical Care Medicine*. **157**(4), pp.1034-1043
- VENN, AJ., LEWIS, SA., COOPER, M., HUBBARD, R., & BRITTON, J. (2001). Living near a main road and the risk of wheezing illness in children. *American Journal of Respiratory and Critical Care Medicine*. **164**(12), pp.2177-2180
- VICHA, O. (2011). The Polluter-Pays Principle in OECD recommendations and its application in international and EC/EU law. *Czech Yearbook of Public & Private International Law*. **2**, pp.57-67
- VIDAL-HALL J. (2003). Leicester: City of migration. *Index on Censorship*. **32**(2), p132-141
- VIEIRA, V., WEBSTER, T., WEINBERG, J., ASCHENGRAU, A., & OZONOFF, D. (2005). Spatial analysis of lung, colorectal, and breast cancer on Cape Cod: An application of generalized additive models to case-control data. *Environmental Health*, **4**(11)
- WALL MM. (2004). A close look at the spatial structure implied by the CAR and SAR models. *Journal of Statistical Planning and Inference*. **121**(2), pp.311-324
- WARD, D., ROBERTS, K., JONES, N., HARRISON, R., AYRES, J., HUSSAIN, S., & WALTERS, S. (2002). Effects of daily variation in outdoor particulates and ambient acid species in normal and asthmatic children. *Thorax*. **57**(6), pp.489-502
- WARD, DJ., & AYRES, JG. (2004). Particulate air pollution and panel studies in children: A systematic review. *Occupational and Environmental Medicine*. **61**(4)
- WARD, JPT., WARD, J., & LEACH, RM. (2010). *The Respiratory System at a Glance, 3rd edition*. Chichester, UK: Wiley-Blackwell
- WARDLE, J., CARNELL, S., HAWORTH, CM., & PLOMIN, R. (2008). Evidence for a strong genetic influence on childhood adiposity despite the force of the obesogenic environment. *The American Journal of Clinical Nutrition*, **87**(2), pp.398-404
- WARDLE, J., LLEWELLYN, C., SANDERSON, S., & PLOMIN, R. (2009). The FTO gene and measured food intake in children. *International Journal of Obesity*, **33**(1), pp.42-45
- WEBSTER, R., & OLIVER, MA. (2007). *Geostatistics for environmental scientists, 2nd edition*. Chichester, UK: John Wiley & Sons Ltd

- WEINBERGER, SE., COCKRILL, BA., & MANDEL, J. (2008). Principles of pulmonary medicine, 5th edition. Philadelphia, USA: Saunders/Elsevier
- WEINMAYR, G., ROMEO, E., DE-SARIO, M., WEILAND, SK., & FORASTIERE, F. (2010). Short-term effects of PM₁₀ and NO₂ on respiratory health among children with asthma or asthma-like symptoms: A systematic review and meta-analysis. *Environmental Health Perspectives*. **118**(4), pp.449-457
- WHEELER, BW., & BEN-SHLOMO, Y. (2005). Environmental equity, air quality, socioeconomic status, and respiratory health: A linkage analysis of routine data from the Health Survey for England. *Journal of Epidemiology and Community Health*. **59**(11), pp.948-954
- WHEELER, D., & TIEFELSDORF, M. (2005). Multicollinearity and correlation among local regression coefficients in geographically weighted regression. *Journal of Geographical Systems*. **7**(2), pp.161-187
- WILKINSON, P., ELLIOTT, P., GRUNDY, C., SHADDICK, G., THAKRAR, B., WALLS, P., & FALCONER, S. (1999). Case-control study of hospital admission with asthma in children aged 5-14 years: relation with road traffic in North West London. *Thorax*. **54**(12), pp.1070-1074
- WILKINSON, RG., & PICKETT, KE. (2006). Income inequality and population health: A review and explanation of the evidence. *Social Science & Medicine*. **62**(7), pp.1768-1784
- WJST, M., REITMEIR, P., DOLD, S., WULFF, A., NICOLAI, T., VON-LOEFFELHOLZ-COLBERG, EF., & VON-MUTIUS, E. (1993). Road traffic and adverse effects on respiratory health in children. *BMJ*. **307**(6904), pp.596-600
- WOERDMAN, E., ARCURI, A., & CLO, S. (2007). Emissions trading and the Polluter-Pays Principle: Do polluters pay under Grandfathering? *Rotterdam Institute of Law and Economics (RILE) Working Paper Series*. **14**
- WOMBLE, WH. (1951). Differential systematics. *Science*, **114**(2961), pp.315-322
- WONG, C., THACH, TQ., CHAU, PYK., CHAN, EKP., CHUNG, RY., OU, C., YANG, L., PEIRIS, JSM., THOMAS, GN., LARN, T., WONG, T., & HEDLEY, AJ. (2010). Interaction between air pollution and respiratory viruses: Time-series study of daily mortality and hospital admissions in Hong Kong. *Health Effects Institute (HEI) Research Report*, **154**: pp.283-362
- WOODS, L., RACHET, B., RIGA, M., STONE, N., SHAH, A., & COLEMAN, MP. (2005). Geographical variation in life expectancy at birth in England and Wales is largely explained by deprivation. *Journal of Epidemiology and Community Health*. **59**(2), pp.115-120
- WORLD HEALTH ORGANIZATION. (WHO 2000). Air Quality Guidelines for Europe, 2nd edition. *WHO Regional Publications, European Series*. **91**, pp.1-11
- WORLD HEALTH ORGANIZATION. (WHO 2002). *The world health report 2002: Reducing risks and promoting healthy life*. Geneva, SUI: WHO Publications
- WORLD HEALTH ORGANIZATION. (WHO 2005) *Health effects of transport-related air pollution*. Copenhagen, DEN: WHO Regional Office for Europe

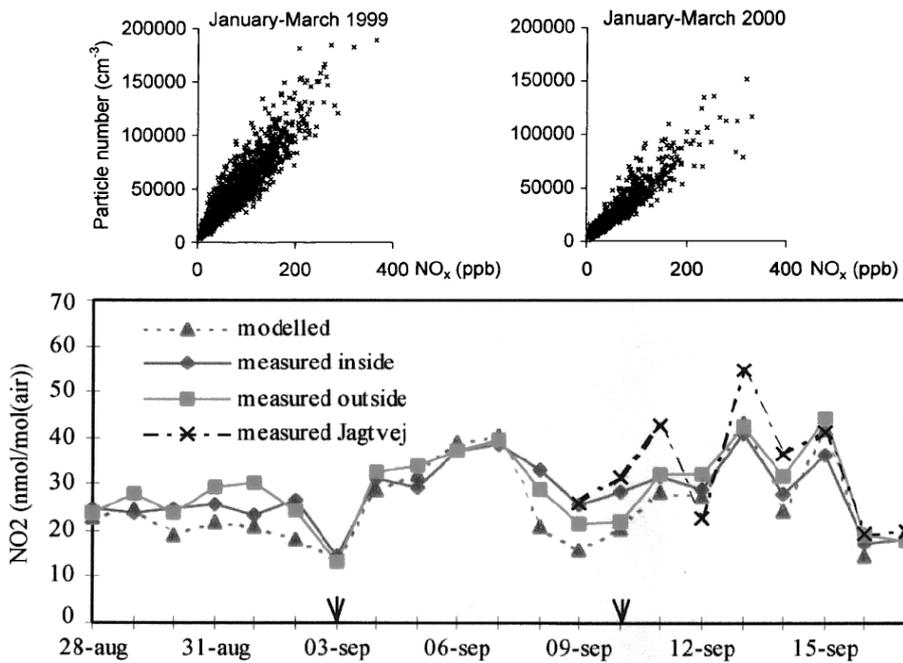
- REFERENCES -

- WORLD HEALTH ORGANIZATION. (WHO 2006). *Air quality guidelines for particulate matter, ozone, nitrogen dioxide and sulphur dioxide: Global update 2005*. Geneva, SUI: WHO Publications
- WORLD HEALTH ORGANIZATION. (WHO 2008). *The global burden of disease: 2004 Update*. Geneva, SUI: WHO Publications
- WORLD HEALTH ORGANIZATION. (WHO 2010). *ICD-10: International statistical classification of diseases and related health problems, 10th Edition [Revised]*. Geneva, SUI: Department of Measurement and Health Information Systems.
- YOUNG, GS., FOX, MA., TRUSH, M., KANAREK, N., GLASS, TA., & CURRIERO, FC. (2012). Differential exposure to hazardous air pollution in the united states: A multilevel analysis of urbanization and neighbourhood socioeconomic deprivation. *International Journal of Environmental Research and Public Health*. **9**(6), pp.2204-2225
- ZELIKOFF, JT., CHEN, LC., COHEN, MD., FANG, K., GORDON, T., LI, Y., NADZIEJKO, C., & SCHLESINGER, RB. (2003). Effects of inhaled ambient particulate matter on pulmonary antimicrobial immune defence. *Inhalation Toxicology*. **15**(2), pp.131-150
- ZHOU, Y., & LEVY, JI. (2007). Factors influencing the spatial extent of mobile source air pollution impacts: A meta-analysis. *BMC Public Health*. **7**(89)

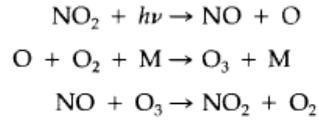
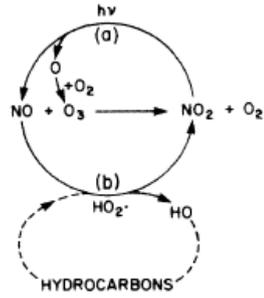
APPENDIX A



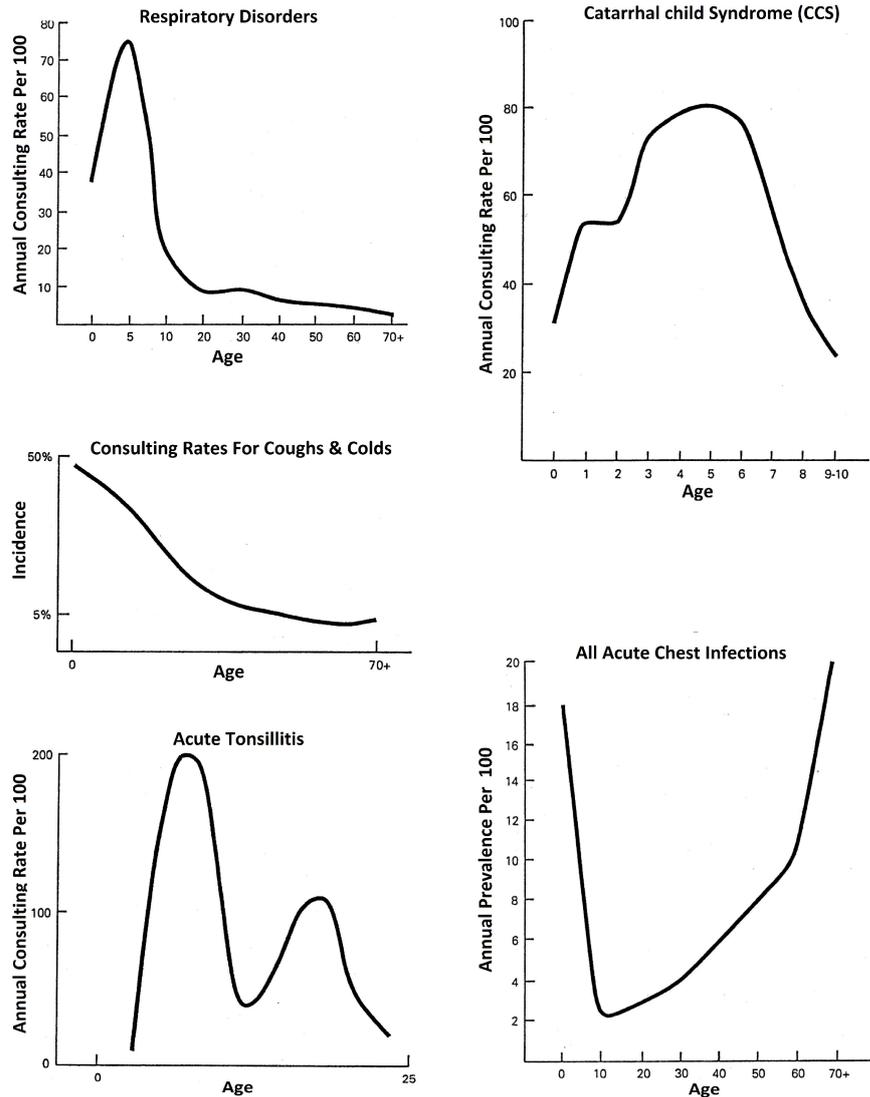
APPENDIX A1: Daily air pollution and deaths during the 1952 London episode (Boubel et al 1994)



APPENDIX A2: A correlation of daily nitrogen dioxide mixing ratios recorded by urban networks (background sources) and equipment fitted to public transport (traffic sources) in Copenhagen. Strong linear relations were also identified for half-hour measurements of roadside particulates and NO_x (Hertel et al 2001)



APPENDIX A3: Tropospheric Photochemical cycle of NO, NO₂, O₃ and free radicals (Boubel et al 1994)



APPENDIX A4: Annual patient consulting rates for various respiratory complaints within a Greater London general practice, depicting the rich amalgamation of conditions that occur almost exclusively during childhood (Adapted from Fry & Sandler 1993)

APPENDIX A5 [PAGE 368]: Descriptive overview of the studies identified for inclusion within Ward & Ayres (2004) review of particulate pollutants impact on children's lung function, as measured by peak expiratory flow (PEF)

Authors	Study Setting	Age Group [Sample]	Symptomatic Participants	24-hour PM ₁₀ µg/m ³	Time-series: Linear Model	Analytical Approach	Model Parameters*
Pope et al (1991)	Utah Valley, USA Dec 1989 - Mar 1990	9-11 years [41]	Unselected	Max: 195 Mean: 46	Single Pollutant	[A]	
Pope & Dockery (1992)	Utah Valley, USA Dec 1990 - Mar 1991	9-11 years [79]	49%	Max: 251 Mean: 76	Single Pollutant	[A]	
Hoek & Brunekreef (1994)	The Netherlands Nov-Feb 1987/88/89/90	9-11 years [225-351]	Unselected	Max: 126 Mean: 45	Single Pollutant	[B]	[I]
Roemer et al (1993)	The Netherlands Nov 1990 – Feb 1991	6-12 years [73]	100%	Max: 113 Mean: 42	Single Pollutant	[B]	[I]
Romieu et al (1996)	Mexico City Apr 1991 - Feb 1992	5-13 years [71]	100%	Max: 363 Mean: 167	Multi-Pollutant: O ₃	[B]	
Peters et al (1996)	Eastern Europe [3 Cities] Sept - Mar 1990/91/92	7-15 years [163]	100%	Max: 171 - 247 Mean: 55 - 64	Single Pollutant	[A]	[I, II, IV]
Gielen et al (1997)	The Netherlands Apr - Jul 1995	7-13 years [61]	77%	Max: 60 Mean: 31	Single Pollutant	[C]	[IV]
Vedal et al (1998)	Vancouver, Canada May 1990 - Mar 1992	6-13 years [206]	64%	Max: 159 Mean: 27	Single Pollutant	[C]	
Gold et al (1999)	Mexico City Jan - Nov 1991	8-11 years [40]	53%	Max: 87 Mean: 49	Multi-Pollutant: O ₃	[A]	[II, III]
Neas et al (1999)	Philadelphia, USA Jul - Sept 1993	6-11 years [156]	Unselected	Max: 50 Mean: 32	Single Pollutant	[D]	
Jalaludin et al (2000)	Sydney, Australia Feb - Dec 1994	7-10 years [125]	100%	Max: 123 Mean: 23	Multi-Pollutant: O ₃	[D]	[II, III]
Roemer et al (1998)	PEACE Project [14 European Cities] Nov 1993 - Feb 1994	7-11 years [2,010]	100%	Max: 29 - 242 Mean: 11 - 99	Single Pollutant	[E]	[II, IV]
Ward et al (2002)	West Midlands, UK Jan - Jul 1997	9-10 years [132]	24%	Max: 46 Mean: 20	Single Pollutant	[A]	[III, IV]

* All models include the following parameters: linear trend, temperature, autocorrelation

Footnotes: [A] Participant daily mean PEF deviation; [B] Participants daily mean PEF: z-scores; [C] Two-stage Approach: Subject specific PEF, Weighted polling
[D] Participant daily mean % change in PEF x300; [E] Meta-analysis of panel participant daily mean PEF deviation
[I] Day-of-study; [II] Quadratic trends; [III] Weather variables; [IV] Weekend indicator

Author	Location	Age: Years	Year/Condition: Last 12 Months	Total Participants	Control-Road Distance	Near-Road Distance	Odds Ratio
Wjst et al (1993)	Munich, Germany	9-11	1989: [G]	6,537	≤2km ≥25,000 vehicles/day	≤2km ≥50,000 vehicles/day	1.08 [1.01, 1.16]
Duhme et al (1996)	Munster, Germany	12-15	1994: [G]	3,703	Residential Street No Truck Traffic	Residential Street Constant Truck Traffic	2.47 [1.74, 3.52]
Keil et al (1996)	Bochum, Germany	12-15	1991: [G]	2,050	Residential Street No Truck Traffic	Residential Street Constant Truck Traffic	1.94 [1.26, 2.99]
Oosterlee et al (1996)	Haarlem, Netherlands	0-15	1991: [G]	291	Residential Street: ≥10,000 vehicles/day	Residential Street: ≥30,000 vehicles/day	2.1 [0.99, 4.40]
Ciccone et al (1998)	Italy	6-14	1994: [D]	10,955	Residential Street	Residential Street HGV Route	1.29 [1.15, 1.45]
Venn et al (2001)	Nottingham, UK	4-11	1995: [G]	6,147	150m	<30m	1.34 [0.93, 1.89]
Venn et al (2001)	Nottingham, UK	11-16	1995: [G]	3,709	150m	<30m	1.82 [1.06, 3.22]
Janssen et al (2003)	Netherlands	7-12	1997: [B]	2,037	400m: Highway	100m	1.21 [0.87, 1.68]
Janssen et al (2003)	Netherlands	7-12	1997: [B]	2,037	400m: Highway	100m	1.18 [0.60, 2.31]
Shima et al (2003)	Japan	6-9	1992-95: [E]	640	Rural Area	<50m Highway	3.77 [1.00, 14.16]
Gauderman et al (2005)	California, USA	10	1993-2000: [A]	208	Minor Roads	Freeways	2.22 [1.36, 3.36]
Ryan et al (2005)	Cincinnati, USA	≤1	2003: [G]	446	400m	<100m: Bus/HGV Route	2.50 [1.15, 5.24]
McConnel et al (2006)	California, USA	5-7	2003: [A]	4,742	300m	<75m	1.50 [1.16, 1.95]
McConnel et al (2006)	California, USA	5-7	2003: [A]	1,856	300m Life-term residents	<75m Life-term residents	1.64 [1.10, 2.44]

Author	Location	Age: Years	Year/Condition: Last 12 Months	Total Participants	Control-Road Distance	Near-Road Distance	Odds Ratio
Kim et al (2008)	California, USA	8-10	2001: [F]	1,080	300m	<300m Downwind-Highway	1.42 [0.87, 2.33]
Kim et al (2008)	California, USA	8-10	2001: [F]	1,081	300m	<300m Upwind-Highway	1.13 [0.66, 1.95]
Pujades-Rodriguez et al (2009)	England	2-6	1995/96, 2001: [E]	3,500	150m	<30m	1.17 [0.71, 1.93]
Pujades-Rodriguez et al (2009)	England	7-15	1995/96, 2001: [E]	6,015	150m	<30m	1.35 [0.95, 1.91]
Middleton et al (2010)	Nicosia, Cyprus	7-8	1999: [G]	1,917	300m	<50m	1.30 [0.86, 1.97]
Miyake et al (2010)	Neyagawa, Japan	≤1	2001-03: [A]	756	200m	<50m	4.01 [1.44, 11.24]
Andersson et al (2011)	Lulea, Sweden	7-8	2006: [A]	1,357	200m ≥100 HGV's/Day	<200m	1.29 [0.80, 2.08]
Andersson et al (2011)	Lulea, Sweden	7-8	2006: [A]	1,357	200m ≥250 HGV's/Day	<200m	1.74 [1.06, 2.87]
MacIntyre et al (2011)	Vancouver, Canada	0-2	1999: [C]	45,513	150m Highway	<50m	1.11 [0.97, 1.25]

Footnotes:

[A] Doctor-diagnosed asthma; [B] Doctor-diagnosed Bronchitis; [C] Doctor-diagnosed Otitis Media;
[D] Parent-reported Acute Respiratory Disease; [E] Parent-reported Asthma; [F] Parent-reported bronchitis; [G] Parent-reported wheeze

APPENDIX A6 [PAGES 369-370]: Literature review of the associations between proximity based exposures to road-transport and children's self/clinician diagnosed minor ailments of the respiratory system

APPENDIX B

ROW LABELS	ANNUAL LLSOA J00-99 ADMISSION COUNTS: 2000-09										
	2000	2001	2002	2003	2004	2005	2006	2007	2008	2009	MEAN
E01013761	21	17	27	34	29	42	46	35	41	36	32.8
E01013600	36	47	33	23	36	20	21	22	37	19	29.4
E01013755	14	18	23	25	25	31	56	33	32	31	28.8
E01013691	25	28	35	23	19	29	38	27	31	20	27.5
E01013640	14	29	19	19	23	35	26	32	38	34	26.9
E01013652	31	28	23	34	24	27	12	30	30	29	26.8
E01013701	18	21	9	12	10	28	33	37	47	38	25.3
E01013620	24	26	17	25	20	16	30	29	31	34	25.2
E01013748	28	25	24	30	23	25	14	26	28	24	24.7
E01013632	28	21	14	20	23	26	26	32	23	33	24.6
E01013676	26	42	21	18	23	28	19	21	15	33	24.6
E01013731	39	30	34	22	22	22	23	16	17	18	24.3
E01013754	24	14	25	21	30	21	20	16	32	26	22.9
E01013638	15	21	27	25	19	24	22	21	31	23	22.8
E01013619	11	32	31	16	21	18	20	31	29	18	22.7
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E01013609	6	6	8	3	2	6	2	5	11	4	5.3
E01013650	5	7	8	2	4	6	1	7	6	5	5.1
E01013784	8	4	2	6	5	4	5	5	6	6	5.1
E01013666	7	7	7	3	2	4	3	4	6	7	5
E01013774	3	5	6	7	2	5	2	6	6	7	4.9
E01013779	2	4	7	2	13	1	5	6	4	3	4.7
E01013710	7	3	5	1	4	6	3	5	5	7	4.6
E01013776	4	1	4	2	4	3	3	2	6	13	4.2
E01013782	3	1	3	7	8	2	1	7	6	4	4.2
E01013781	8	4	1	4	3	2	3	3	4	5	3.7
E01013644	4	1	2	0	1	5	5	6	4	6	3.4
E01013649	2	4	4	1	1	4	8	4	4	2	3.4
E01013711	3	3	4	7	2	3	2	4	2	2	3.2
E01013712	3	1	1	7	5	0	3	2	3	5	3
E01013645	0	1	1	0	0	3	3	2	6	5	2.1
TOTAL	2555	2614	2617	2424	2096	2416	2277	2360	2612	2584	2455.5

APPENDIX B1: A sample of LLSOA raw hospital admission counts, showing the upper and lower tails of the spectrum for ICD-10: J00-99 respiratory conditions

- APPENDIX B -

ROW LABELS	ANNUAL LLSOA J00-06 ADMISSION COUNTS: 2000-09										
	2000	2001	2002	2003	2004	2005	2006	2007	2008	2009	MEAN
E01013761	8	9	11	19	16	23	23	21	20	19	16.9
E01013755	8	6	9	10	13	12	31	16	15	16	13.6
E01013600	13	17	13	12	17	10	9	8	18	8	12.5
E01013640	7	13	9	10	15	10	13	18	13	17	12.5
E01013748	17	13	9	14	9	16	9	13	12	13	12.5
E01013676	10	25	9	7	9	14	7	8	11	14	11.4
E01013652	11	16	12	11	9	10	8	15	14	7	11.3
E01013654	8	13	13	14	12	9	12	13	7	11	11.2
E01013754	8	7	13	8	10	11	6	8	21	14	10.6
E01013701	4	10	6	6	6	10	8	14	21	20	10.5
E01013679	12	15	16	12	11	7	10	4	9	6	10.2
E01013651	17	4	10	15	6	6	10	7	12	10	9.7
E01013602	7	11	12	6	7	11	5	9	13	12	9.3
E01013621	3	15	9	13	13	3	7	15	4	11	9.3
E01013658	11	8	6	9	8	12	8	5	10	16	9.3
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E01013609	1	2	1	1	1	3	2	3	6	2	2.2
E01013644	3	1	1	0	0	3	3	4	4	3	2.2
E01013666	2	2	4	0	1	2	2	3	2	4	2.2
E01013715	1	5	3	6	1	1	1	0	2	2	2.2
E01013641	3	3	7	0	1	2	3	0	1	1	2.1
E01013711	2	3	1	5	1	1	2	2	2	1	2.0
E01013781	4	1	0	1	3	1	3	2	3	2	2.0
E01013782	1	0	2	5	2	1	1	2	2	3	1.9
E01013710	2	1	1	1	1	4	1	1	3	3	1.8
E01013734	0	2	1	3	1	4	1	2	4	0	1.8
E01013636	3	2	4	1	0	1	0	2	0	3	1.6
E01013649	1	2	1	1	1	4	1	3	1	1	1.6
E01013735	1	2	0	5	1	1	0	2	1	3	1.6
E01013712	0	0	0	4	1	0	3	1	2	3	1.4
E01013645	0	0	1	0	0	1	2	0	1	3	0.8
TOTAL	931	1042	1038	1034	761	888	1025	1101	1215	1193	1022.8

APPENDIX B2: A sample of LLSOA raw hospital admission counts, showing the upper and lower tails of the spectrum for ICD-10: J00-06 respiratory conditions

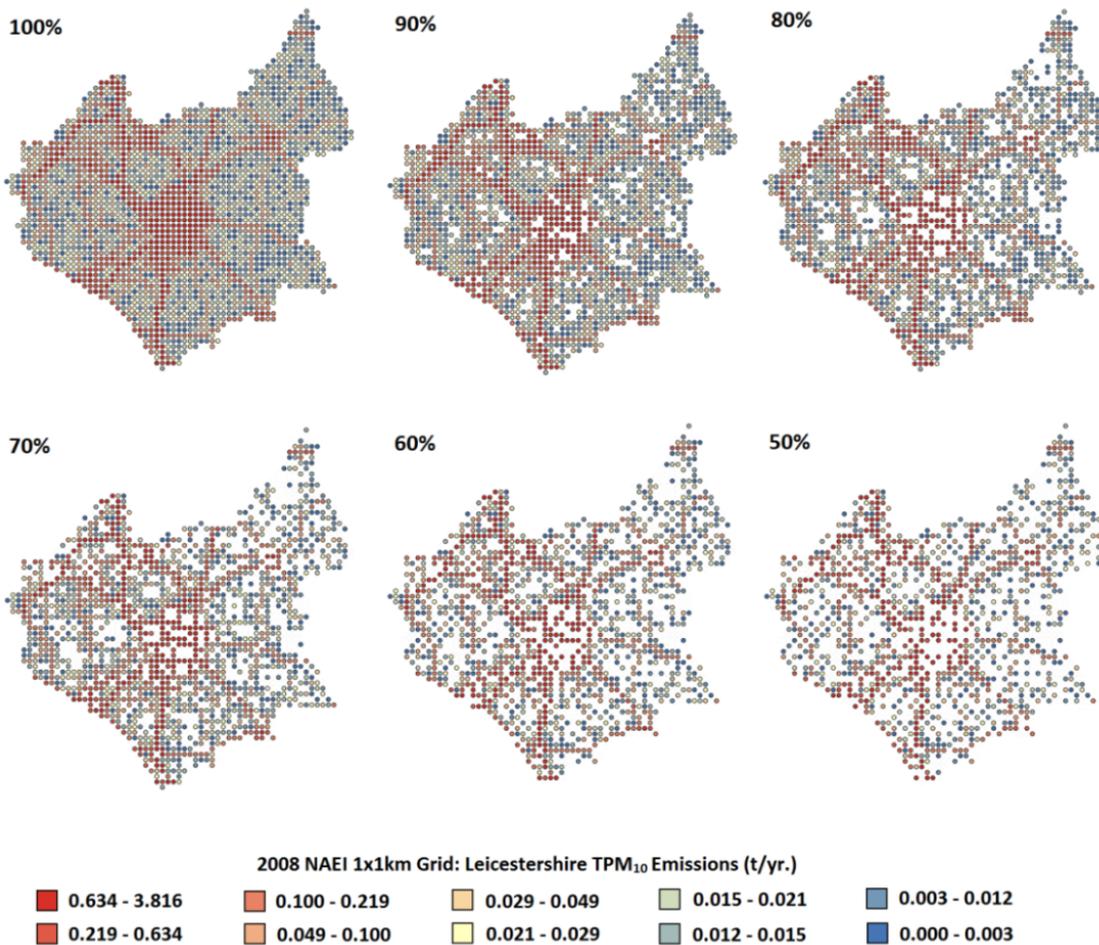
- APPENDIX B -

ROW LABELS	ANNUAL LLSOA J20-22 ADMISSION COUNTS: 2000-09										
	2000	2001	2002	2003	2004	2005	2006	2007	2008	2009	MEAN
E01013755	1	6	9	7	4	8	10	9	8	10	7.2
E01013640	0	4	3	4	7	15	5	6	14	10	6.8
E01013632	5	4	0	3	9	8	9	10	10	7	6.5
E01013652	7	4	2	7	7	9	0	7	9	13	6.5
E01013761	5	1	10	5	6	8	7	3	8	8	6.1
E01013731	5	3	4	3	8	7	9	9	6	4	5.8
E01013676	3	3	3	6	7	7	6	8	1	11	5.5
E01013701	4	4	2	3	0	10	6	12	10	4	5.5
E01013600	5	5	5	3	8	5	3	6	6	6	5.2
E01013620	6	5	2	1	4	5	6	5	9	7	5.0
E01013621	4	1	6	6	9	6	2	6	7	3	5.0
E01013691	3	4	8	1	4	6	5	4	6	6	4.7
E01013754	9	2	4	2	9	3	5	3	4	5	4.6
E01013637	5	2	7	2	0	7	4	3	8	5	4.3
E01013692	4	9	1	4	6	2	2	2	6	6	4.2
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E01013645	0	1	0	0	0	2	0	1	2	1	0.7
E01013669	1	0	0	0	1	1	0	0	4	0	0.7
E01013710	0	1	0	0	1	0	1	1	1	2	0.7
E01013609	0	1	1	0	0	0	0	1	2	1	0.6
E01013672	0	0	0	0	0	1	2	1	2	0	0.6
E01013700	1	0	0	1	0	0	0	2	1	1	0.6
E01013712	0	0	0	3	2	0	0	0	0	1	0.6
E01013783	0	0	0	0	1	1	1	3	0	0	0.6
E01013606	0	0	1	0	0	2	0	2	0	0	0.5
E01013735	0	0	0	1	1	1	1	0	0	1	0.5
E01013779	0	0	2	0	0	0	0	2	0	1	0.5
E01013666	1	1	1	0	0	0	0	0	1	0	0.4
E01013711	0	0	1	0	0	1	0	2	0	0	0.4
E01013643	0	1	0	1	0	0	1	0	0	0	0.3
E01013649	1	1	0	0	0	0	1	0	0	0	0.3
TOTAL	357	377	362	307	361	485	318	422	574	579	414.2

APPENDIX B3: A sample of LLSOA raw hospital admission counts, showing the upper and lower tails of the spectrum for ICD-10: J20-22 respiratory conditions

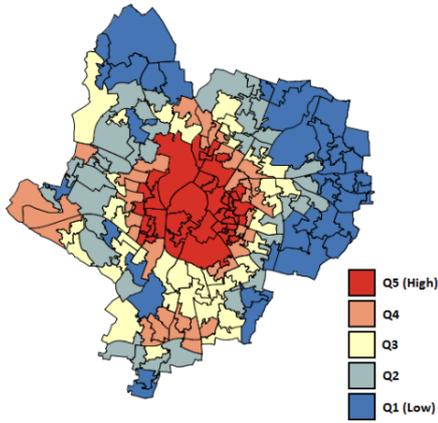
J00-99 Admissions Per 1,000 Children	Year	Children's J00-99 Admission Count	R ²
2001 UK Census Vs. 2002 Mid-Year Estimate	2002	2,617	0.96
2001 UK Census Vs. 2003 Mid-Year Estimate	2003	2,424	0.93
2001 UK Census Vs. 2004 Mid-Year Estimate	2004	2,096	0.92
2001 UK Census Vs. 2005 Mid-Year Estimate	2005	2,416	0.90
2001 UK Census Vs. 2006 Mid-Year Estimate	2006	2,277	0.84
2001 UK Census Vs. 2007 Mid-Year Estimate	2007	2,360	0.79
2001 UK Census Vs. 2008 Mid-Year Estimate	2008	2,612	0.77

APPENDIX B4: Discrepancies in Leicester UA LLSOA J00-99 admissions per 1,000 children caused by standardisation in the form of ONS 2001 UK Census population counts and ONS mid-year population estimates: 2002-2008

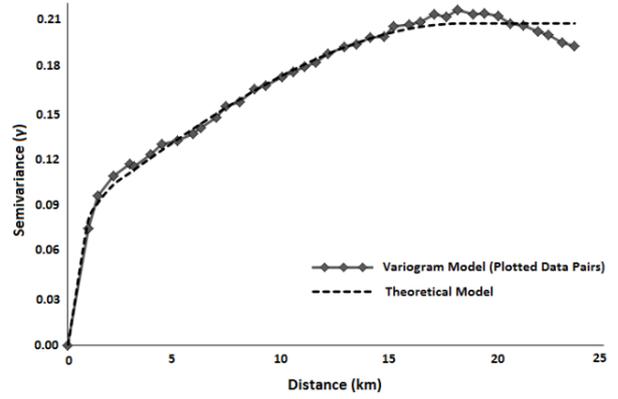


APPENDIX B5: Progressively omitted fraction of the 2,157 datum points used by the validity kriging models, interpolating the 2008 1x1km PM₁₀ road-transport emission grid of Leicestershire

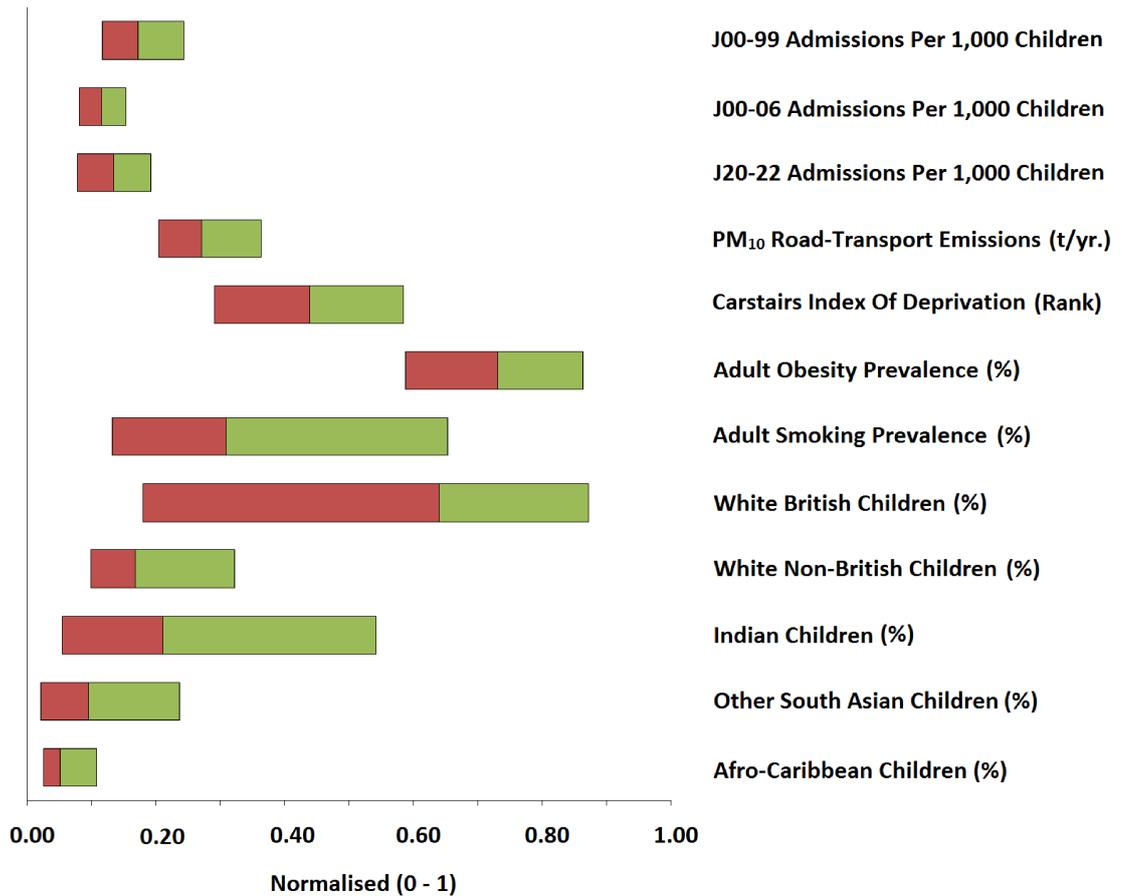
TPM₁₀ Kriging Estimate (Quantiles)



NAEI 1x1km TPM₁₀ Emission Grid: Leicestershire County 2008



APPENDIX B6: Display of the fit between the Leicestershire theoretical 2008 TPM₁₀ emission model and traditional variogram plot, alongside the interpolated output for Leicester UA

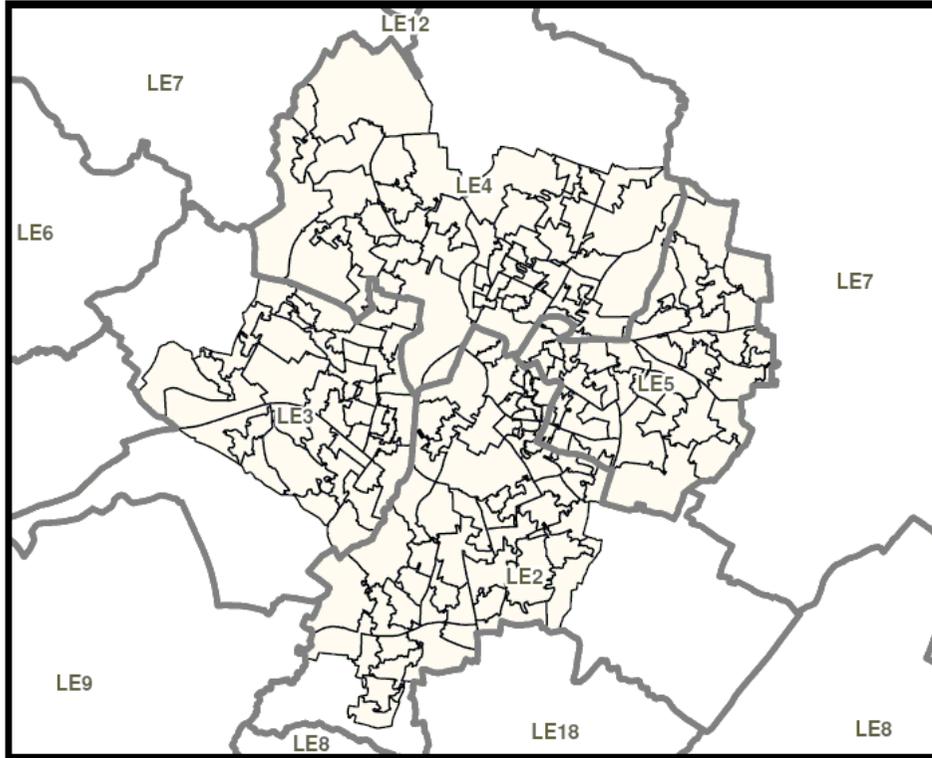


APPENDIX B7: Box-Whisker plots of social-lifestyle, health and environmental factors recorded across Leicester UA

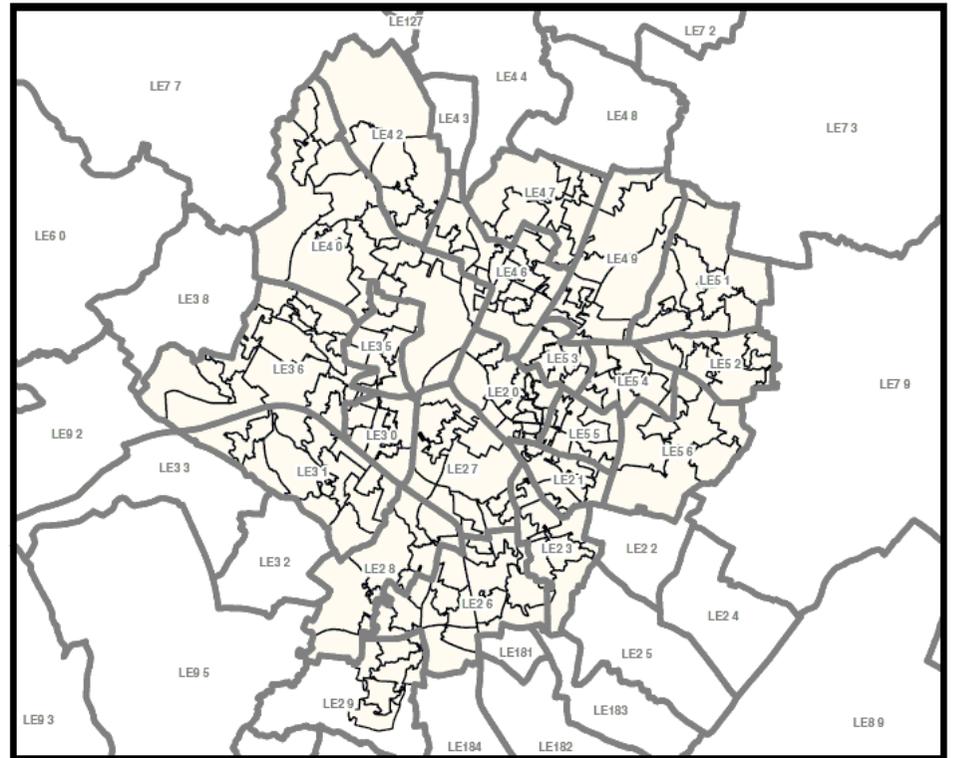
		Y: THEORETICAL MODEL											
		[A]	[B]	[C]	[D]	[E]	[F]	[G]	[H]	[I]	[J]	[K]	[L]
X: PLOTTED VARIOGRAM POINTS	J00-99 Admissions Per 1,000 Children [A]	0.92	0.92	0.91	0.78	0.56	0.65	0.67	0.47	0.91	0.48	0.60	0.92
	J00-06 Admissions Per 1,000 Children [B]	0.94	0.94	0.93	0.82	0.59	0.68	0.71	0.48	0.93	0.49	0.63	0.94
	J20-22 Admissions Per 1,000 Children [C]	0.89	0.90	0.90	0.75	0.57	0.63	0.65	0.50	0.90	0.51	0.60	0.90
	Carstairs Index: Leicester [D]	0.81	0.79	0.73	0.93	0.79	0.87	0.89	0.66	0.73	0.66	0.83	0.77
	TPM ₁₀ Emissions [E]	0.65	0.63	0.55	0.90	0.99	0.98	0.97	0.96	0.55	0.96	0.99	0.59
	Adult Smoking Prevalence [F]	0.67	0.65	0.59	0.83	0.91	0.88	0.87	0.94	0.59	0.94	0.90	0.63
	Adult Obesity Prevalence [G]	0.73	0.70	0.63	0.96	0.96	0.98	0.98	0.90	0.63	0.90	0.97	0.67
	White British Children [H]	0.54	0.52	0.45	0.81	0.98	0.93	0.91	0.99	0.45	0.99	0.96	0.49
	White Non-British Children [I]	0.90	0.90	0.92	0.76	0.65	0.69	0.71	0.59	0.92	0.60	0.67	0.91
	Indian Children [J]	0.60	0.58	0.52	0.83	0.97	0.93	0.92	0.99	0.52	0.99	0.96	0.55
	Other South Asian Children [K]	0.68	0.66	0.59	0.93	0.99	0.99	0.99	0.95	0.59	0.95	0.99	0.63
	Afro-Caribbean Children [L]	0.91	0.92	0.93	0.75	0.61	0.67	0.68	0.53	0.93	0.54	0.64	0.93

APPENDIX B8: Comparing the goodness-of-fit (R-squared) of an individual variables constructed theoretical model to real-world spatial continuity measurements provided by variograms of itself and all other variables across Leicester UA

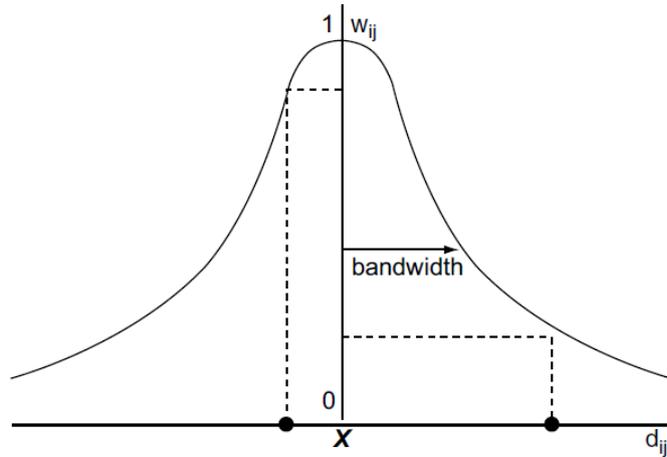
Leicester UA Postal Districts



Leicester UA Postal Sectors

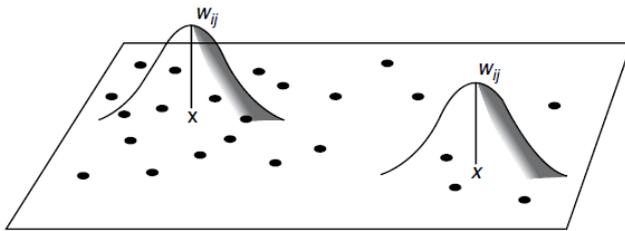


APPENDIX B9: Cartographic portrayal of how postal district and postal sector outputs correspond to the LLSOA's of Leicester UA



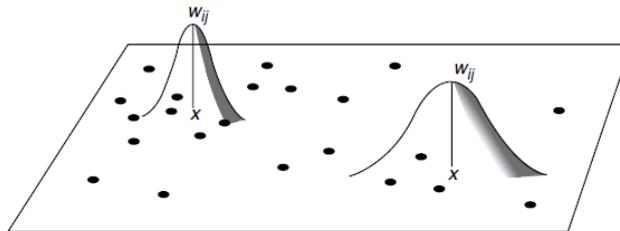
X regression point w_{ij} is the weight of data point j at regression point i
 ● data point d_{ij} is the distance between regression point i and data point j

GWR With Fixed Spatial Kernels



x regression point
 ● data point

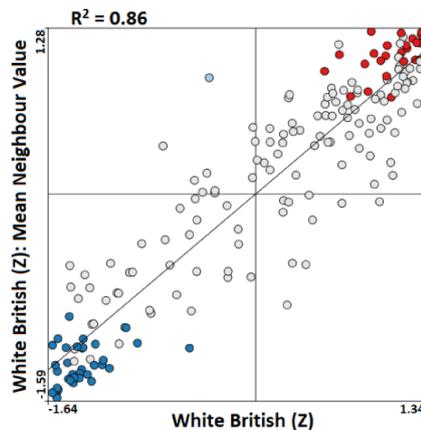
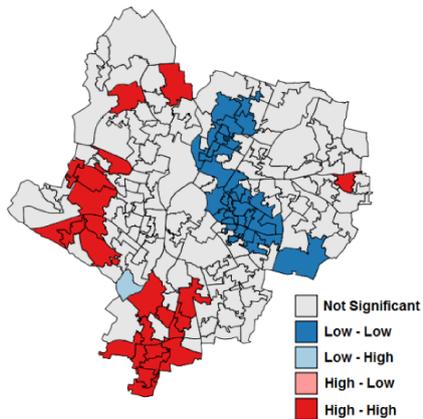
GWR With Adaptive Spatial Kernels



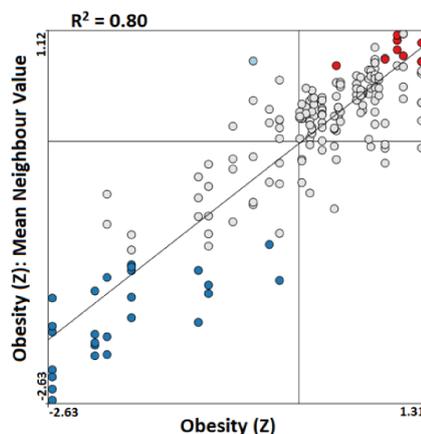
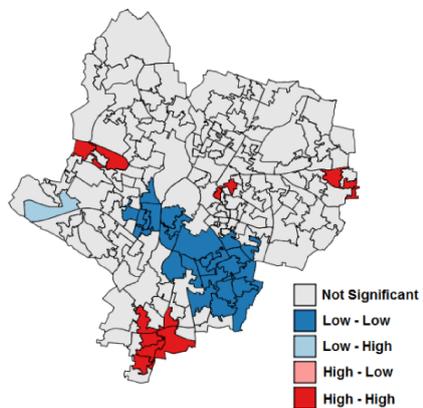
x regression point
 ● data point

APPENDIX B10: Construction of spatial kernels describing how weighting is distributed to observations based on proximity within the local regression models of a GWR analysis (Adapted from Fotheringham et al 2002)

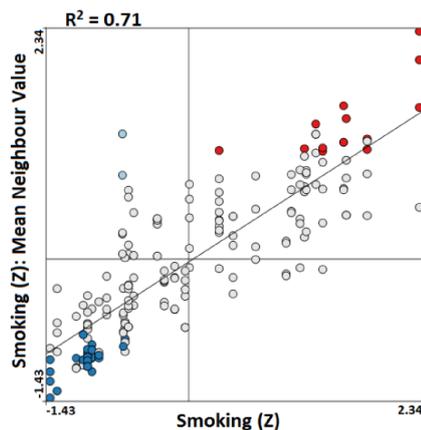
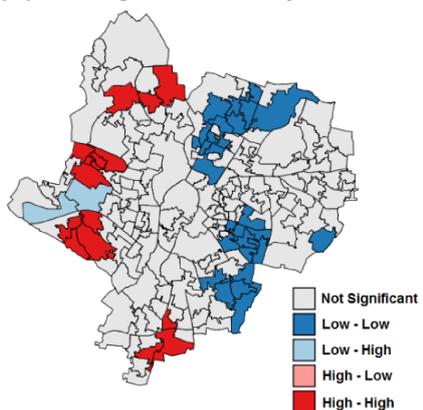
(%) White British Children



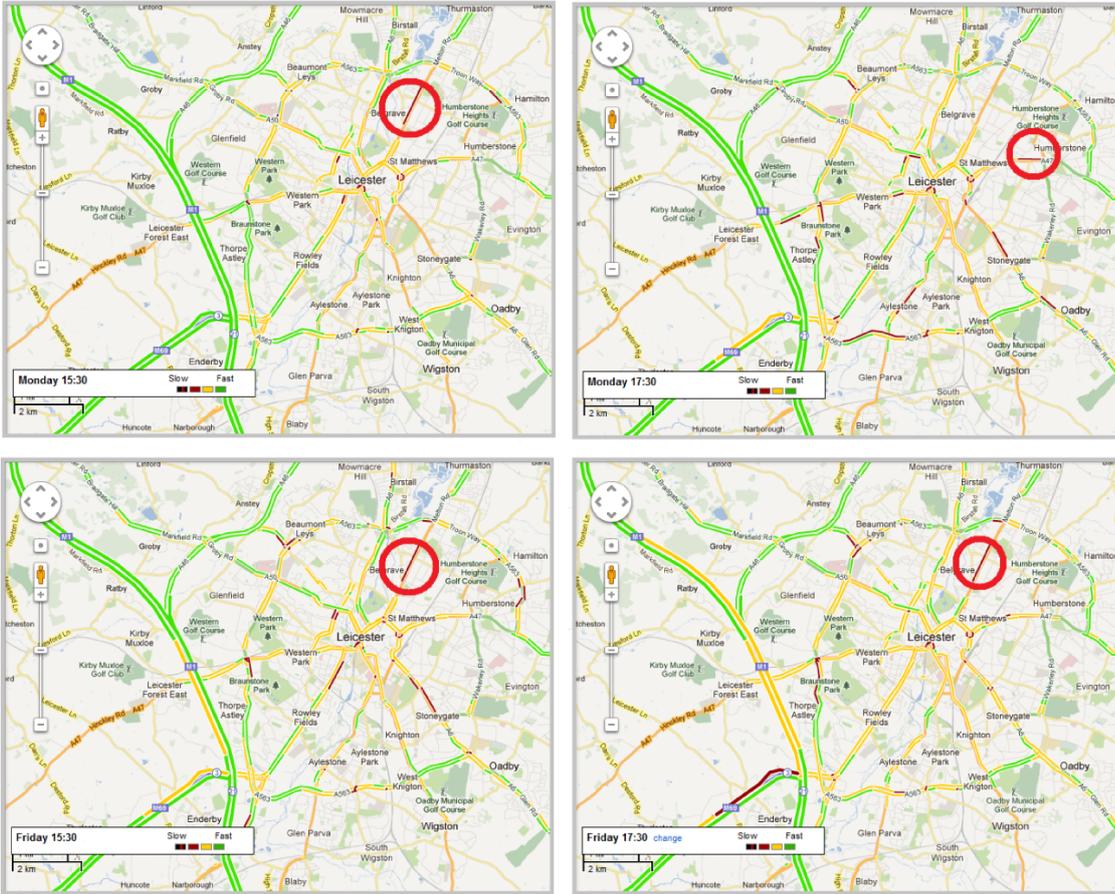
(%) Obesity Prevalence 16yrs.+



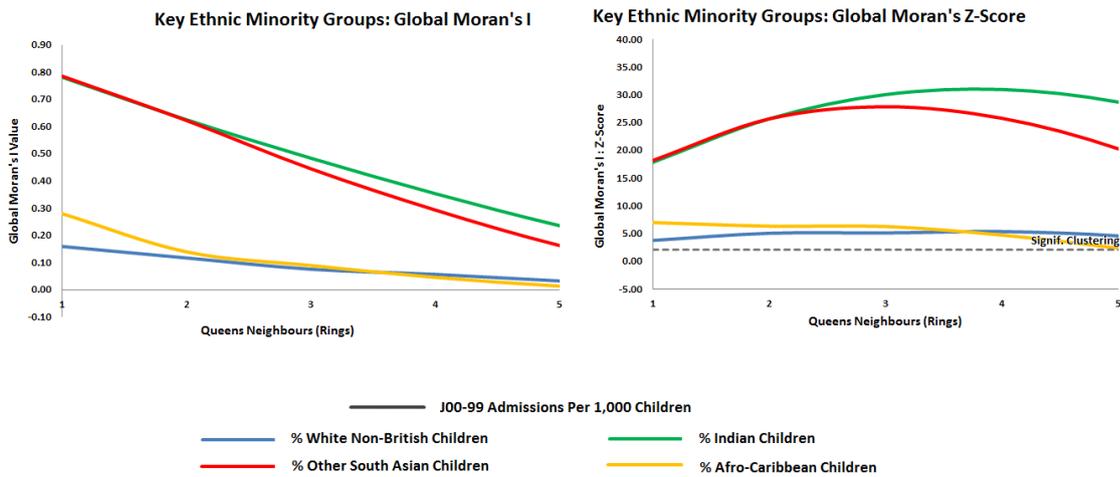
(%) Smoking Prevalence 16yrs.+



APPENDIX C1: Local Moran's I cluster and outlier analysis of 'White British' residency, and levels of smoking and obesity prevalence



APPENDIX C2: Estimated traffic flow conditions across Leicester UA during September 2012, based upon past conditions. (Upper Left): Monday 15:30, (Upper Right) Monday 17:30, (Lower Left): Friday 15:30, (Lower Right): Friday 17:30. ©Google Maps UK, 2012



APPENDIX C3: Spatial correlograms of Global Moran's statistical outputs, portraying the level of decay in autocorrelation between neighbouring LLSOA community ethnic minority levels, as a function of distance

	Health	Socio-Environmental		Adult Healthy Lifestyle (%)		Children's Social Lifestyle (%)				
	J00-99 Admissions	Carstairs Index [Leicester]	TPM₁₀ (t/yr.)	Smoking Prevalence	Obesity Prevalence	White British	White Non-British	Indian	Other S. Asian	Afro-Caribbean
J00-99 Admissions [N=7]	80.65	4.11	2.19	33.11	20.67	34.38	2.08	26.95	11.58	12.41
Carstairs Index [N=15]	50.97	3.90	1.60	23.92	26.10	20.21	1.33	53.30	13.13	5.64
TPM₁₀ Emissions [N=20]	57.15	2.68	1.82	27.70	22.46	30.49	2.22	35.21	12.92	7.58
Adult Smoking [N=14]	47.46	2.23	0.89	45.16	28.02	86.63	1.30	3.50	0.72	1.47
Adult Obesity [N=12]	43.68	1.26	0.99	37.11	28.97	74.51	0.87	15.06	2.26	1.69
White British [N=24]	47.41	0.73	0.88	39.46	27.33	86.21	1.67	3.85	0.93	1.53
White Non-British [N=5]	29.78	-2.24	1.31	27.12	17.66	51.96	3.35	25.49	5.36	4.72
Indian [N=34]	32.61	1.94	1.15	17.11	25.53	12.63	0.95	67.31	11.31	2.47
Other South Asian [N=19]	44.68	2.25	1.38	19.53	23.56	12.59	1.09	52.92	21.25	5.60
Afro-Caribbean [N=5]	49.18	3.06	1.53	17.86	23.58	13.43	1.42	41.60	25.41	8.48

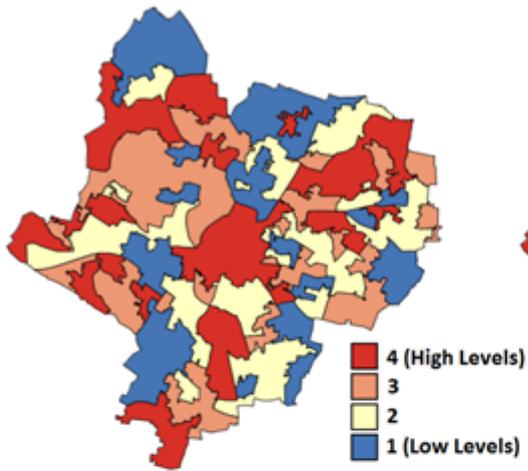
APPENDIX C4 (ABOVE): Overview of children's respiratory health and residentially experienced socio-environmental influences within Local Moran's hot-spots (H-H)

APPENDIX C5 (BENEATH): Overview of children's respiratory health and residentially experienced socio-environmental influences within Local Moran's cold-spots (C-C)

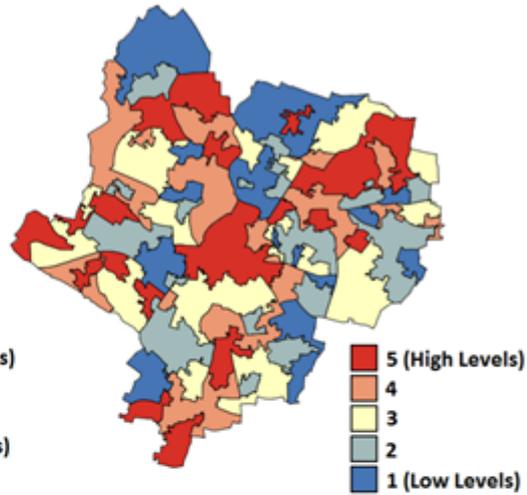
	Health	Socio-Environmental		Adult Healthy Lifestyle (%)		Children's Social Lifestyle (%)				
	J00-99 Admissions	Carstairs Index [Leicester]	TPM₁₀ (t/yr.)	Smoking Prevalence	Obesity Prevalence	White British	White Non-British	Indian	Other S. Asian	Afro-Caribbean
J00-99 Admissions [N=10]	27.64	-0.12	1.14	17.97	21.73	26.12	2.08	59.73	4.68	1.07
Carstairs Index [N=15]	30.92	-4.27	0.83	16.65	17.89	66.71	1.84	19.76	3.24	1.32
TPM₁₀ Emissions [N=19]	38.52	-2.04	0.59	24.57	26.34	63.05	1.11	24.18	2.92	1.72
Smoking Prevalence [N=28]	31.40	-0.78	0.96	15.04	22.27	24.19	1.25	57.75	9.17	1.88
Obesity Prevalence [N=26]	35.66	-2.83	1.26	23.54	15.55	59.41	2.59	19.19	5.37	2.56
White British [N=36]	35.46	2.40	1.24	17.63	25.43	11.24	1.01	65.62	13.31	3.12
White Non-British [N=4]	38.94	3.08	1.23	25.53	26.93	28.51	0.91	50.87	12.34	2.36
Indian [N=26]	45.61	0.44	0.90	38.71	26.84	86.55	1.40	3.91	0.82	1.44
Other South Asian [N=19]	44.78	0.49	0.90	39.25	26.77	81.68	1.92	6.52	1.25	1.91
Afro-Caribbean [N=6]	40.96	2.06	0.83	34.28	28.55	62.17	1.29	27.44	1.64	0.61

J00-99 Hospital Admission Per 1,000 Children

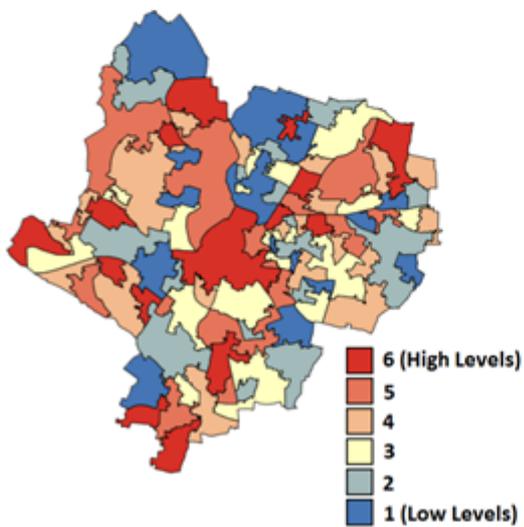
4-Quantiles



5-Quantiles



6-Quantiles



187 LLSOA's



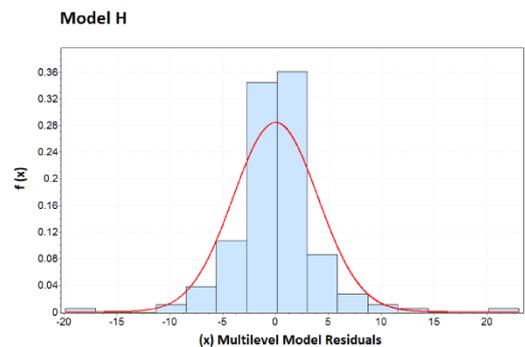
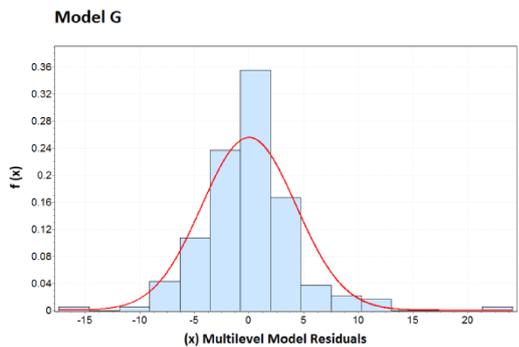
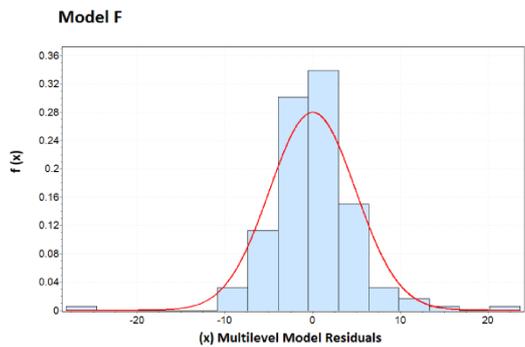
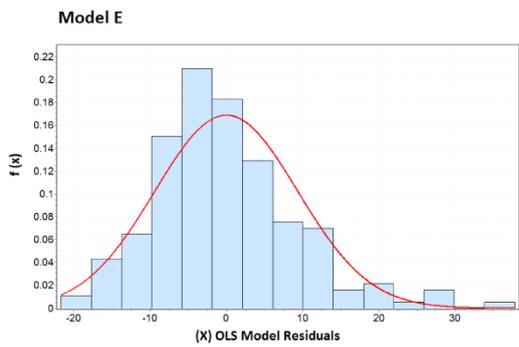
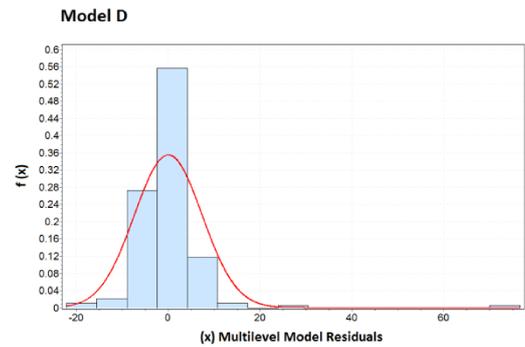
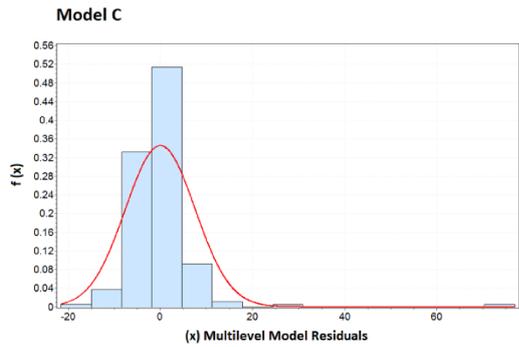
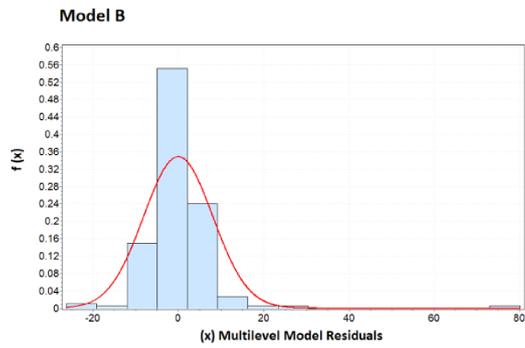
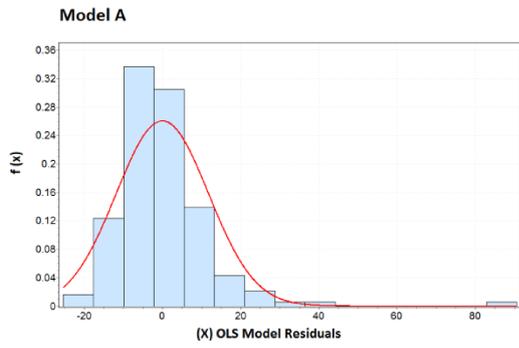
APPENDIX C6: Quantile distribution plots of 2000-09 annual average children's respiratory admissions (ICD-10: J00-99)

J00-99 Admission Rate Quantiles	Upper Level: Random Effect Intercept Value (Disease Prevalence)							
	Model A	Model B	Model C	Model D	Model E	Model F	Model G	Model H
1 (Low)		-13.66	-15.37	-16.26		-13.27	-14.67	-15.38
2		-4.98	-6.08	-7.57		-4.32	-6.18	-8.07
3		2.36	-1.76	-4.19		2.57	-1.13	-3.05
4		16.28	4.06	1.42		15.02	4.55	1.20
5			19.15	5.72			17.43	6.42
6 (High)				20.88				18.90

APPENDIX C7: Estimates of the unmeasured spatial component associated with disease prevalence, recorded within upper level nesting structures

	Fixed Intercept (Baseline)	Carstairs Index	TPM ₁₀ Emissions	Smoking Prevalence	Obesity Prevalence	White Non-British	Indian	Other South Asian	Afro- Caribbean	Random Intercept: Prevalence	Residuals
Model A	71.74	22.29	15.10	1.79	3.62	-3.63	-19.89	5.67	4.23	N/A	-0.92
Model B	85.69	3.40	17.69	-3.82	0.95	-1.35	-4.64	-0.57	1.88	1.38	-0.60
Model C	83.98	4.32	16.99	-1.64	0.49	-1.51	-2.80	-1.27	1.13	0.89	-0.58
Model D	85.72	3.47	15.81	-1.20	0.43	-1.87	-2.13	-1.92	1.37	0.85	-0.53
Model E	74.20	20.87	1.36	0.80	9.51	-1.24	-17.91	5.58	6.99	N/A	-0.14
Model F	86.99	3.30	5.26	-4.29	6.03	0.67	-3.50	-0.43	4.57	1.33	0.06
Model G	85.68	4.46	4.85	-2.48	5.17	0.55	-1.83	-1.05	3.76	0.83	0.05
Model H	87.50	3.55	3.59	-2.21	5.44	0.33	-1.52	-1.70	4.07	0.86	0.09

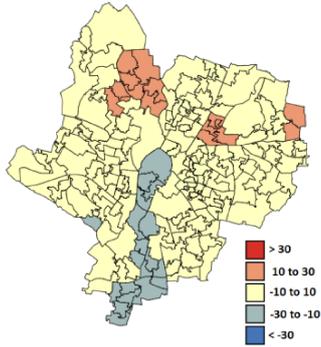
APPENDIX C8: Percentage (%) of citywide annual children's respiratory admissions associated with background and socio-environmental influences recorded within Leicester UA: 2000-09



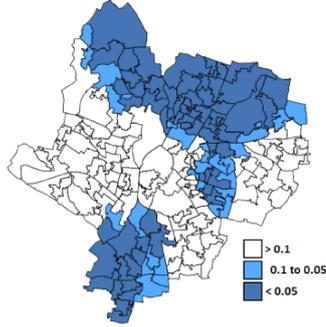
APPENDIX C9: Frequency plots of residuals from Models A-H, fitted with normal distributions

GWR 80NN: Hospitalisation Rates Associated With Smoking Prevalence

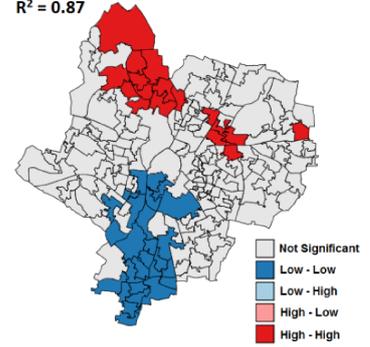
Admissions Per 1,000 Children



GWR Coefficient P-Value

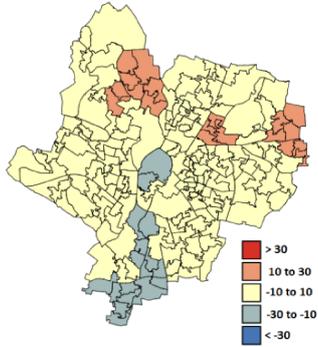


Local Moran's I: Children's Admission Rate
 $R^2 = 0.87$

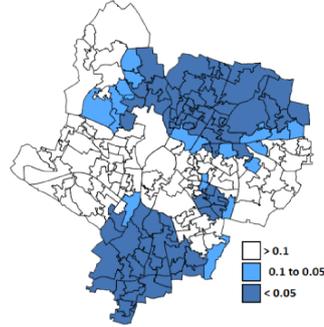


GWR 60NN: Hospitalisation Rates Associated With Smoking Prevalence

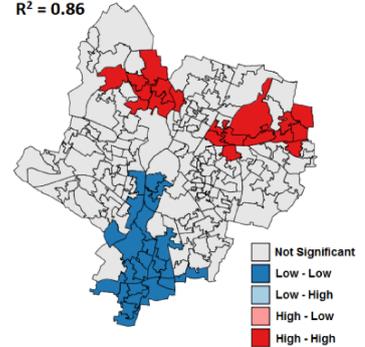
Admissions Per 1,000 Children



GWR Coefficient P-Value

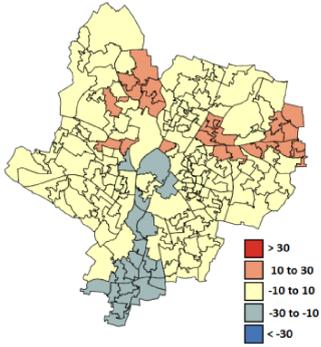


Local Moran's I: Children's Admission Rate
 $R^2 = 0.86$

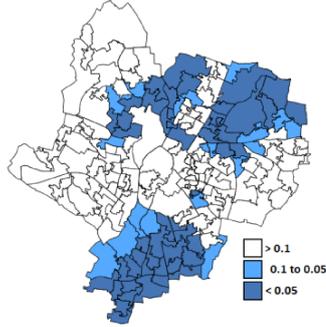


GWR 40NN: Hospitalisation Rates Associated With Smoking Prevalence

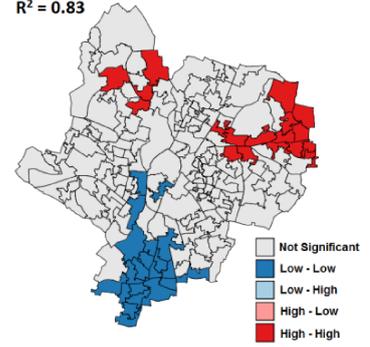
Admissions Per 1,000 Children



GWR Coefficient P-Value



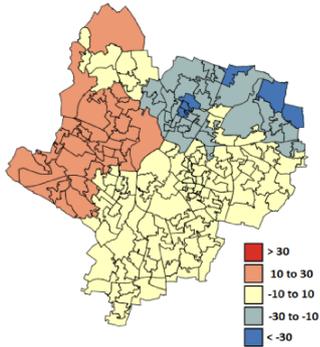
Local Moran's I: Children's Admission Rate
 $R^2 = 0.83$



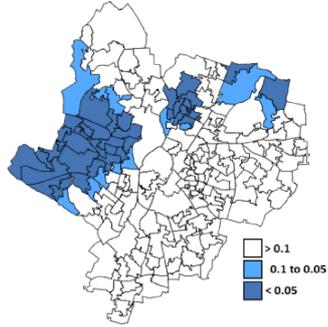
APPENDIX C10: GWR modelled children's J00-99 hospital admission rates associated with levels of passive smoking as recorded via adult smoking prevalence

GWR 80NN: Hospitalisation Rates Associated With Obesity Prevalence

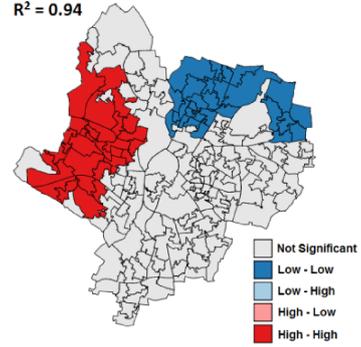
Admissions Per 1,000 Children



GWR Coefficient P-Value

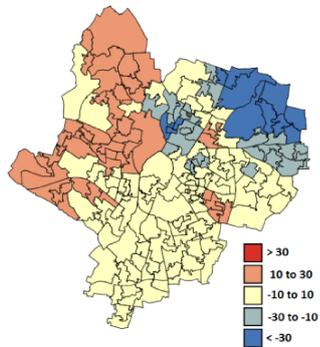


Local Moran's I: Children's Admission Rate
R² = 0.94

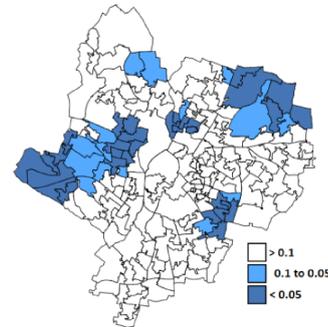


GWR 60NN: Hospitalisation Rates Associated With Obesity Prevalence

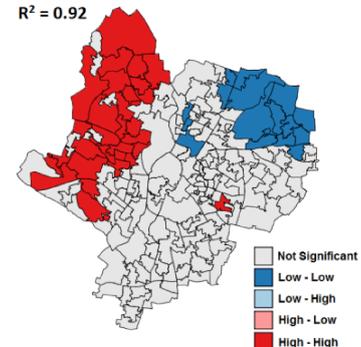
Admissions Per 1,000 Children



GWR Coefficient P-Value

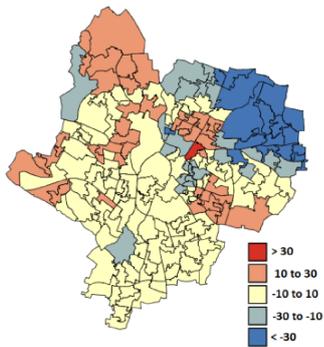


Local Moran's I: Children's Admission Rate
R² = 0.92

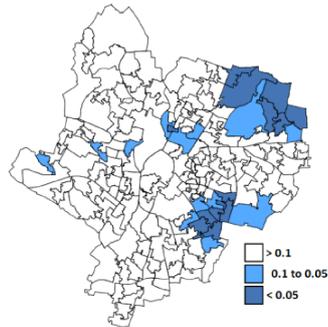


GWR 40NN: Hospitalisation Rates Associated With Obesity Prevalence

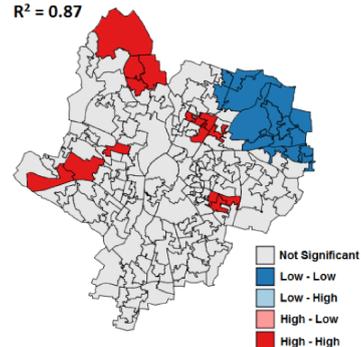
Admissions Per 1,000 Children



GWR Coefficient P-Value



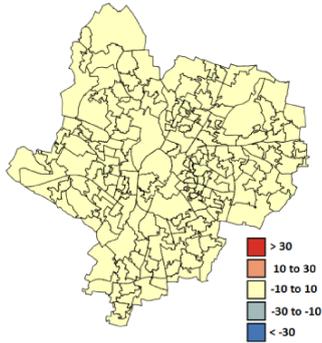
Local Moran's I: Children's Admission Rate
R² = 0.87



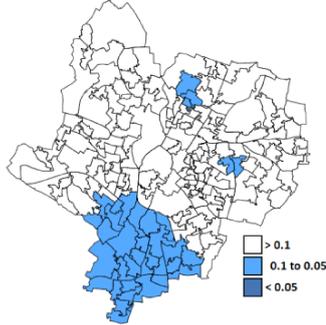
APPENDIX C11: GWR modelled children's J00-99 hospital admission rates associated with children's exercise levels and dietary intake as recorded via adult obesity prevalence

GWR 80NN: Hospitalisation Rates Associated With Levels Of White Non-British Children

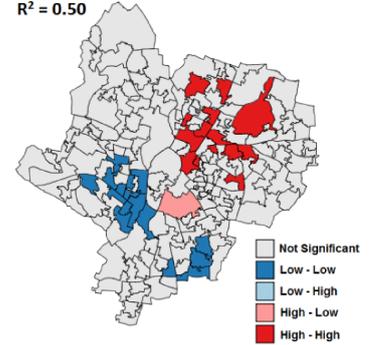
Admissions Per 1,000 Children



GWR Coefficient P-Value

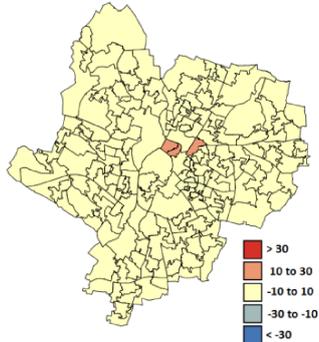


Local Moran's I: Children's Admission Rate
R² = 0.50

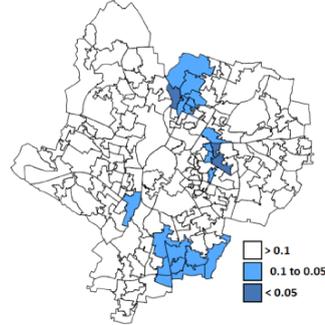


GWR 60NN: Hospitalisation Rates Associated With Levels Of White Non-British Children

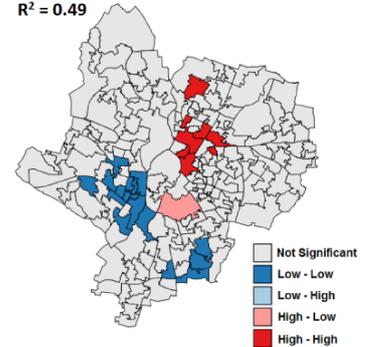
Admissions Per 1,000 Children



GWR Coefficient P-Value

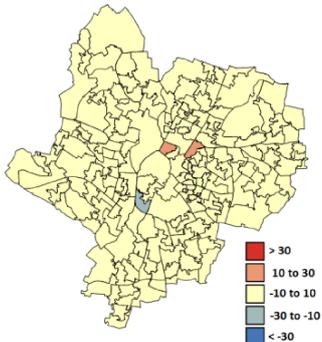


Local Moran's I: Children's Admission Rate
R² = 0.49

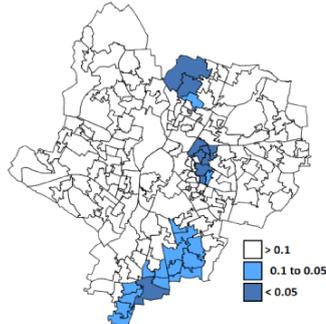


GWR 40NN: Hospitalisation Rates Associated With Levels Of White Non-British Children

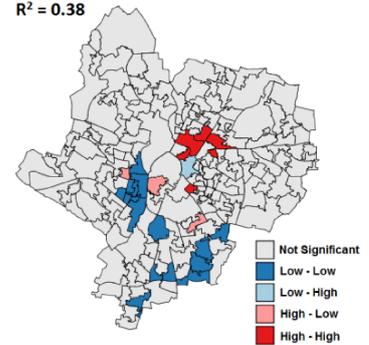
Admissions Per 1,000 Children



GWR Coefficient P-Value



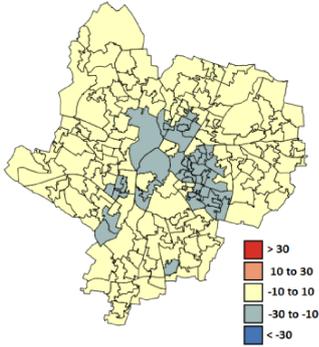
Local Moran's I: Children's Admission Rate
R² = 0.38



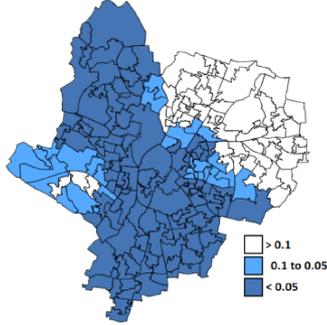
APPENDIX C12: GWR modelled children's J00-99 hospital admission rates associated with the social lifestyle of 'White Non-British' residents

GWR 80NN: Hospitalisation Rates Associated With Levels Of Indian Children

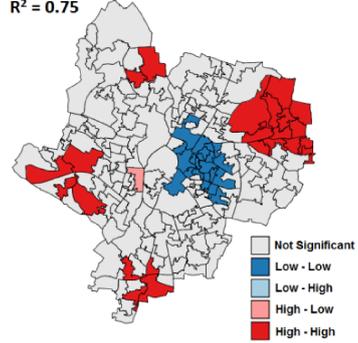
Admissions Per 1,000 Children



GWR Coefficient P-Value

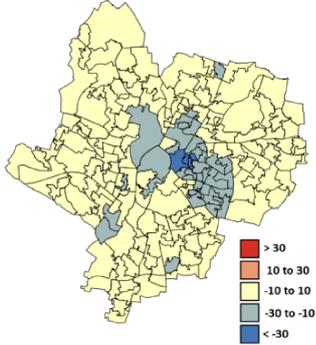


Local Moran's I: Children's Admission Rate
R² = 0.75

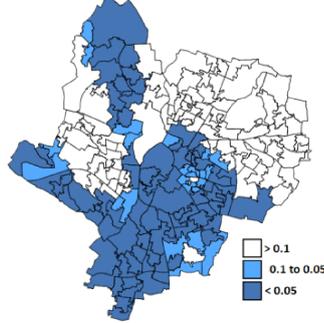


GWR 60NN: Hospitalisation Rates Associated With Levels Of Indian Children

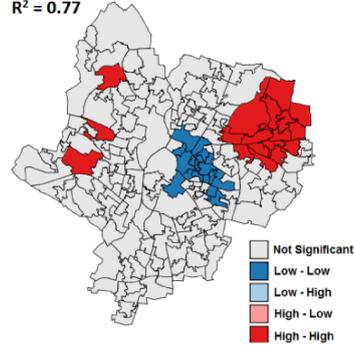
Admissions Per 1,000 Children



GWR Coefficient P-Value

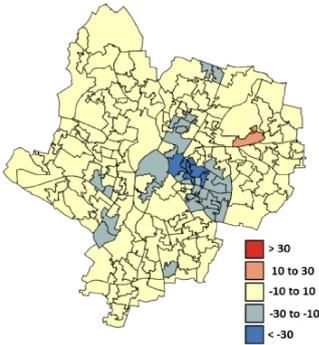


Local Moran's I: Children's Admission Rate
R² = 0.77

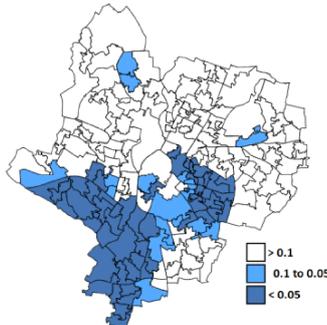


GWR 40NN: Hospitalisation Rates Associated With Levels Of Indian Children

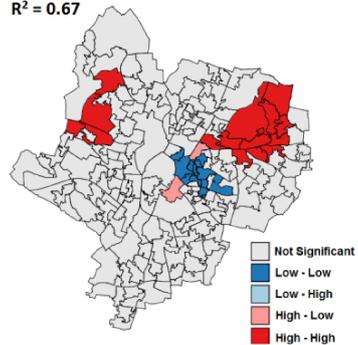
Admissions Per 1,000 Children



GWR Coefficient P-Value



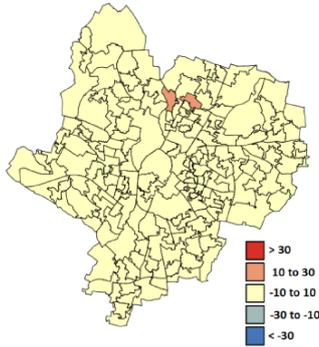
Local Moran's I: Children's Admission Rate
R² = 0.67



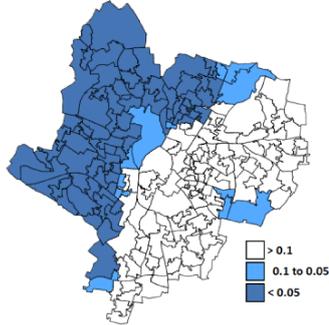
APPENDIX C13: GWR modelled children's J00-99 hospital admission rates associated with the social lifestyle of 'Indian' residents

GWR 80NN: Hospitalisation Rates Associated With Levels Of 'Other South Asian' Children

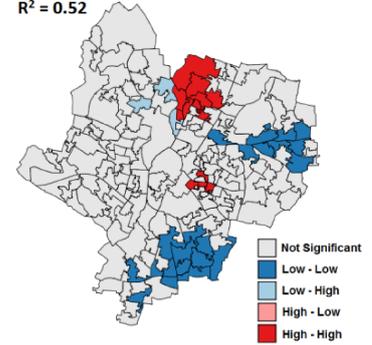
Admissions Per 1,000 Children



GWR Coefficient P-Value

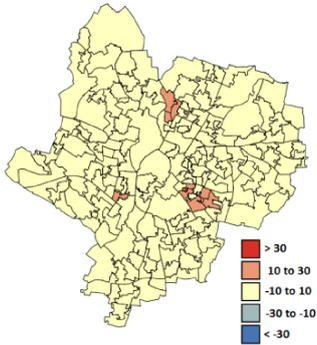


Local Moran's I: Children's Admission Rate
R² = 0.52

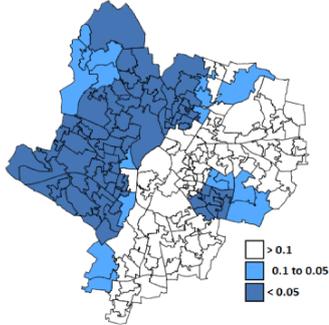


GWR 60NN: Hospitalisation Rates Associated With Levels Of 'Other South Asian' Children

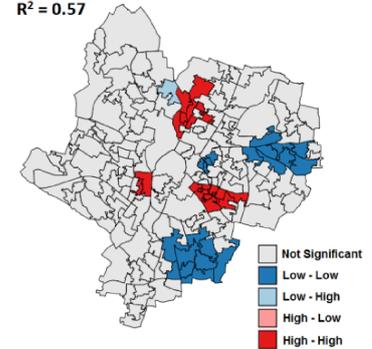
Admissions Per 1,000 Children



GWR Coefficient P-Value

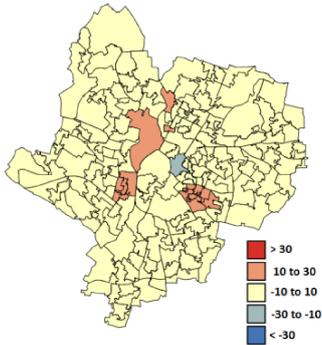


Local Moran's I: Children's Admission Rate
R² = 0.57

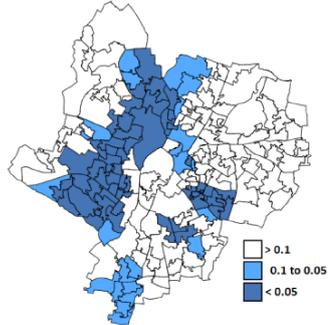


GWR 40NN: Hospitalisation Rates Associated With Levels Of 'Other South Asian' Children

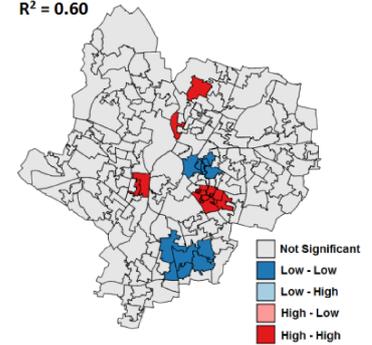
Admissions Per 1,000 Children



GWR Coefficient P-Value



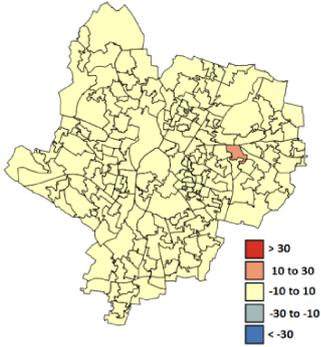
Local Moran's I: Children's Admission Rate
R² = 0.60



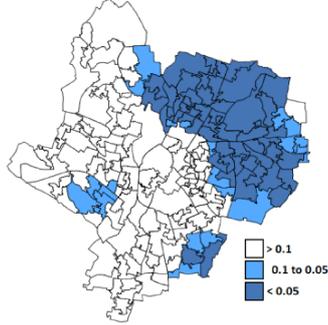
APPENDIX C14: GWR modelled children's J00-99 hospital admission rates associated with the social lifestyle of 'Other South Asian' residents

GWR 80NN: Hospitalisation Rates Associated With Levels Of Afro-Caribbean Children

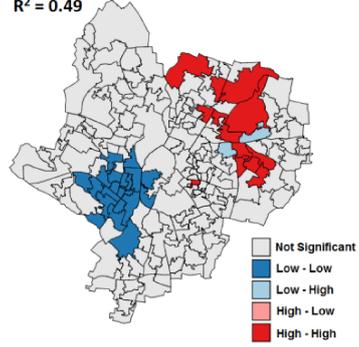
Admissions Per 1,000 Children



GWR Coefficient P-Value

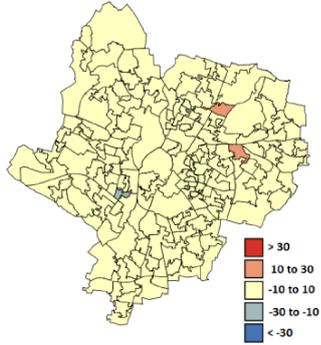


Local Moran's I: Children's Admission Rate
R² = 0.49

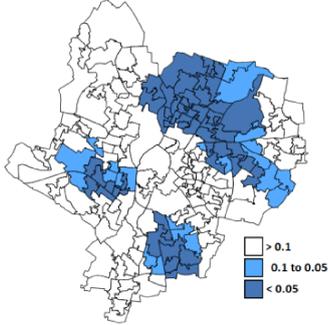


GWR 60NN: Hospitalisation Rates Associated With Levels Of Afro-Caribbean Children

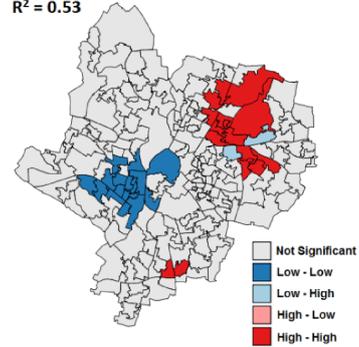
Admissions Per 1,000 Children



GWR Coefficient P-Value

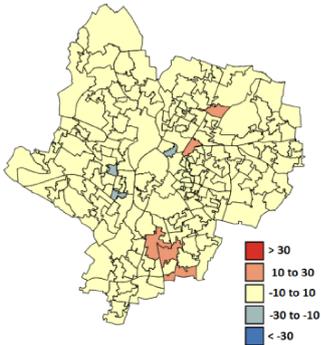


Local Moran's I: Children's Admission Rate
R² = 0.53

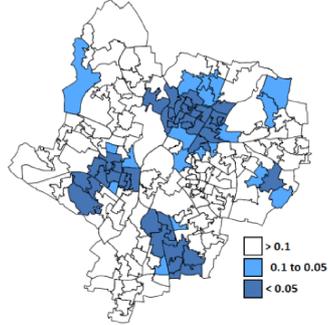


GWR 40NN: Hospitalisation Rates Associated With Levels Of Afro-Caribbean Children

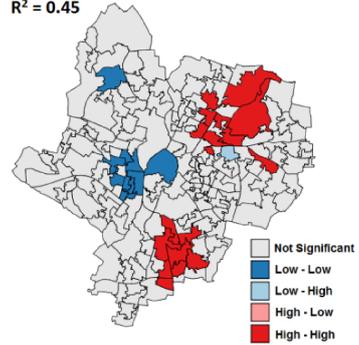
Admissions Per 1,000 Children



GWR Coefficient P-Value



Local Moran's I: Children's Admission Rate
R² = 0.45

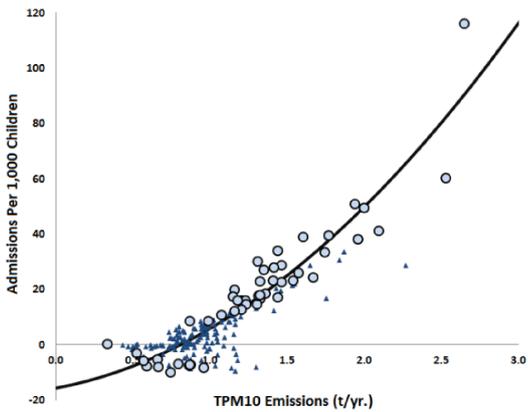


APPENDIX C15: GWR modelled children's J00-99 hospital admission rates associated with the social lifestyle of 'Afro-Caribbean' residents

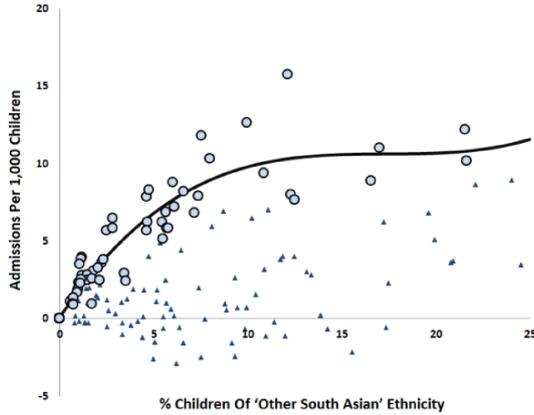
Independent Variable	GWR Model	Observations (P<0.05)	Optimum Model (P<0.05)	R ²	Constant (B0)	B1	B2	B3
TPM ₁₀ Emissions (t/y)	80NN	46	Quadratic	0.92	3.043	-2.339	11.808	
	60NN	19	Quadratic	0.96	-5.593	-2.738	17.620	
	40NN	14	Quadratic	0.99	9.650	-55.783	41.159	
Carstairs Index (Rank for Leicester)	80NN	42	Cubic	0.80	26.437	4.097	-0.068	-0.024
	60NN	41	Cubic	0.63	29.759	4.462	-0.357	-0.045
	40NN	37	Cubic	0.32	35.682	4.783	-1.517	-0.264
(%) Smoking Prevalence	80NN	7	Cubic	0.99	-24.857	2.256	0.000	-0.002
	60NN	20	Cubic	0.98	-8.971	0.895	-0.003	0.000
	40NN	9	Cubic	0.99	-29.216	2.385	0.000	-0.001
(%) Obesity Prevalence	80NN	16	N/A					
	60NN	8	N/A					
	40NN	3	N/A					
(%) White Non-British Children	80NN	0	N/A					
	60NN	0	N/A					
	40NN	0	N/A					
(%) Indian Children	80NN	64	Cubic	0.82	-0.425	-0.229	-0.009	1E-04
	60NN	34	Cubic	0.90	-0.041	-0.459	-0.004	-9E-05
	40NN	27	Cubic	0.91	-0.347	-0.580	-1E-04	-5E-05
(%) Other South Asian Children	80NN	53	Cubic	0.97	-0.096	2.628	-0.377	0.022
	60NN	49	Cubic	0.90	0.072	2.213	-0.151	0.003
	40NN	30	Cubic	0.98	0.319	1.979	-0.080	0.001
(%) Afro-Caribbean Children	80NN	33	Cubic	0.94	0.078	1.439	-0.001	-2E-04
	60NN	27	Cubic	0.96	-0.063	2.167	-0.443	0.075
	40NN	18	N/A					

APPENDIX C16: Stimulus-response models describing the relationship between socio-environmental variables and their specific GWR modelled hospital admissions rates per 1,000 children (If coefficient P≤0.01)

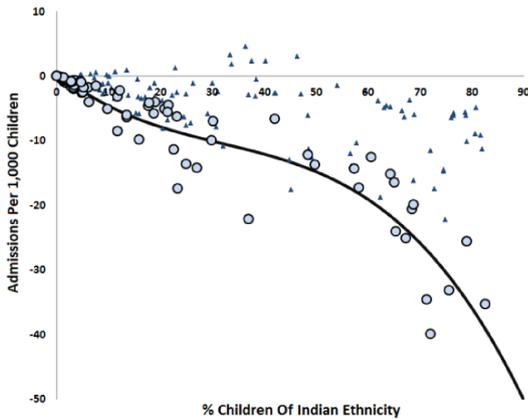
GWR 60NN: J00-99 Hospitalisation Rates Associated With TPM₁₀ Emissions (P≤0.05)



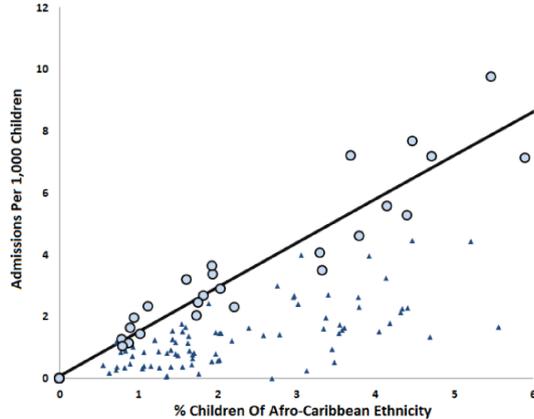
GWR 60NN: J00-99 Hospitalisation Rates Associated With TPM₁₀ Emissions (P≤0.05)



GWR 60NN: J00-99 Hospitalisation Rates Associated With Indian Residency (P≤0.05)



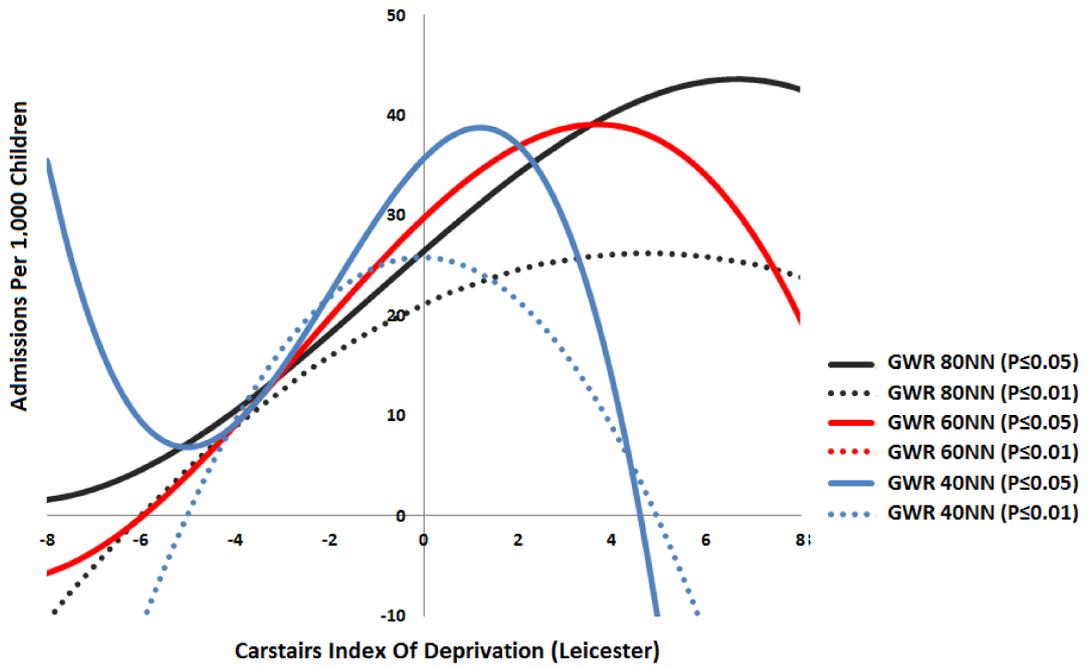
GWR 80NN: J00-99 Hospitalisation Rates Associated With Afro-Caribbean Residency (P≤0.01)



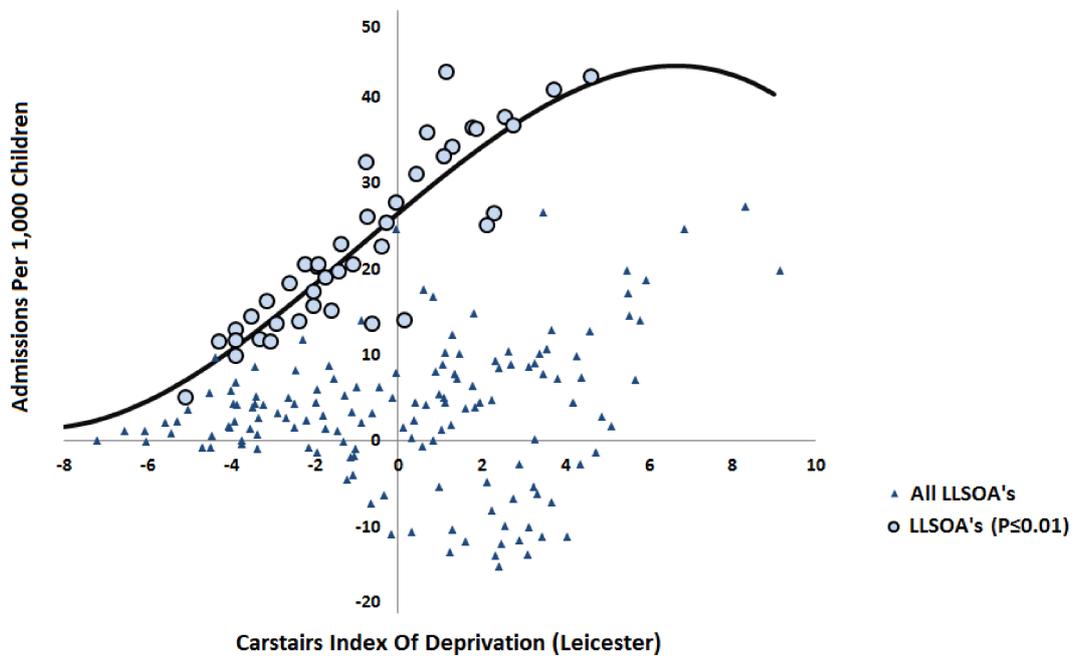
▲ All LLSOA's
● LLSOA's (P≤0.05 or P≤0.01)

APPENDIX C17: Best fitted universal J00-99 stimulus-responses associated with residentially experienced TPM₁₀ emissions, and residential levels of 'Other South Asian', 'Indian' and 'Afro-Caribbean' ethnicities.

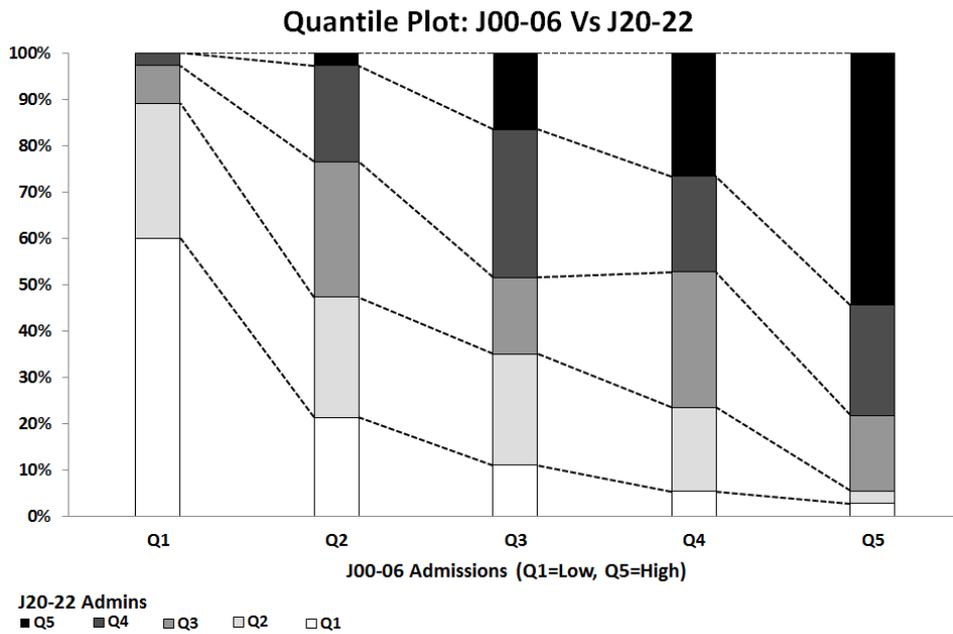
J00-99 Hospitalisation Rates Associated With Carstairs Index Rank ($P \leq 0.05$, $P \leq 0.01$)



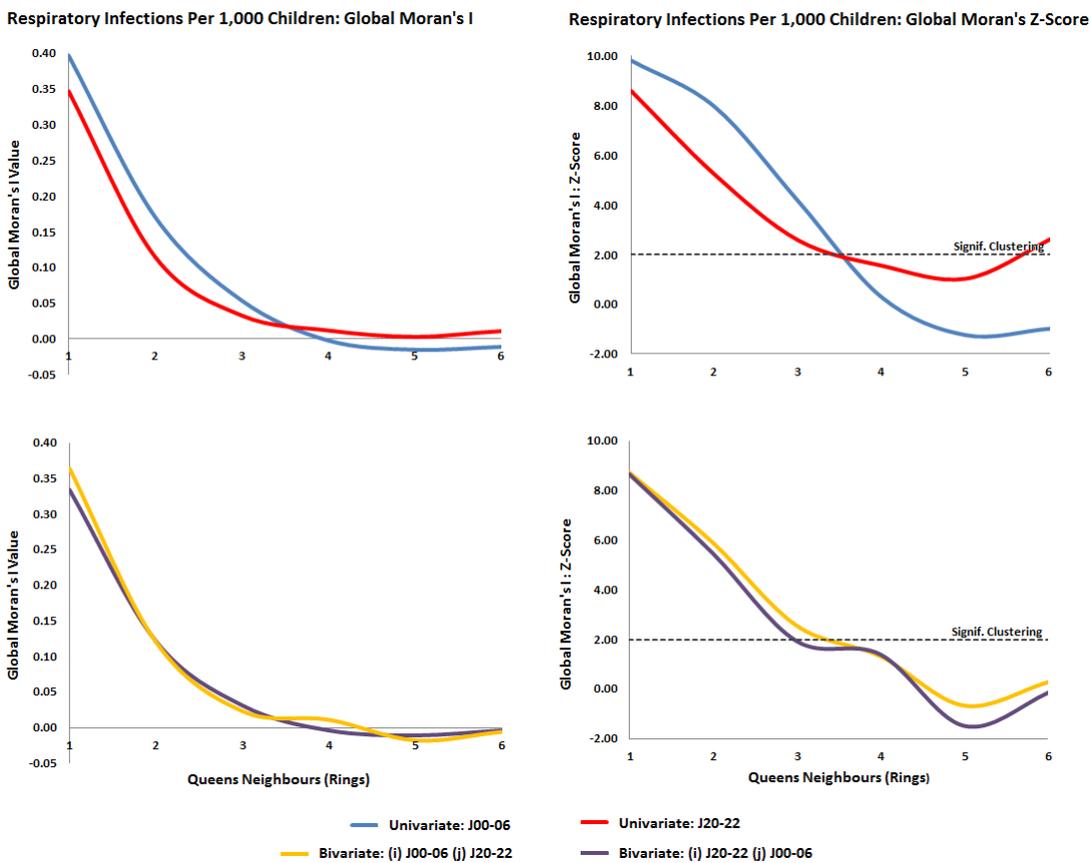
GWR 80NN: J00-99 Hospitalisation Rates Associated With Carstairs Index Rank ($P \leq 0.01$)



APPENDIX C18: Universal J00-99 stimulus-responses associated with Leicester UA's Carstairs Index of deprivation, created from local GWR model outputs of significant ($P \leq 0.05$; $P \leq 0.01$)



APPENDIX D1: Quantile Plot of annual LLSOA levels of children’s J00-06 admission rates Vs. J20-22 admission rates, across Leicester UA: 2000-09



APPENDIX D2: Spatial correlograms of Global Moran's statistical outputs, portraying the level of decay in autocorrelation between neighbouring LLSOA community’s URT and LRT health, as a function of distance.

Local Classification: J00-06 Moran's I	Adult Lifestyle Choices (%)		Socio-Environmental Issues		Ethnic Composition (%)					Admissions Per 1,000 Children	
	Smoking Prevalence	Obesity Prevalence	Carstairs Index	TPM ₁₀ (t/yr.)	White British	White Non-British	Indian	Other South Asian	Afro-Caribbean	J00-06	J20-22
Hot-Spot (H-H)	33.11	20.67	4.11	2.19	34.38	2.08	26.96	11.58	12.41	42.43	17.60
Cold-Spot (L-L)	18.35	24.07	-0.36	0.95	29.92	1.55	54.76	5.58	0.99	12.11	4.72
Not Signif. (P >0.05)	27.49	24.47	-0.15	1.00	56.19	1.54	27.03	5.51	2.55	16.09	6.37

APPENDIX D3: Summary of mean community characteristics for areas classified by the J00-06 Local Moran's I statistic

Local Classification: J20-22 Moran's I	Adult Lifestyle Choices (%)		Socio-Environmental Issues		Ethnic Composition (%)					Admissions Per 1,000 Children	
	Smoking Prevalence	Obesity Prevalence	Carstairs Index	TPM ₁₀ (t/yr.)	White British	White Non-British	Indian	Other South Asian	Afro-Caribbean	J00-06	J20-22
Hot-Spot (H-H)	32.00	21.65	4.80	2.25	32.29	1.90	29.18	10.04	12.91	43.13	18.25
Cold-Spot (L-L)	14.29	21.54	-0.96	0.99	25.14	1.24	60.77	6.37	0.95	10.58	3.75
High Outlier (H-L)	12.80	20.60	-2.20	0.82	40.98	2.57	41.52	4.39	1.07	13.11	7.46
Not Signif. (P >0.05)	27.72	24.57	-0.09	1.00	55.83	1.54	27.26	5.59	2.58	16.22	6.39

APPENDIX D4: Summary of mean community characteristics for areas classified by the J20-22 Local Moran's I statistic

Local Classification: Bivariate Moran's I (i) J00-06; (j) J20-22	Adult Lifestyle Choices (%)		Socio-Environmental Issues		Ethnic Composition (%)					Admissions Per 1,000 Children	
	Smoking Prevalence	Obesity Prevalence	Carstairs Index	TPM ₁₀ (t/yr.)	White British	White Non-British	Indian	Other South Asian	Afro-Caribbean	J00-06	J20-22
Hot-Spot (H-H)	32.00	21.65	4.80	2.25	32.29	1.90	29.18	10.04	12.91	43.13	18.25
Cold-Spot (L-L)	13.84	21.26	-1.33	0.94	29.89	1.64	55.00	5.78	0.98	11.34	4.86
Not Signif. (P >0.05)	27.72	24.57	-0.09	1.00	55.83	1.54	27.26	5.59	2.58	16.22	6.39

APPENDIX D5: Summary of mean community characteristics for areas classified by the Bivariate Local Moran's I statistic, where (i) = J00-06 and (j) = J20-22

Local Classification: Bivariate Moran's I (i) J20-22; (j) J00-06	Adult Lifestyle Choices (%)		Socio-Environmental Issues		Ethnic Composition (%)					Admissions Per 1,000 Children	
	Smoking Prevalence	Obesity Prevalence	Carstairs Index	TPM ₁₀ (t/yr.)	White British	White Non-British	Indian	Other South Asian	Afro-Caribbean	J00-06	J20-22
Hot-Spot (H-H)	33.11	20.67	4.11	2.19	34.38	2.08	26.95	11.58	12.41	42.43	17.60
Cold-Spot (L-L)	16.00	24.75	-0.14	0.98	16.90	0.93	68.32	6.37	0.86	29.88	11.07
High Outlier (H-L)	23.05	22.70	-0.79	0.90	55.96	2.78	27.63	4.01	1.27	14.19	8.00
Not Signif. (P >0.05)	27.49	24.47	-0.15	1.00	56.19	1.54	27.03	5.51	2.55	16.09	6.37

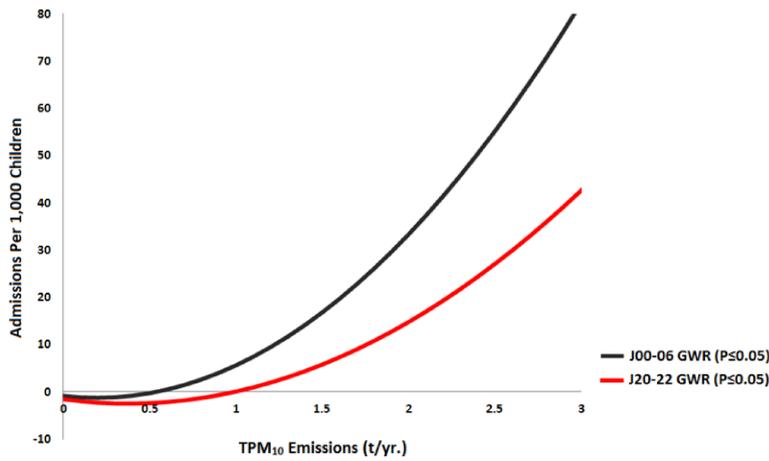
APPENDIX D6: Summary of mean community characteristics for areas classified by the Bivariate Local Moran's I statistic, where (i) = J20-22 and (j) = J00-06

	Goodness-Of-Fit Measures			Relative Goodness-Of-Fit: Accuracy Vs. Complexity		Global Moran's I: Residual Patterning		F-Test: Relative Improvement GWR Vs. OLS		
	R ²	CV-R ²	RSS	AIC	AICc	Moran's I	Z-Score	FBC-F	LMZ-F1	LMZ-F2
<i>J00-06 Admissions:</i>										
OLS	0.37	0.34	9302.33	1279.28	1282.53	0.13	3.29			
GWR 80 NN	0.59	0.48	6058.16	1219.49	1281.64	-0.03	-0.64	1.54*	0.84	1.54*
GWR 70 NN	0.63	0.50	5485.24	1206.37	1281.41	-0.05	-1.12	1.70*	0.80	1.55*
GWR 60 NN	0.68	0.53	4812.37	1189.37	1284.49	-0.09	-2.02*	1.93*	0.76*	1.53*
GWR 50 NN	0.73	0.56	3980.79	1164.01	1291.61	-0.13	-2.95*	2.34*	0.69*	1.50*
GWR 40 NN	0.78	0.58	3206.82	1137.36	1321.89	-0.15	-3.57*	2.90*	0.65*	1.39*
GWR 30 NN	0.85	0.61	2173.13	1085.71	1400.06	-0.19	-4.31*	4.28*	0.59*	1.28
GWR 20 NN	0.93	0.63	980.24	970.24	1722.77	-0.19	-4.51*	9.48*	0.49*	1.14
<i>J20-22 Admissions:</i>										
OLS	0.42	0.27	1605.69	950.77	954.02	0.05	1.33			
GWR 80 NN	0.61	0.43	1074.96	896.15	958.30	-0.04	-0.84	1.49*	0.87	1.46*
GWR 70 NN	0.64	0.45	992.82	886.74	961.78	-0.06	-1.14	1.62*	0.84	1.44*
GWR 60 NN	0.68	0.48	889.43	873.65	968.77	-0.08	-1.65	1.81*	0.81	1.41*
GWR 50 NN	0.72	0.50	766.12	855.85	983.46	-0.10	-2.05*	2.10*	0.77*	1.37*
GWR 40 NN	0.78	0.51	606.65	825.99	1010.52	-0.11	-2.42*	1.85*	0.71*	1.32*
GWR 30 NN	0.85	0.51	417.28	777.13	1091.48	-0.15	-3.16*	3.85*	0.65*	1.23
GWR 20 NN	0.92	0.50	223.18	693.38	1445.92	-0.18	-3.92*	7.19*	0.65*	1.10

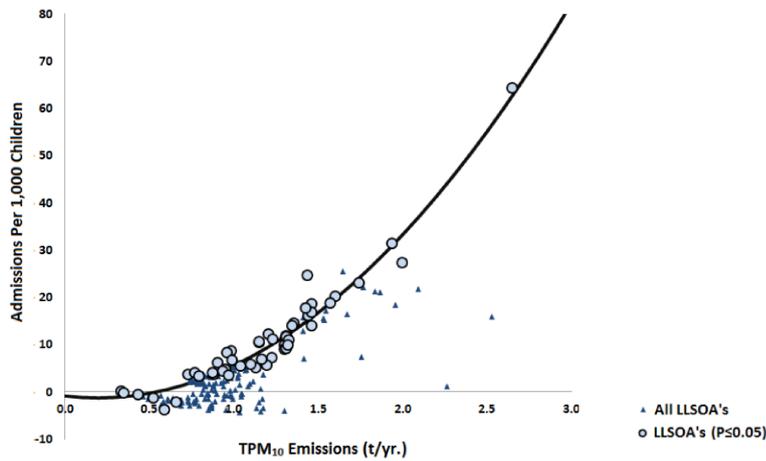
Footnote: Moran's I: * P ≤ 0.05 (Significant Dispersion); (b) GWR F-Tests: * P ≤ 0.05

APPENDIX D7: Diagnostics of the J00-06 and J20-22 respiratory subset GWR models containing a bi-square adaptive weighting scheme

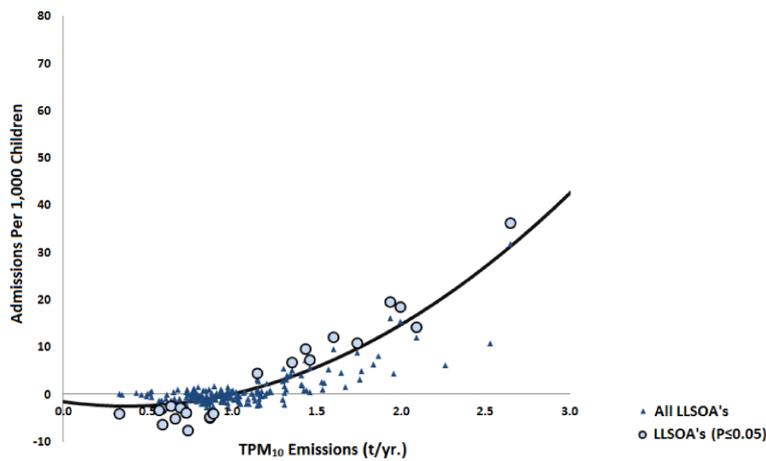
Hospitalisation Rates Associated With TPM₁₀ Emissions (P≤0.05)



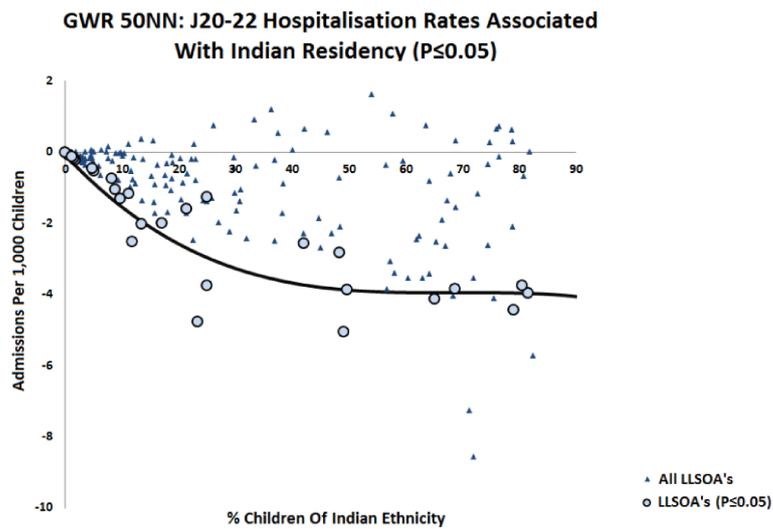
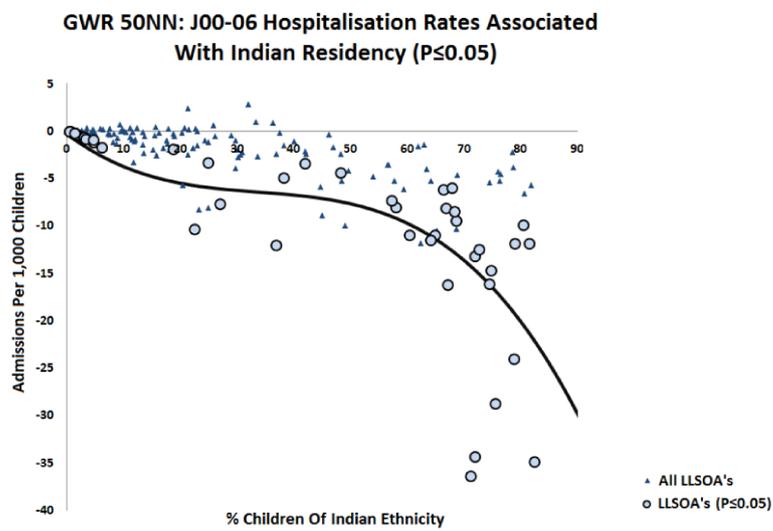
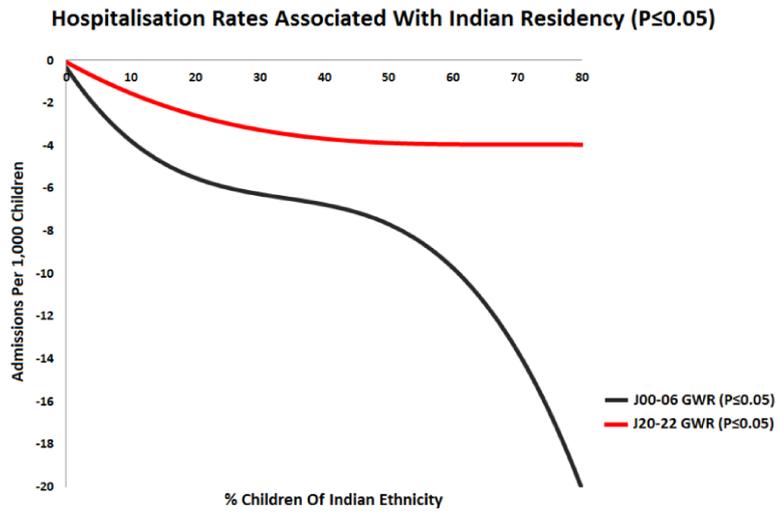
GWR 50NN: J00-06 Hospitalisation Rates Associated With TPM₁₀ Emissions (P≤0.05)



GWR 50NN: J20-22 Hospitalisation Rates Associated With TPM₁₀ Emissions (P≤0.05)

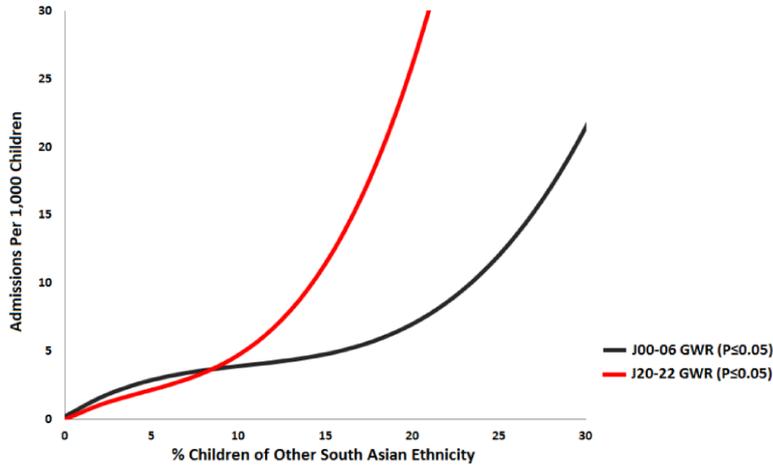


APPENDIX D8: Universal J00-06 and J20-22 stimulus-responses associate to residentially experienced TPM₁₀ emissions, created from local GWR model outputs (P≤0.05)

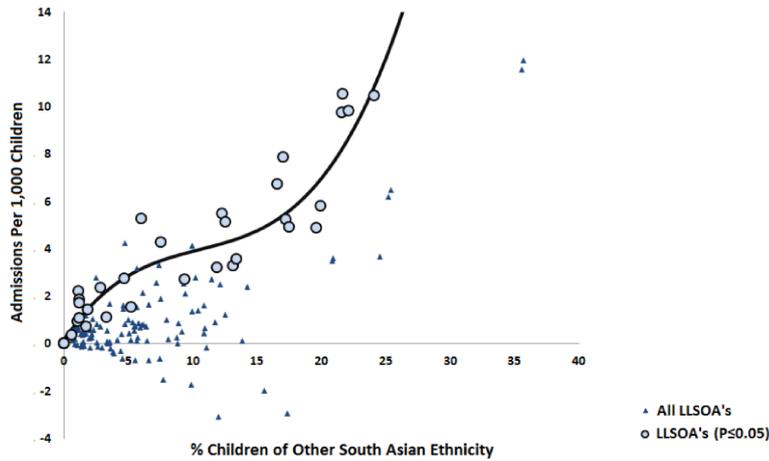


APPENDIX D9: Universal J00-06 and J20-22 stimulus-responses associated to levels of 'Indian' residency, created from local GWR model outputs ($P \leq 0.05$)

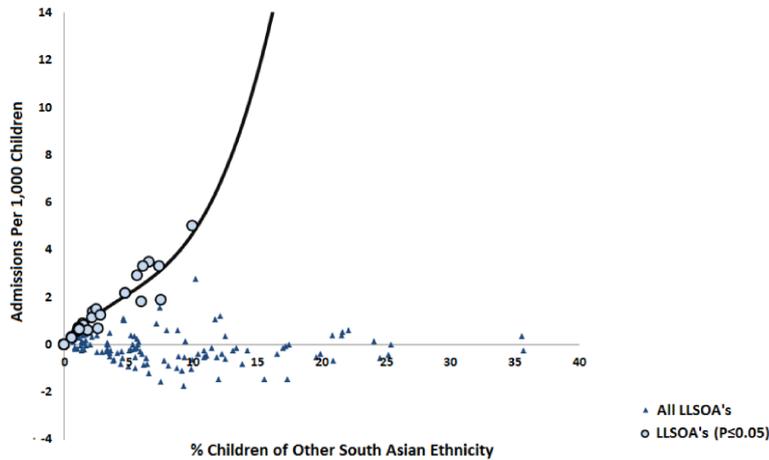
Hospitalisation Rates Associated With Other S.Asian Residency ($P \leq 0.05$)



GWR 50NN: J00-06 Hospitalisation Rates Associated With Other S.Asian Residency ($P \leq 0.05$)

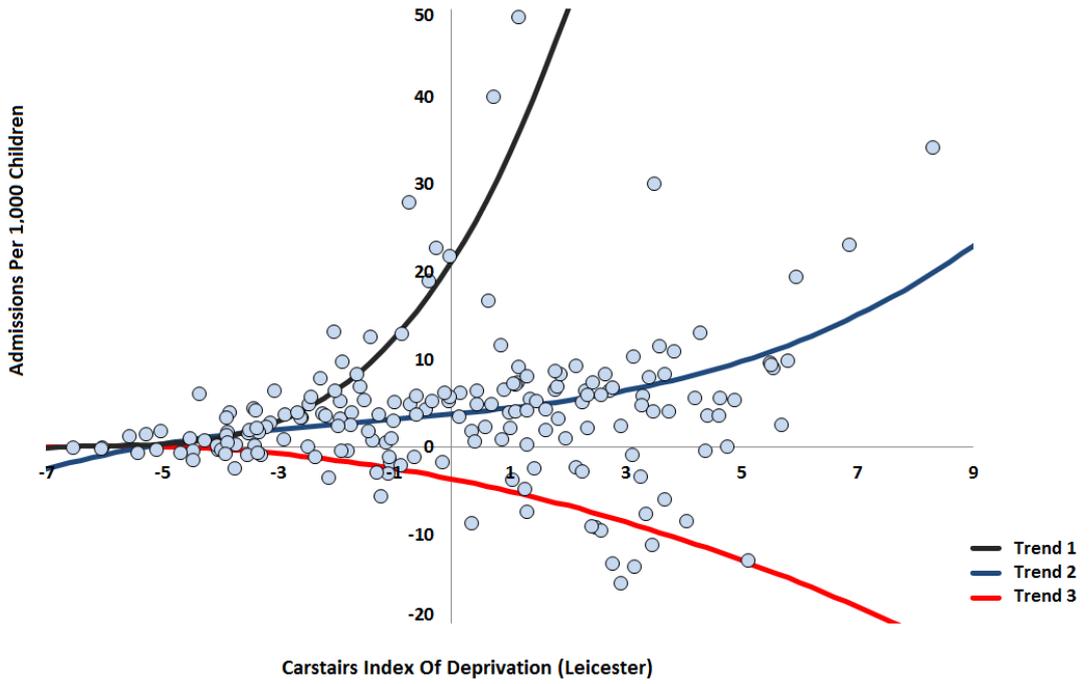


GWR 50NN: J20-22 Hospitalisation Rates Associated With Other S.Asian Residency ($P \leq 0.05$)



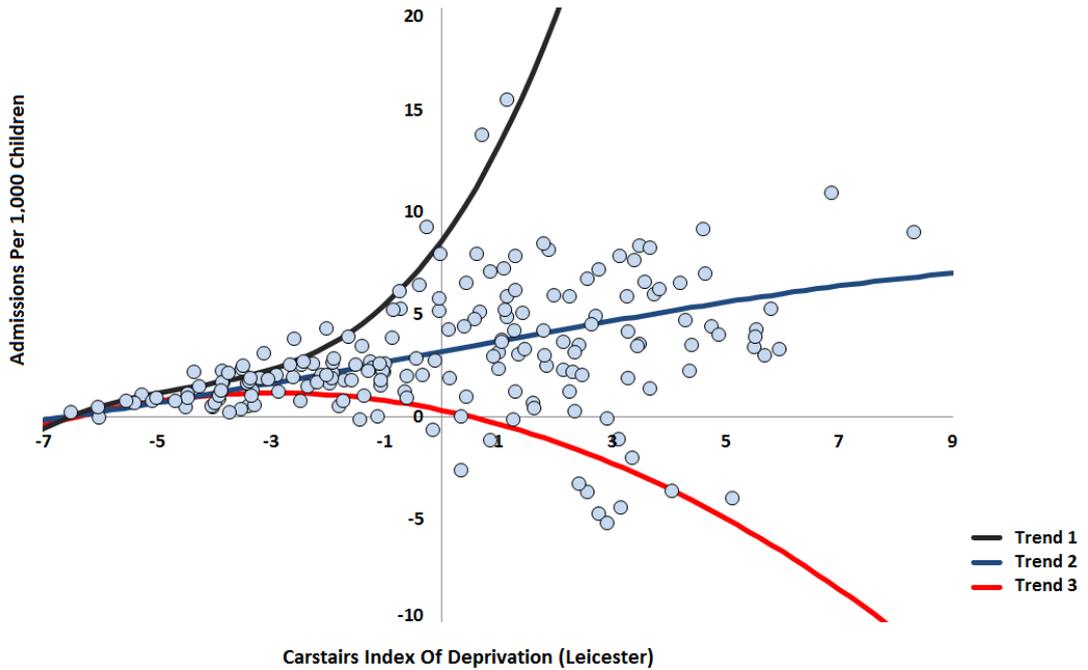
APPENDIX D10: Universal J00-06 and J20-22 stimulus-responses associated to levels of 'Other South Asian' residency, created from local GWR model outputs ($P \leq 0.05$)

GWR 50NN: J00-06 Hospitalisation Rates Associated With Carstairs Index Rank (3-Trends: All LLSOA's)



APPENDIX D11: Universal 3-Trend J00-06 stimulus-responses associated with Leicester UA's Carstairs Index of deprivation, created from all 187 local GWR model outputs

GWR 50NN: J20-22 Hospitalisation Rates Associated With Carstairs Index Rank (3-Trends: All LLSOA's)



APPENDIX D12: Universal 3-Trend J20-22 stimulus-responses associated with Leicester UA's Carstairs Index of deprivation, created from all 187 local GWR model outputs

- APPENDIX D -

Carstairs Index (Leicester)	LLSOA's	Optimum Model (P<0.05)	R ²	Constant (B0)	B1	B2	B3
Trend 1 (Upper)	66	Cubic	0.77	21.224	10.479	1.762	0.100
Trend 2 (Middle)	141	Cubic	0.48	3.823	0.617	0.051	0.013
Trend 3 (Lower)	58	Quadratic	0.72	-3.589	-1.297	-0.112	

APPENDIX D13: Universal 3-Trend J00-06 stimulus-responses associated with Leicester UA's Carstairs Index of deprivation, created from all 187 local GWR model outputs

Carstairs Index (Leicester)	LLSOA's	Optimum Model (P<0.05)	R ²	Constant (B0)	B1	B2	B3
Trend 1 (Upper)	36	Cubic	0.94	8.615	3.801	0.727	0.053
Trend 2 (Middle)	132	Cubic	0.58	3.181	0.517	-0.001	-0.001
Trend 3 (Lower)	68	Quadratic	0.62	0.334	-0.573	-0.097	

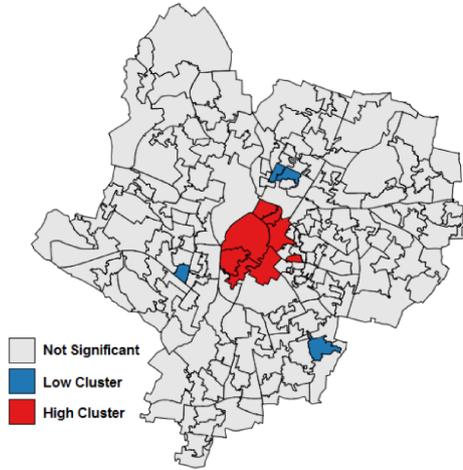
APPENDIX D14: Universal 3-Trend J20-22 stimulus-responses associated with Leicester UA's Carstairs Index of deprivation, created from all 187 local GWR model outputs

	Total	Hot Season (%) MAY - OCT	Cold Season (%) NOV - APR
J00-06: Acute Upper Respiratory Infections (URI)	10228	43.4	56.6
▪ J00: Acute Nasopharyngitis [Common Cold]	263	39.9	60.1
▪ J01: Acute Sinusitis	30	60.0	40.0
▪ J02: Acute Pharyngitis	316	48.4	51.6
▪ J03: Acute Tonsillitis	3081	47.9	52.1
▪ J04: Acute Laryngitis [Croup] & Tracheitis	22	27.3	72.7
▪ J05: Acute Obstructive Laryngitis/Epiglottitis	798	45.4	54.6
▪ J06: Acute URI - Multiple/Unspecified Sites	5882	40.7	59.3
J20-22: Other Acute Lower Respiratory Infections (LRI)	4142	23.0	77.0
▪ J20: Acute Bronchitis	26	23.1	76.9
▪ J21: Acute Bronchiolitis	2548	15.1	84.9
▪ J22: Unspecified Acute LRI	1581	35.7	64.3

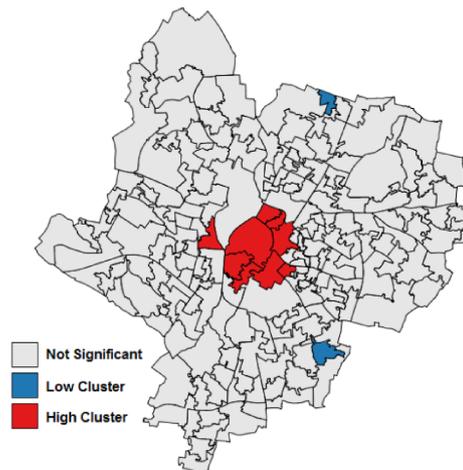
APPENDIX D15: Leicester UA Admission Count, including seasonal decomposition: 2000-09

APPENDIX E

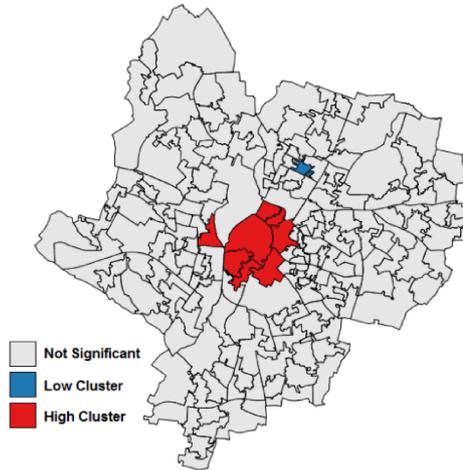
J00-99 Admissions Per 1,000 Children



J00-06 Admissions Per 1,000 Children

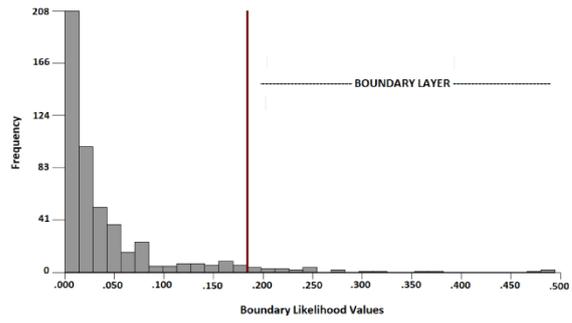
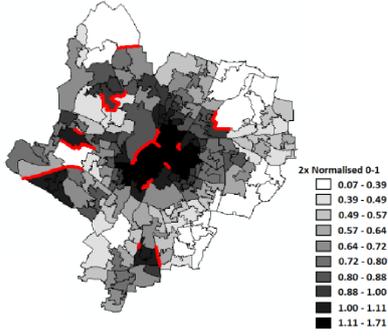


J20-22 Admissions Per 1,000 Children

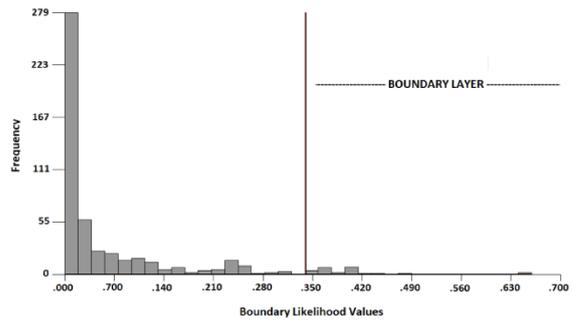
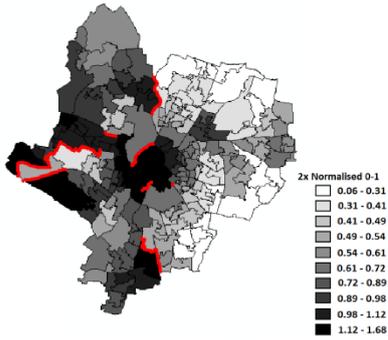


APPENDIX E1: Getis-Ord G_i^* hot-spot analysis ($P \leq 0.05$) of annual average children's admissions of the complete respiratory set (J00-99), in addition to rates relating to the URTI (J00-06) and LRTI (J20-22) subsets

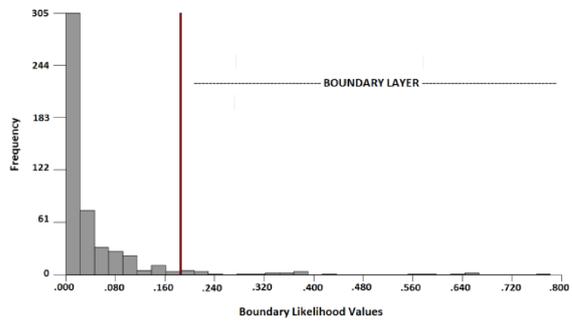
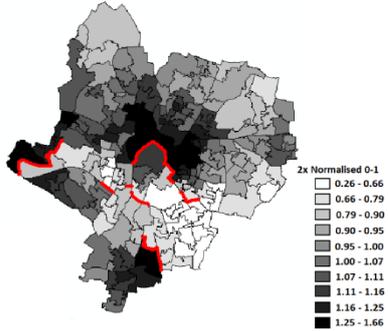
TPM₁₀ & Carstairs Index [Leicester]



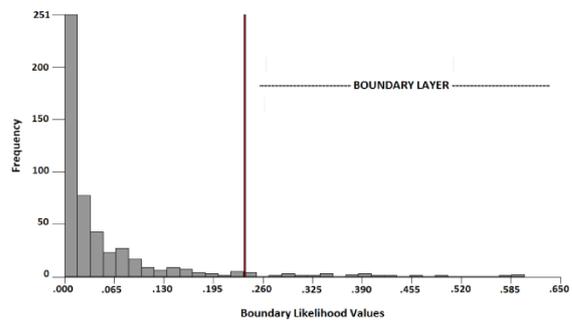
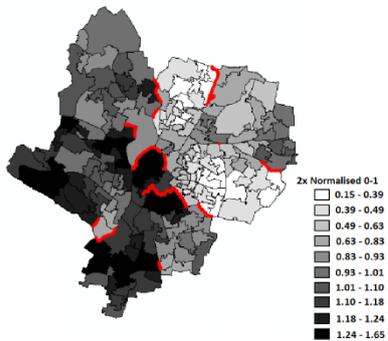
TPM₁₀ & Adult Smoking Prevalence



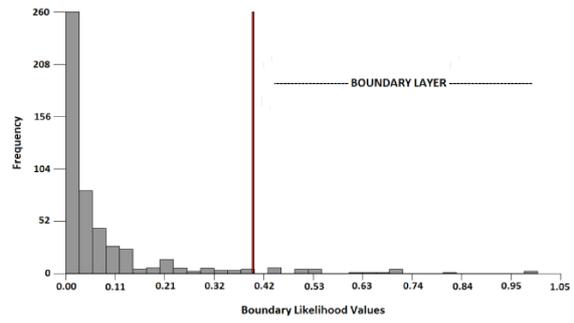
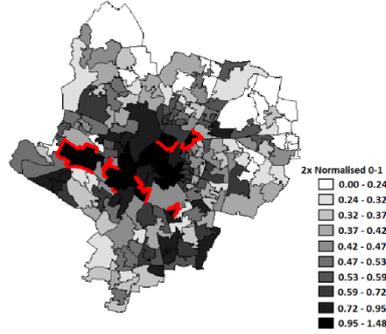
TPM₁₀ & Adult Obesity Prevalence



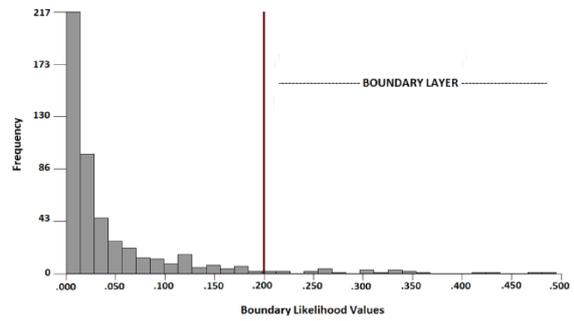
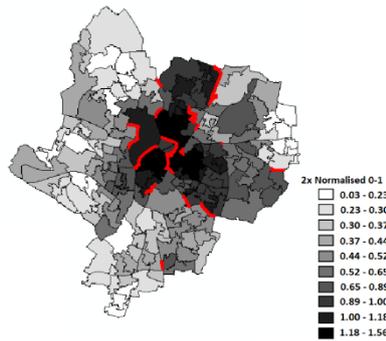
TPM₁₀ & White British Children



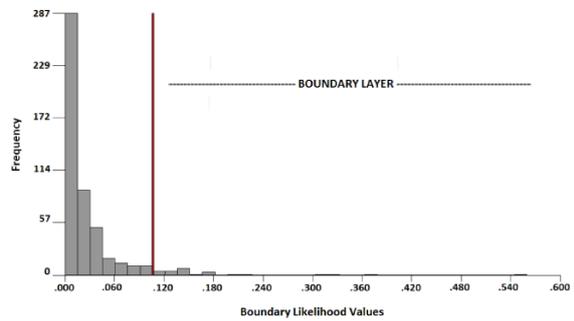
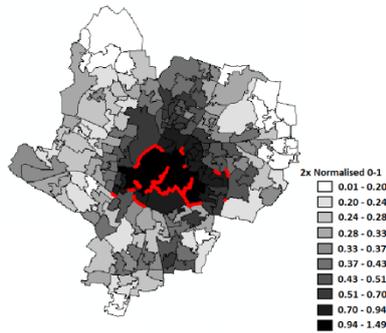
TPM₁₀ & White Non-British Children



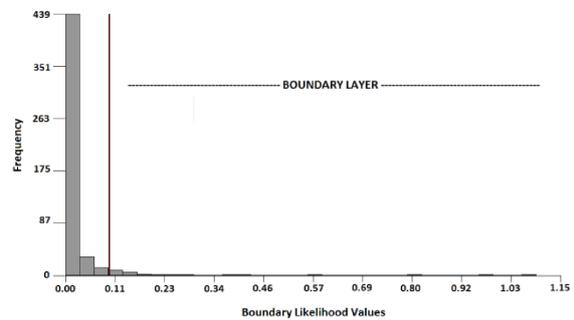
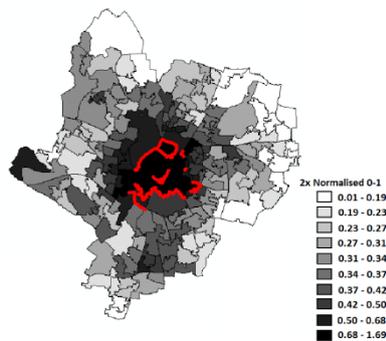
TPM₁₀ & Indian Children



TPM₁₀ & Other South Asian Children

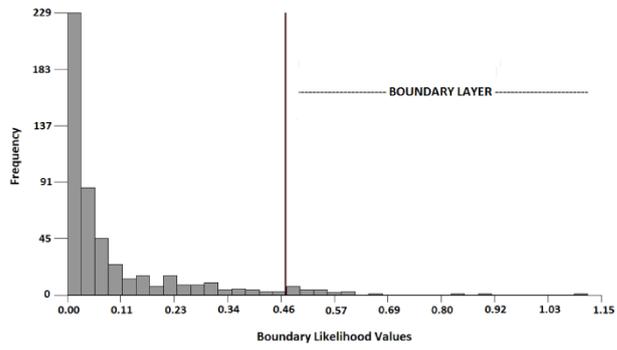
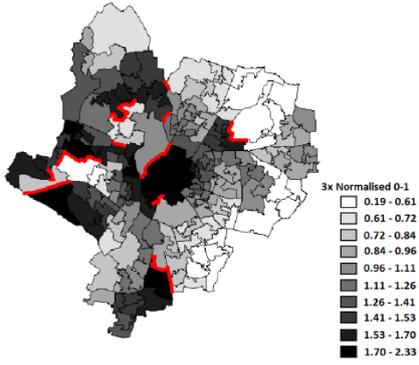


TPM₁₀ & Afro-Caribbean Children

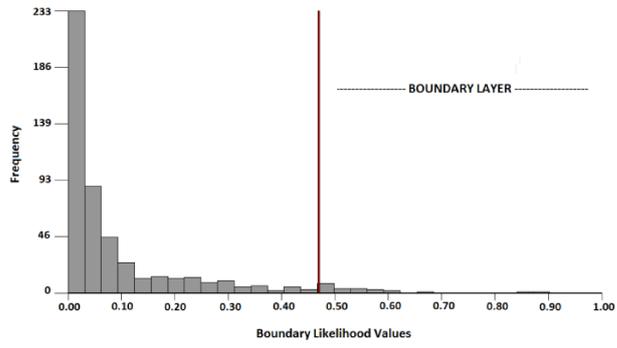
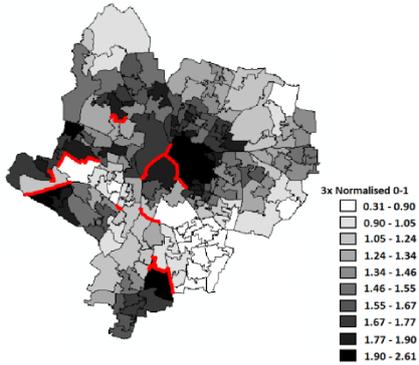


APPENDIX E2: [PAGES 404-505]: Maps illustrating the top 5% of bivariate Boundary Elements (red) across respective decile distributions describing normalised TPM₁₀ emissions (0-1) combined with a normalised social parameter (0-1).

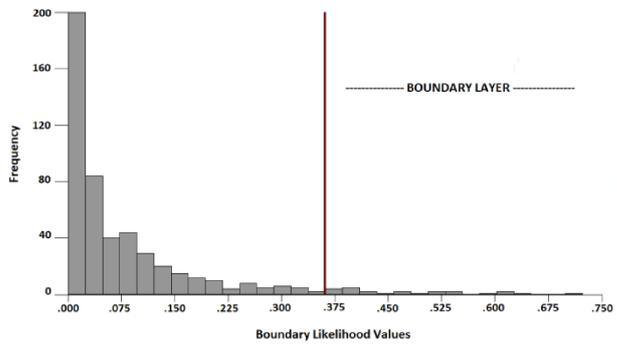
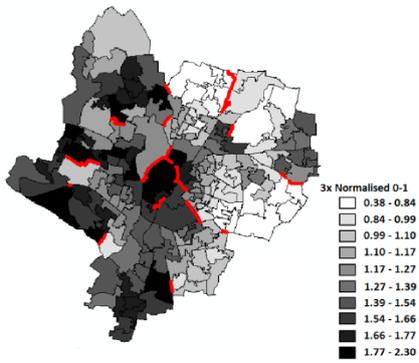
TPM₁₀, Carstairs Index & Adult Smoking Prevalence



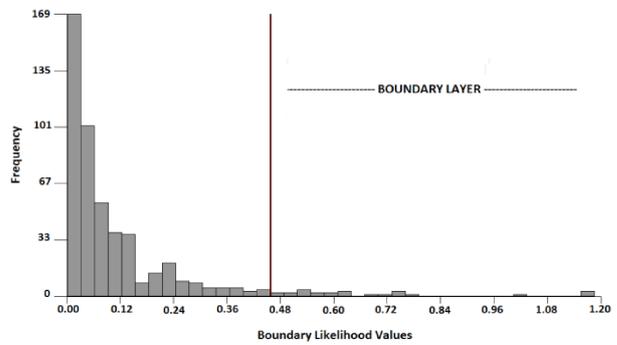
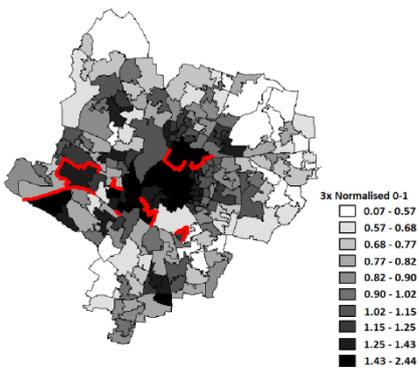
TPM₁₀, Carstairs Index & Obesity Prevalence



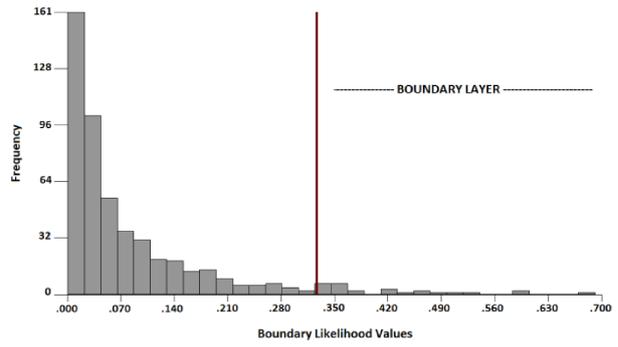
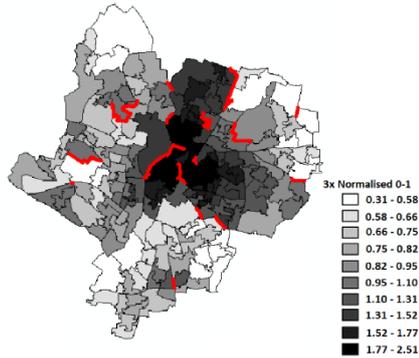
TPM₁₀, Carstairs Index & White British Children



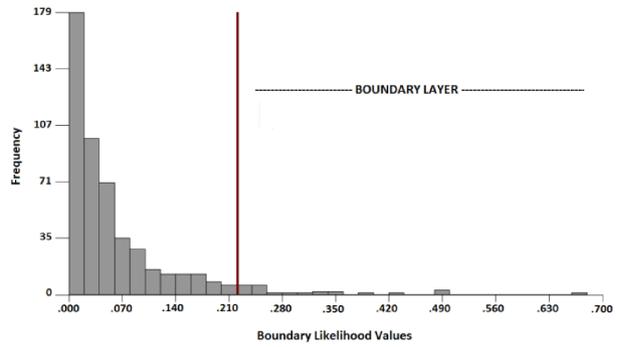
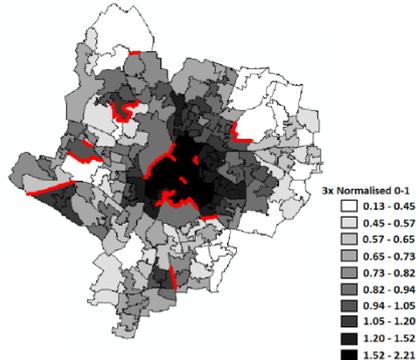
TPM₁₀, Carstairs Index & White Non-British Children



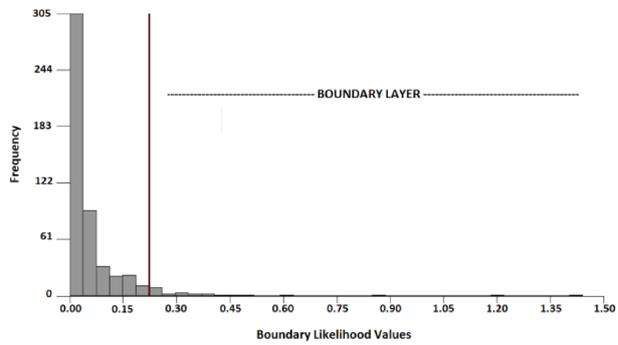
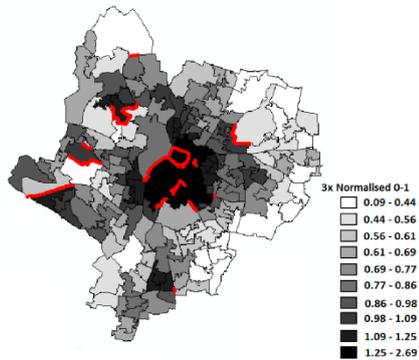
TPM₁₀, Carstairs Index & Indian Children



TPM₁₀, Carstairs Index & Other South Asian Children



TPM₁₀, Carstairs Index & Afro-Caribbean Children



APPENDIX E3 [PAGES 406-407]: Maps illustrating the top 5% of Trivariate Boundary Elements (red) across respective decile distributions describing normalised TPM₁₀ emissions (0-1) and Carstairs Index measures of deprivation (0-1), combined with a normalised social parameter (0-1).

- APPENDIX E -

Boundaries Overlapped (G,H) H= J00-06 Admissions Per 1,000 Children	Statistic	Observed (meters)	Expected (meters)	P↑	P↓
(G) TPM₁₀	O_G	257	1009 (±321)	1.00	0.00*
	O_H	372	902 (±330)	0.97	0.03*
	O_{GH}	315	956 (±269)	1.00	0.00*
	O_S (count)	14	3 (±2)	0.00*	1.00
(G) Carstairs Index of Deprivation	O_G	1063	987 (±288)	0.35	0.65
	O_H	409	699 (±271)	0.88	0.12
	O_{GH}	736	843 (±227)	0.65	0.35
	O_S (count)	8	3 (±2)	0.04*	0.98
(G) Smoking Prevalence	O_G	1019	993 (±285)	0.40	0.60
	O_H	728	693 (±270)	0.37	0.63
	O_{GH}	876	845 (±223)	0.41	0.59
	O_S (count)	5	3 (±2)	0.29	0.83
(G) Obesity Prevalence	O_G	859	1023 (±307)	0.67	0.33
	O_H	552	730 (±282)	0.72	0.28
	O_{GH}	712	878 (±244)	0.74	0.26
	O_S (count)	8	3 (±2)	0.05*	0.98
(G) White British Children	O_G	1370	1004 (±271)	0.10	0.90
	O_H	559	635 (±237)	0.59	0.41
	O_{GH}	964	819 (±210)	0.23	0.77
	O_S (count)	4	3 (±2)	0.43	0.75
(G) White Non-British Children	O_G	313	1003 (±332)	1.00	0.00*
	O_H	433	836 (±302)	0.93	0.07
	O_{GH}	373	920 (±265)	1.00	0.00*
	O_S (count)	13	3 (±2)	0.00*	1.00
(G) Indian Children	O_G	964	1007 (±290)	0.52	0.48
	O_H	504	699 (±242)	0.79	0.21
	O_{GH}	734	853 (±221)	0.68	0.32
	O_S (count)	6	3 (±2)	0.15	0.92
(G) Other South Asian Children	O_G	707	899 (±306)	0.71	0.29
	O_H	877	970 (±346)	0.55	0.45
	O_{GH}	792	934 (±265)	0.70	0.30
	O_S (count)	5	4 (±3)	0.35	0.77
(G) Afro-Caribbean Children	O_G	245	1019 (±376)	1.00	0.00*
	O_H	537	1062 (±396)	0.95	0.05*
	O_{GH}	391	1040 (±323)	0.99	0.01*
	O_S (count)	17	3 (±3)	0.00*	1.00

* P≤0.05

APPENDIX E4: Univariate boundary overlap analysis with annual average children's admissions of the URT (J00-06)

- APPENDIX E -

Boundaries Overlapped (G,H) H= J00-06 Admissions Per 1,000 Children	Statistic	Observed (meters)	Expected (meters)	P↑	P↓
(G) TPM₁₀ & Carstairs Index	O _G	834	1001 (±293)	0.68	0.32
	O _H	78	825 (±279)	1.00	0.00*
	O _{GH}	456	913 (±229)	0.99	0.01*
	O _S (count)	11	3 (±2)	0.00*	1.00
(G) TPM₁₀ & Smoking prevalence	O _G	1015	1039 (±326)	0.47	0.53
	O _H	458	774 (±281)	0.89	0.11
	O _{GH}	736	907 (±246)	0.75	0.25
	O _S (count)	6	3 (±2)	0.16	0.91
(G) TPM₁₀ & Obesity prevalence	O _G	855	1032 (±308)	0.71	0.29
	O _H	544	745 (±272)	0.76	0.24
	O _{GH}	700	888 (±239)	0.79	0.21
	O _S (count)	7	3 (±2)	0.08	0.96
(G) TPM₁₀ & White British Children	O _G	1245	1033 (±308)	0.20	0.80
	O _H	459	661 (±259)	0.79	0.21
	O _{GH}	852	847 (±237)	0.45	0.55
	O _S (count)	6	3 (±2)	0.13	0.93
(G) TPM₁₀ & White Non-British Children	O _G	313	1002 (±311)	1.00	0.00*
	O _H	433	825 (±327)	0.92	0.08
	O _{GH}	373	914 (±259)	0.99	0.01*
	O _S (count)	13	3 (±2)	0.00*	1.00
(G) TPM₁₀ & Indian Children	O _G	863	1023 (±290)	0.69	0.31
	O _H	304	706 (±263)	0.97	0.03*
	O _{GH}	584	864 (±229)	0.91	0.09
	O _S (count)	8	3 (±2)	0.04*	0.99
(G) TPM₁₀ & Other South Asian Children	O _G	448	1036 (±367)	0.99	0.01*
	O _H	562	955 (±355)	0.90	0.10
	O _{GH}	505	996 (±293)	0.98	0.02*
	O _S (count)	10	3 (±2)	0.01*	1.00
(G) TPM₁₀ & Afro-Caribbean Children	O _G	209	1005 (±377)	1.00	0.00*
	O _H	509	79 (±395)	0.96	0.04*
	O _{GH}	359	1042 (±318)	1.00	0.00*
	O _S (count)	15	3 (±3)	0.00*	1.00

* P≤0.05

APPENDIX E5: Bivariate boundary overlap analysis of TPM₁₀ emissions and an individual social parameter, with annual average children's admissions of the URT (J00-06)

- APPENDIX E -

Boundaries Overlapped (G,H) H= J00-06 Admissions Per 1,000 Children	Statistic	Observed (meters)	Expected (meters)	P↑	P↓
(G) TPM₁₀, Carstairs Index & Smoking Prevalence	O _G	1110	1028 (±309)	0.35	0.65
	O _H	396	722 (±294)	0.91	0.09
	O _{GH}	753	875 (±251)	0.66	0.34
	O _{S (count)}	6	3 (±2)	0.18	0.91
(G) TPM₁₀, Carstairs Index & Obesity Prevalence	O _G	965	987 (±280)	0.47	0.53
	O _H	435	699 (±248)	0.85	0.15
	O _{GH}	700	843 (±216)	0.76	0.24
	O _{S (count)}	9	3 (±2)	0.02*	0.99
(G) TPM₁₀, Carstairs Index & White British Children	O _G	1066	994 (±281)	0.35	0.65
	O _H	277	666 (±248)	0.98	0.02*
	O _{GH}	671	830 (±215)	0.76	0.24
	O _{S (count)}	9	3 (±2)	0.01*	0.99
(G) TPM₁₀, Carstairs Index & White Non-British Children	O _G	316	1017 (±322)	1.00	0.00*
	O _H	410	808 (±286)	0.97	0.03*
	O _{GH}	363	912 (±255)	1.00	0.00*
	O _{S (count)}	13	3 (±2)	0.00*	1.00
(G) TPM₁₀, Carstairs Index & Indian Children	O _G	1148	1013 (±282)	0.26	0.74
	O _H	277	696 (±236)	0.99	0.01*
	O _{GH}	713	854 (±203)	0.74	0.26
	O _{S (count)}	7	3 (±2)	0.08	0.96
(G) TPM₁₀, Carstairs Index & Other South Asian Children	O _G	791	993 (±318)	0.71	0.29
	O _H	67	862 (±317)	1.00	0.00*
	O _{GH}	429	927 (±263)	0.99	0.01*
	O _{S (count)}	11	3 (±2)	0.01*	0.99
(G) TPM₁₀, Carstairs Index & Afro-Caribbean Children	O _G	567	1017 (±345)	0.93	0.07
	O _H	50	960 (±360)	1.00	0.00*
	O _{GH}	309	988 (±296)	1.00	0.00*
	O _{S (count)}	14	3 (±3)	0.00*	1.00

* p≤0.05

APPENDIX E6: Trivariate boundary overlap analysis of TPM₁₀ emissions, Carstairs Index measurements of deprivation and an individual social parameter, with annual average children's admissions of the URT (J00-06)

- APPENDIX E -

Boundaries Overlapped (G,H) H= J20-22 Admissions Per 1,000 Children	Statistic	Observed (meters)	Expected (meters)	P↑	P↓
(G) TPM₁₀	O_G	185	935 (±325)	1.00	0.00*
	O_H	657	907 (±337)	0.76	0.24
	O_{GH}	421	921 (±279)	0.99	0.01*
	O_S (count)	16	4 (±3)	0.00*	1.00
(G) Carstairs Index of Deprivation	O_G	956	927 (±279)	0.38	0.62
	O_H	427	694 (±246)	0.89	0.12
	O_{GH}	691	810 (±213)	0.68	0.32
	O_S (count)	9	3 (±2)	0.01*	0.99
(G) Smoking Prevalence	O_G	1602	911 (±272)	0.02*	0.98
	O_H	1011	723 (±261)	0.13	0.87
	O_{GH}	1312	818 (±216)	0.02*	0.98
	O_S (count)	1	4 (±2)	0.95	0.17
(G) Obesity Prevalence	O_G	1222	924 (±289)	0.13	0.87
	O_H	1108	728 (±270)	0.09	0.91
	O_{GH}	1167	827 (±231)	0.08	0.92
	O_S (count)	6	3 (±2)	0.16	0.90
(G) White British Children	O_G	947	921 (±262)	0.42	0.58
	O_H	552	638 (±233)	0.60	0.40
	O_{GH}	749	780 (±200)	0.52	0.48
	O_S (count)	4	4 (±2)	0.48	0.70
(G) White Non-British Children	O_G	742	942 (±320)	0.73	0.27
	O_H	930	858 (±301)	0.35	0.65
	O_{GH}	836	900 (±252)	0.57	0.43
	O_S (count)	5	3 (±2)	0.32	0.80
(G) Indian Children	O_G	660	914 (±281)	0.82	0.18
	O_H	558	720 (±258)	0.72	0.28
	O_{GH}	609	817 (±223)	0.83	0.17
	O_S (count)	6	3 (±2)	0.15	0.93
(G) Other South Asian Children	O_G	488	909 (±310)	0.94	0.06
	O_H	870	974 (±360)	0.54	0.46
	O_{GH}	679	942 (±279)	0.84	0.16
	O_S (count)	8	4 (±3)	0.09	0.95
(G) Afro-Caribbean Children	O_G	214	939 (±358)	1.00	0.00*
	O_H	713	1069 (±377)	0.83	0.17
	O_{GH}	464	1004 (±304)	0.99	0.01*
	O_S (count)	14	3 (±3)	0.00*	1.00

* P≤0.05

APPENDIX E7: Univariate boundary overlap analysis with annual average children's admissions of the LRT (J20-22)

- APPENDIX E -

Boundaries Overlapped (G,H) H= J20-22 Admissions Per 1,000 Children	Statistic	Observed (meters)	Expected (meters)	P↑	P↓
(G) TPM₁₀ & Carstairs Index	O _G	880	935 (±325)	0.51	0.49
	O _H	264	808 (±295)	0.99	0.01*
	O _{GH}	572	872 (±256)	0.88	0.12
	O _{S (count)}	11	3 (±2)	0.00*	1.00
(G) TPM₁₀ & Smoking prevalence	O _G	1578	910 (±276)	0.03*	0.97
	O _H	817	757 (±276)	0.35	0.65
	O _{GH}	1198	833 (±222)	0.05*	0.95
	O _{S (count)}	3	3 (±2)	0.63	0.54
(G) TPM₁₀ & Obesity prevalence	O _G	1247	918 (±287)	0.12	0.88
	O _H	1093	756 (±266)	0.11	0.89
	O _{GH}	1170	837 (±229)	0.07	0.93
	O _{S (count)}	6	3 (±2)	0.19	0.91
(G) TPM₁₀ & White British Children	O _G	874	919 (±264)	0.52	0.48
	O _H	430	638 (±221)	0.84	0.16
	O _{GH}	652	779 (±195)	0.71	0.29
	O _{S (count)}	6	3 (±2)	0.16	0.92
(G) TPM₁₀ & White Non-British Children	O _G	744	935 (±302)	0.74	0.26
	O _H	929	834 (±298)	0.31	0.69
	O _{GH}	837	885 (±241)	0.55	0.45
	O _{S (count)}	5	3 (±2)	0.28	0.83
(G) TPM₁₀ & Indian Children	O _G	619	927 (±268)	0.89	0.11
	O _H	408	685 (±244)	0.88	0.12
	O _{GH}	513	806 (±201)	0.95	0.06
	O _{S (count)}	7	4 (±2)	0.09	0.96
(G) TPM₁₀ & Other South Asian Children	O _G	344	934 (±337)	0.99	0.01*
	O _H	885	943 (±333)	0.51	0.49
	O _{GH}	614	939 (±277)	0.89	0.11
	O _{S (count)}	12	4 (±3)	0.01*	0.99
(G) TPM₁₀ & Afro-Caribbean Children	O _G	96	934 (±379)	1.00	0.00*
	O _H	860	58 (±412)	0.67	0.33
	O _{GH}	478	996 (±322)	0.98	0.02*
	O _{S (count)}	17	4 (±3)	0.00*	1.00

* P<0.05

APPENDIX E8: Bivariate boundary overlap analysis of TPM₁₀ emissions and an individual social parameter, with annual average children's admissions of the LRT (J20-22)

- APPENDIX E -

Boundaries Overlapped (G,H) H= J20-22 Admissions Per 1,000 Children	Statistic	Observed (meters)	Expected (meters)	P↑	P↓
(G) TPM₁₀, Carstairs Index & Smoking Prevalence	O _G	1344	922 (±285)	0.08	0.92
	O _H	457	703 (±239)	0.86	0.14
	O _{GH}	900	812 (±217)	0.30	0.70
	O _S (count)	6	4 (±2)	0.18	0.88
(G) TPM₁₀, Carstairs Index & Obesity Prevalence	O _G	1319	942 (±302)	0.11	0.89
	O _H	868	703 (±250)	0.23	0.77
	O _{GH}	1093	822 (±224)	0.10	0.90
	O _S (count)	6	3 (±2)	0.18	0.89
(G) TPM₁₀, Carstairs Index & White British Children	O _G	838	924 (±254)	0.60	0.40
	O _H	374	684 (±239)	0.93	0.07
	O _{GH}	606	804 (±201)	0.84	0.16
	O _S (count)	8	3 (±2)	0.02*	0.98
(G) TPM₁₀, Carstairs Index & White Non-British Children	O _G	840	918 (±332)	0.54	0.46
	O _H	879	789 (±275)	0.34	0.66
	O _{GH}	860	853 (±245)	0.46	0.54
	O _S (count)	6	4 (±3)	0.20	0.87
(G) TPM₁₀, Carstairs Index & Indian Children	O _G	716	930 (±271)	0.79	0.21
	O _H	359	676 (±239)	0.95	0.05*
	O _{GH}	538	803 (±200)	0.95	0.05*
	O _S (count)	8	3 (±2)	0.05*	0.99
(G) TPM₁₀, Carstairs Index & Other South Asian Children	O _G	762	915 (±295)	0.66	0.34
	O _H	248	848 (±306)	0.99	0.01*
	O _{GH}	505	882 (±250)	0.95	0.05*
	O _S (count)	12	3 (±2)	0.01*	0.99
(G) TPM₁₀, Carstairs Index & Afro-Caribbean Children	O _G	511	919 (±326)	0.93	0.07
	O _H	217	958 (±353)	1.00	0.00*
	O _{GH}	364	938 (±276)	1.00	0.00*
	O _S (count)	15	3 (±3)	0.00*	1.00

* p<0.05

APPENDIX E9: Trivariate boundary overlap analysis of TPM10 emissions, Carstairs Index measurements of deprivation and an individual social parameter, with annual average children's admissions of the LRT (J20-22)

- APPENDIX E -

Boundaries Overlapped (G,H) H= TPM₁₀ Emissions	Statistic	Observed (meters)	Expected (meters)	P↑	P↓
(G) Carstairs Index of Deprivation	O_G	1308	895 (±271)	0.08	0.92
	O_H	771	682 (±238)	0.30	0.70
	O_{GH}	1039	788 (±203)	0.11	0.89
	O_S (count)	5	5 (±3)	0.45	0.65
(G) Smoking Prevalence	O_G	1574	905 (±266)	0.02*	0.98
	O_H	1041	711 (±268)	0.12	0.88
	O_{GH}	1313	810 (±220)	0.02*	0.98
	O_S (count)	1	4 (±2)	0.94	0.19
(G) Obesity Prevalence	O_G	1044	895 (±269)	0.25	0.75
	O_H	288	699 (±227)	0.99	0.01*
	O_{GH}	681	798 (±203)	0.69	0.31
	O_S (count)	10	4 (±2)	0.02*	0.99
(G) White British Children	O_G	1397	898 (±248)	0.05*	0.95
	O_H	384	632 (±214)	0.90	0.10
	O_{GH}	890	765 (±18)	0.21	0.79
	O_S (count)	7	5 (±3)	0.23	0.86
(G) White Non-British Children	O_G	811	924 (±298)	0.62	0.38
	O_H	258	853 (±289)	1.00	0.00*
	O_{GH}	535	889 (±242)	0.96	0.04*
	O_S (count)	14	4 (±3)	0.00*	1.00
(G) Indian Children	O_G	1108	901 (±266)	0.18	0.82
	O_H	705	723 (±257)	0.47	0.53
	O_{GH}	906	812 (±213)	0.29	0.71
	O_S (count)	7	4 (±3)	0.22	0.86
(G) Other South Asian Children	O_G	649	930 (±329)	0.81	0.19
	O_H	619	982 (±331)	0.88	0.12
	O_{GH}	634	956 (±278)	0.90	0.10
	O_S (count)	5	3 (±2)	0.27	0.83
(G) Afro-Caribbean Children	O_G	351	892 (±335)	0.98	0.02*
	O_H	302	1035 (±355)	0.99	0.01*
	O_{GH}	326	963 (±288)	1.00	0.00*
	O_S (count)	12	3 (±3)	0.00*	1.00

* P≤0.05

APPENDIX E10: Univariate boundary overlap analysis of TPM₁₀ emissions

- APPENDIX E -

Boundaries Overlapped (G,H) H= Carstairs Index of Deprivation	Statistic	Observed (meters)	Expected (meters)	P↑	P↓
(G) Smoking Prevalence	O_G	368	692 (±195)	0.98	0.02*
	O_H	604	699 (±205)	0.65	0.35
	O_{GH}	484	696 (±164)	0.92	0.08
	O_S (count)	11	5 (±2)	0.01*	0.99
(G) Obesity Prevalence	O_G	444	684 (±192)	0.92	0.08
	O_H	1253	708 (±192)	0.01*	0.99
	O_{GH}	833	696 (±156)	0.20	0.80
	O_S (count)	12	5 (±2)	0.00*	1.00
(G) White British Children	O_G	1102	689 (±187)	0.02*	0.98
	O_H	989	635 (±179)	0.03*	0.97
	O_{GH}	1046	662 (±150)	0.02*	0.98
	O_S (count)	5	5 (±2)	0.52	0.66
(G) White Non-British Children	O_G	548	680 (±231)	0.67	0.33
	O_H	1214	865 (±254)	0.08	0.92
	O_{GH}	881	772 (±206)	0.28	0.72
	O_S (count)	7	4 (±2)	0.14	0.91
(G) Indian Children	O_G	830	693 (±224)	0.23	0.77
	O_H	921	705 (±211)	0.13	0.87
	O_{GH}	875	699 (±179)	0.13	0.87
	O_S (count)	9	5 (±2)	0.06	0.96
(G) Other South Asian Children	O_G	895	671 (±226)	0.16	0.84
	O_H	1598	958 (±285)	0.04*	0.96
	O_{GH}	1247	814 (±212)	0.04*	0.96
	O_S (count)	0	3 (±2)	1.00	0.07
(G) Afro-Caribbean Children	O_G	550	697 (±258)	0.70	0.30
	O_H	1918	1052 (±296)	0.01*	0.99
	O_{GH}	1234	874 (±225)	0.06	0.94
	O_S (count)	3	3 (±2)	0.52	0.62

* P≤0.05

APPENDIX E11: Univariate boundary overlap analysis of Carstairs Index ranks of deprivation across Leicester

- APPENDIX E -

Boundaries Overlapped (G,H) H= Smoking Prevalence	Statistic	Observed (meters)	Expected (meters)	P↑	P↓
(G) Obesity Prevalence	O_G	518	707 (±205)	0.84	0.16
	O_H	656	721 (±233)	0.57	0.43
	O_{GH}	586	714 (±169)	0.77	0.23
	O_S (count)	13	4 (±2)	0.00*	1.00
(G) White British Children	O_G	1218	706 (±204)	0.02*	0.98
	O_H	1083	643 (±196)	0.03*	0.97
	O_{GH}	1149	674 (±158)	0.00*	1.00
	O_S (count)	8	5 (±2)	0.14	0.93
(G) White Non-British Children	O_G	571	715 (±239)	0.70	0.30
	O_H	797	869 (±265)	0.56	0.44
	O_{GH}	686	793 (±213)	0.68	0.32
	O_S (count)	9	4 (±2)	0.03*	0.98
(G) Indian Children	O_G	836	702 (±225)	0.23	0.77
	O_H	1018	704 (±198)	0.07	0.93
	O_{GH}	929	703 (±173)	0.11	0.89
	O_S (count)	9	4 (±2)	0.06	0.96
(G) Other South Asian Children	O_G	709	691 (±241)	0.43	0.57
	O_H	1536	958 (±294)	0.05*	0.95
	O_{GH}	1130	825 (±212)	0.09	0.91
	O_S (count)	5	4 (±2)	0.30	0.82
(G) Afro-Caribbean Children	O_G	689	711 (±260)	0.49	0.51
	O_H	2121	1022 (±313)	0.01*	0.99
	O_{GH}	1419	868 (±233)	0.02*	0.98
	O_S (count)	2	3 (±2)	0.81	0.39

* p≤0.05

APPENDIX E12: Univariate boundary overlap analysis of adult smoking prevalence

- APPENDIX E -

Boundaries Overlapped (G,H) H= Obesity Prevalence	Statistic	Observed (meters)	Expected (meters)	P↑	P↓
(G) White British Children	O_G	1224	715 (±203)	0.02*	0.98
	O_H	677	650 (±193)	0.38	0.62
	O_{GH}	940	683 (±165)	0.07	0.93
	O_S (count)	12	5 (±2)	0.02*	0.99
(G) White Non-British Children	O_G	446	732 (±236)	0.91	0.09
	O_H	564	879 (±256)	0.92	0.08
	O_{GH}	507	806 (±204)	0.95	0.05*
	O_S (count)	11	4 (±2)	0.01*	1.00
(G) Indian Children	O_G	1112	717 (±203)	0.04*	0.96
	O_H	839	712 (±218)	0.23	0.77
	O_{GH}	971	715 (±175)	0.07	0.93
	O_S (count)	12	4 (±2)	0.00*	1.00
(G) Other South Asian Children	O_G	507	719 (±251)	0.80	0.20
	O_H	1231	974 (±308)	0.19	0.81
	O_{GH}	883	848 (±234)	0.42	0.58
	O_S (count)	7	3 (±2)	0.11	0.93
(G) Afro-Caribbean Children	O_G	552	733 (±277)	0.74	0.26
	O_H	1347	1044 (±318)	0.16	0.84
	O_{GH}	965	890 (±246)	0.34	0.66
	O_S (count)	7	3 (±2)	0.07	0.96

* p≤0.05

APPENDIX E13: Univariate boundary overlap analysis of adult obesity prevalence

- APPENDIX E -

Boundaries Overlapped (G,H) H= White British Children	Statistic	Observed (meters)	Expected (meters)	P↑	P↓
(G) White Non-British Children	O _G	1114	635 (±213)	0.03*	0.97
	O _H	1303	857 (±241)	0.05*	0.95
	O _{GH}	1201	746 (±185)	0.02*	0.98
	O _S (count)	2	4 (±2)	0.89	0.27
(G) Indian Children	O _G	171	636 (±188)	1.00	0.00*
	O _H	371	708 (±199)	0.98	0.02*
	O _{GH}	271	672 (±160)	1.00	0.00*
	O _S (count)	19	5 (±2)	0.00*	1.00
(G) Other South Asian Children	O _G	369	658 (±237)	0.90	0.10
	O _H	917	950 (±270)	0.49	0.51
	O _{GH}	643	804 (±203)	0.79	0.21
	O _S (count)	10	4 (±2)	0.00*	1.00
(G) Afro-Caribbean Children	O _G	370	653 (±245)	0.92	0.08
	O _H	1061	1062 (±317)	0.44	0.56
	O _{GH}	716	857 (±224)	0.73	0.27
	O _S (count)	6	3 (±2)	0.13	0.94

* P≤0.05

APPENDIX E14: Univariate boundary overlap analysis of 'White British' residency levels

Boundaries Overlapped (G,H) H= White Non-British Children	Statistic	Observed (meters)	Expected (meters)	P↑	P↓
(G) Indian Children	O _G	967	848 (±256)	0.28	0.72
	O _H	1123	705 (±220)	0.04*	0.96
	O _{GH}	1045	776 (±195)	0.09	0.91
	O _S (count)	4	4 (±3)	0.58	0.57
(G) Other South Asian Children	O _G	920	855 (±278)	0.36	0.64
	O _H	1013	952 (±317)	0.37	0.63
	O _{GH}	966	904 (±252)	0.37	0.63
	O _S (count)	0	4 (±3)	1.00	0.11
(G) Afro-Caribbean Children	O _G	409	855 (±307)	0.96	0.04*
	O _H	1143	1060 (±357)	0.35	0.65
	O _{GH}	776	957 (±281)	0.72	0.28
	O _S (count)	10	3 (±2)	0.01*	0.99

* P≤0.05

APPENDIX E15: Univariate boundary overlap analysis of 'White Non-British' residency levels

- APPENDIX E -

Boundaries Overlapped (G,H) H= Indian Children	Statistic	Observed (meters)	Expected (meters)	P↑	P↓
(G) Other South Asian Children	O _G	312	715 (±269)	0.97	0.03*
	O _H	821	1052 (±309)	0.79	0.21
	O _{GH}	566	883 (±244)	0.91	0.09
	O _S (count)	8	3 (±2)	0.04*	0.99
(G) Afro-Caribbean Children	O _G	357	1038 (±353)	0.99	0.01*
	O _H	569	968 (±381)	0.89	0.11
	O _{GH}	463	1003 (±306)	0.99	0.01*
	O _S (count)	11	3 (±3)	0.02*	1.00

* P≤0.05

APPENDIX E16: Univariate boundary overlap analysis of 'Indian' residency levels

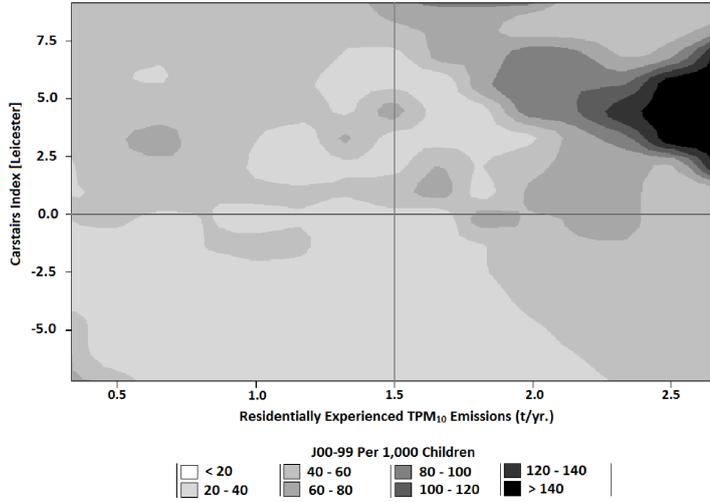
Boundaries Overlapped (G,H) H= Other South Asian Children	Statistic	Observed (meters)	Expected (meters)	P↑	P↓
(G) Afro-Caribbean Children	O _G	569	967 (±381)	0.89	0.11
	O _H	357	1038 (±353)	0.99	0.01*
	O _{GH}	463	1003 (±306)	0.99	0.01*
	O _S (count)	11	3 (±3)	0.02*	1.00

* P≤0.05

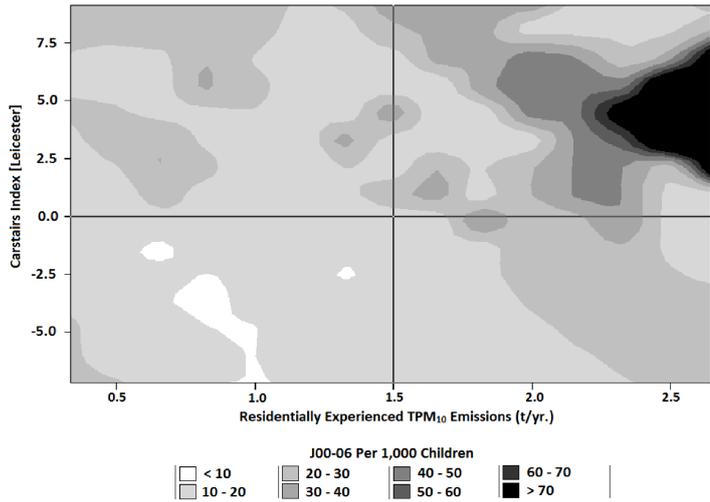
APPENDIX E17: Univariate boundary overlap analysis of 'Other South Asian' residency levels

APPENDIX F

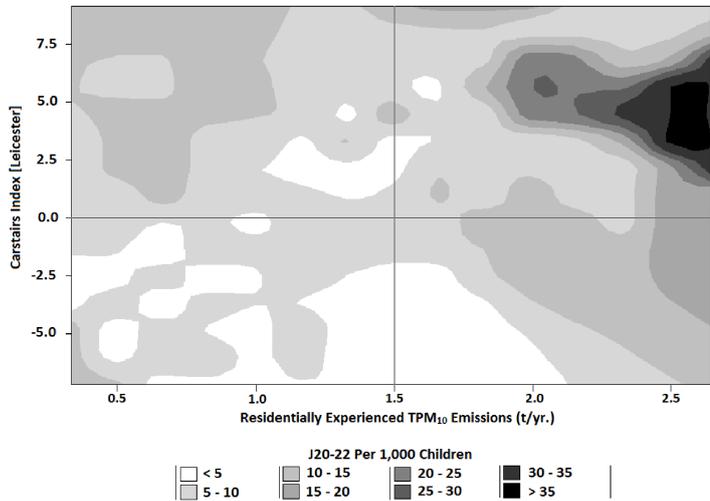
Contour Plots Of Children's J00-99 Hospital Admissions Vs Deprivation & Experienced TPM₁₀ Emissions



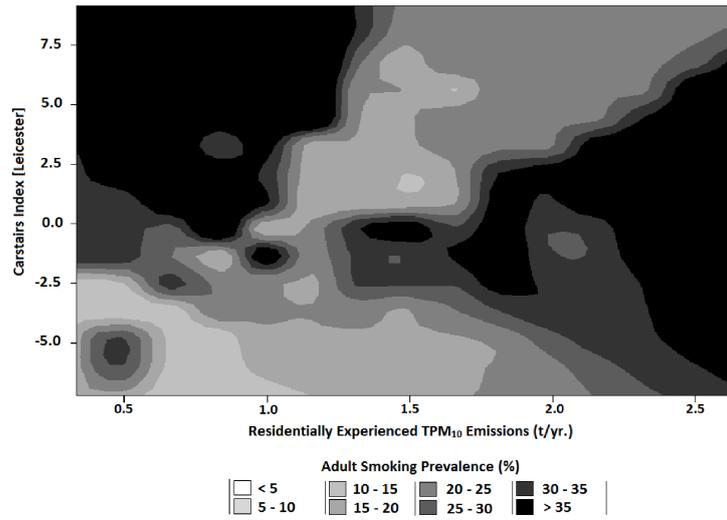
Contour Plots Of Children's J00-06 Hospital Admissions Vs Deprivation & Experienced TPM₁₀ Emissions



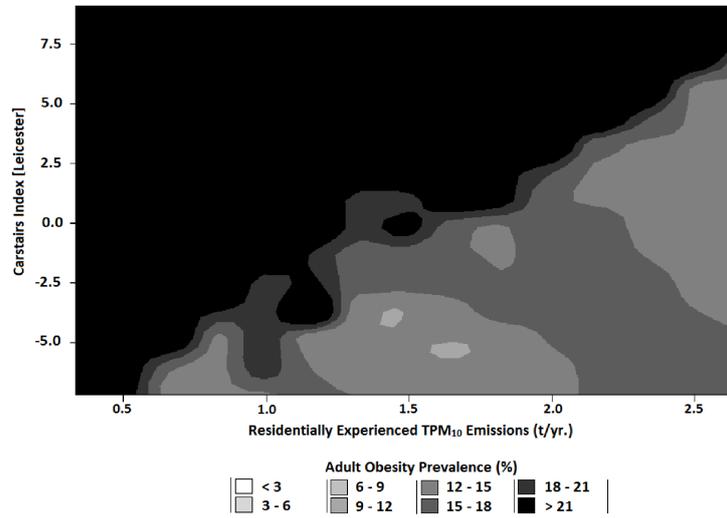
Contour Plots Of Children's J20-22 Hospital Admissions Vs Deprivation & Experienced TPM₁₀ Emissions



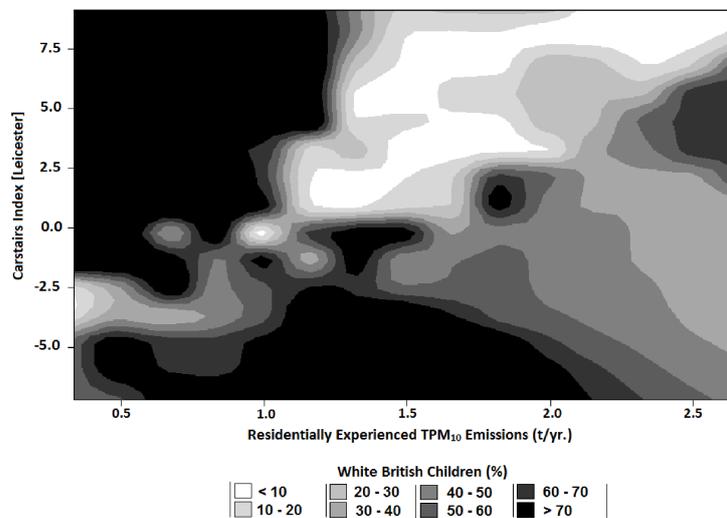
Contour Plots Of Smoking Prevalence Vs Deprivation & Experienced TPM₁₀ Emissions



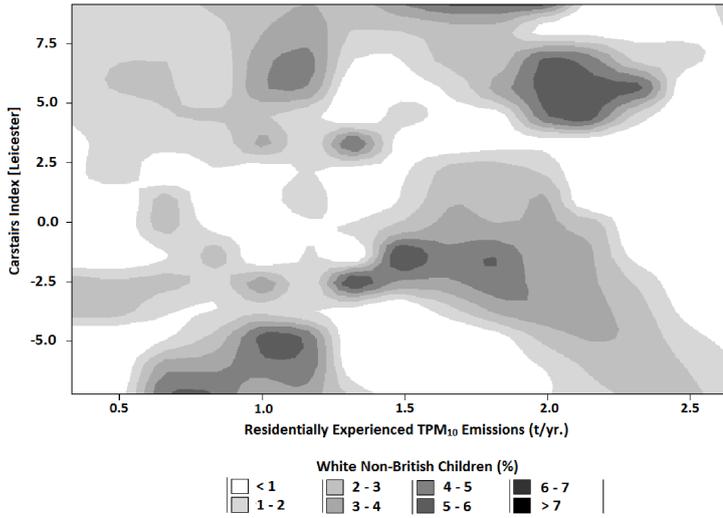
Contour Plots Of Obesity Prevalence Vs Deprivation & Experienced TPM₁₀ Emissions



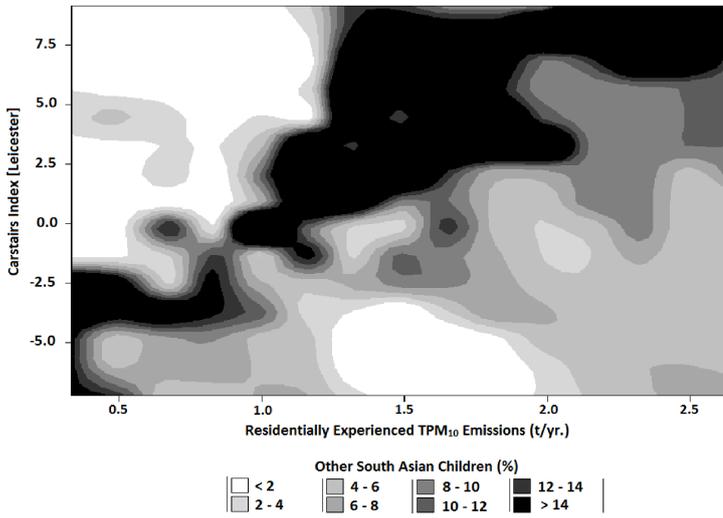
Contour Plots Of White British Residency Vs Deprivation & Experienced TPM₁₀ Emissions



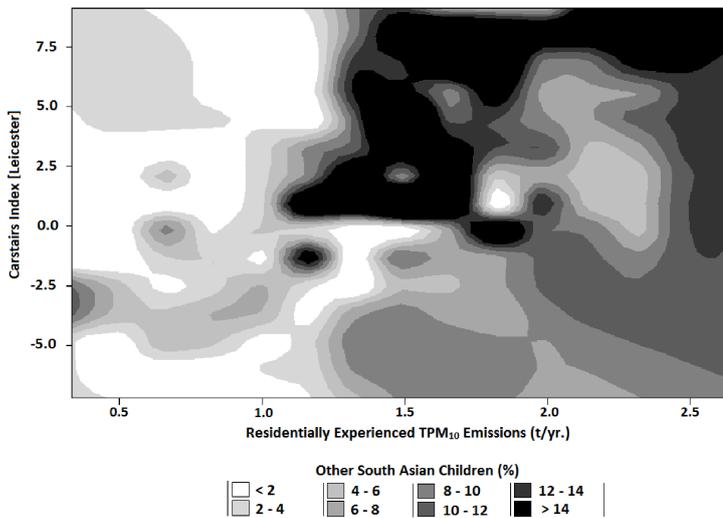
Contour Plots Of White Non-British Residency Vs Deprivation & Experienced TPM₁₀ Emissions

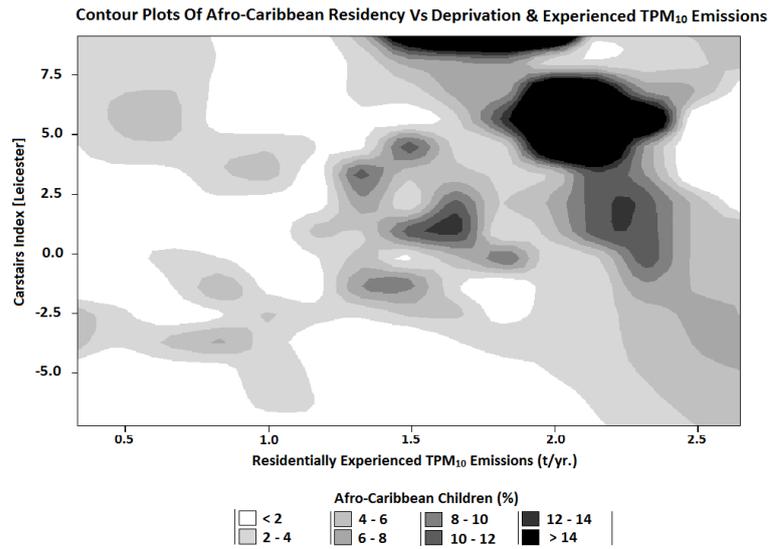


Contour Plots Of Indian Residency Vs Deprivation & Experienced TPM₁₀ Emissions



Contour Plots Of Other South Asian Residency Vs Deprivation & Experienced TPM₁₀ Emissions





APPENDIX F1 [PAGES 420-423]: Contour plots, exploring Leicester’s ‘Double Burden’ through simultaneously plotting levels of deprivation and residentially experienced TPM₁₀ emissions against a third measurement of respiratory or social status

- APPENDIX F -

Boundaries Overlapped (G,H) H= Created TPM₁₀ Emissions	Statistic	Observed (meters)	Expected (meters)	P↑	P↓
(G) Experienced TPM₁₀ Emissions	O_G	293	794 (±263)	1.00	0.00*
	O_H	1183	908 (±287)	0.17	0.83
	O_{GH}	738	851 (±225)	0.69	0.31
	O_S (count)	8	4 (±2)	0.07	0.95
(G) Carstairs Index of Deprivation	O_G	305	786 (±224)	1.00	0.00*
	O_H	446	701 (±243)	0.87	0.13
	O_{GH}	375	743 (±194)	0.99	0.01*
	O_S (count)	14	5 (±3)	0.00*	1.00
(G) Smoking Prevalence	O_G	461	791 (±237)	0.95	0.05*
	O_H	996	696 (±231)	0.11	0.89
	O_{GH}	723	744 (±191)	0.50	0.50
	O_S (count)	9	4 (±2)	0.04*	0.98
(G) Obesity Prevalence	O_G	332	789 (±235)	0.99	0.01*
	O_H	1294	714 (±246)	0.04*	0.96
	O_{GH}	795	752 (±197)	0.37	0.63
	O_S (count)	14	4 (±2)	0.00*	1.00
(G) White British Children	O_G	1292	797 (±235)	0.04*	0.96
	O_H	1071	648 (±216)	0.04*	0.96
	O_{GH}	1182	722 (±183)	0.02*	0.98
	O_S (count)	6	5 (±3)	0.32	0.79
(G) White Non-British Children	O_G	340	795 (±247)	0.99	0.01*
	O_H	1173	872 (±294)	0.15	0.85
	O_{GH}	756	833 (±218)	0.61	0.39
	O_S (count)	9	4 (±2)	0.05*	0.97
(G) Indian Children	O_G	1195	783 (±241)	0.05*	0.95
	O_H	1213	723 (±249)	0.04*	0.96
	O_{GH}	1204	753 (±201)	0.03*	0.97
	O_S (count)	4	5 (±3)	0.64	0.51
(G) Other South Asian Children	O_G	861	785 (±288)	0.34	0.66
	O_H	1316	961 (±305)	0.12	0.88
	O_{GH}	1089	873 (±243)	0.17	0.83
	O_S (count)	3	3 (±2)	0.62	0.56
(G) Afro-Caribbean Children	O_G	593	788 (±277)	0.75	0.25
	O_H	1690	1035 (±322)	0.04*	0.96
	O_{GH}	1142	911 (±247)	0.18	0.82
	O_S (count)	5	3 (±2)	0.24	0.87

* P≤0.05

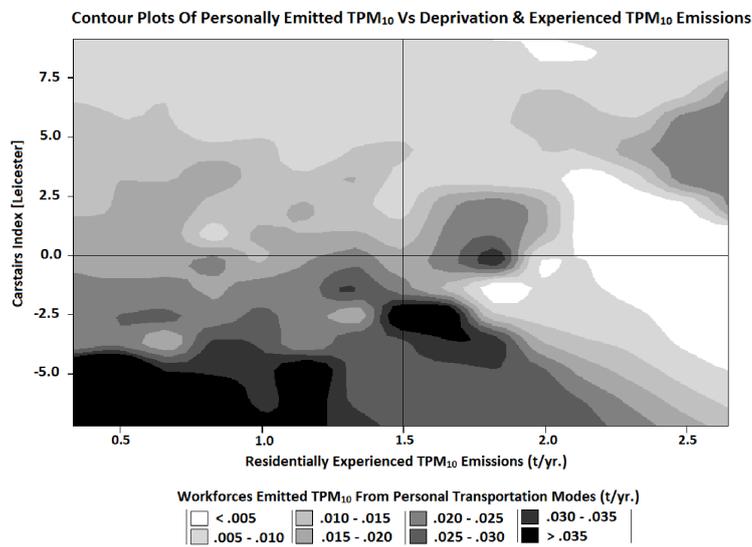
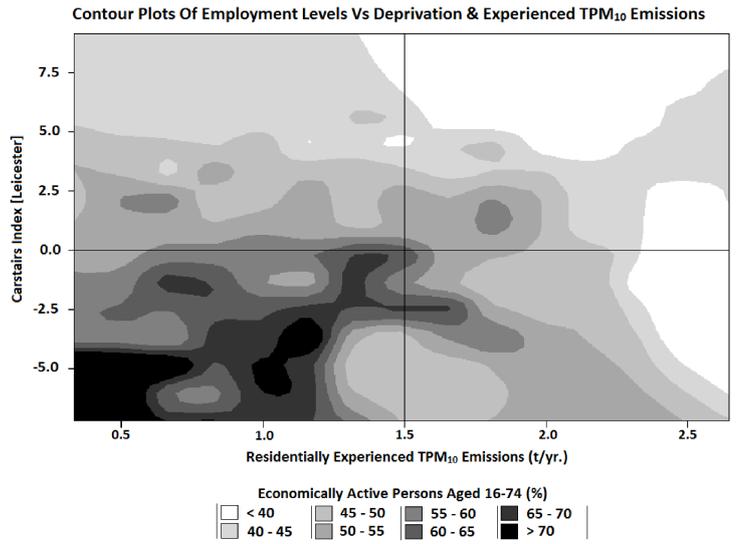
APPENDIX F2: Univariate boundary overlap analysis of community created transport emission levels (from private modes) and social-environmental influences of interest

- APPENDIX F -

Boundaries Overlapped (G,H) H= Created TPM ₁₀ Emissions	Statistic	Observed (meters)	Expected (meters)	P↑	P↓
(G) Carstairs Index, Experienced TPM₁₀ Emissions & Smoking Prevalence	O _G	369	777 (±235)	0.99	0.01*
	O _H	639	690 (±225)	0.55	0.45
	O _{GH}	504	733 (±183)	0.92	0.08
	O _S (count)	13	4 (±2)	0.00*	1.00
(G) Carstairs Index, Experienced TPM₁₀ Emissions & Obesity Prevalence	O _G	156	758 (±222)	1.00	0.00*
	O _H	842	692 (±215)	0.21	0.79
	O _{GH}	499	725	0.93	0.07
	O _S (count)	18	4 (±2)	0.00*	1.00
(G) Carstairs Index, Experienced TPM₁₀ Emissions & White British Children	O _G	809	806 (±242)	0.43	0.57
	O _H	626	664 (±211)	0.52	0.48
	O _{GH}	717	735 (±179)	0.48	0.52
	O _S (count)	12	4 (±2)	0.00*	1.00
(G) Carstairs Index, Experienced TPM₁₀ Emissions & White Non-British Children	O _G	265	793 (±259)	1.00	0.00*
	O _H	1144	819 (±268)	0.11	0.89
	O _{GH}	705	806 (±218)	0.65	0.35
	O _S (count)	14	4 (±3)	0.00*	1.00
(G) Carstairs Index, Experienced TPM₁₀ Emissions & Indian Children	O _G	700	780 (±225)	0.61	0.39
	O _H	589	688 (±216)	0.64	0.36
	O _{GH}	645	734 (±179)	0.71	0.29
	O _S (count)	12	4 (±2)	0.00*	1.00
(G) Carstairs Index, Experienced TPM₁₀ Emissions & Other South Asian Children	O _G	352	784 (±265)	0.98	0.02*
	O _H	380	874 (±285)	0.98	0.02*
	O _{GH}	366	829 (±230)	0.99	0.01*
	O _S (count)	15	4 (±3)	0.00*	1.00
(G) Carstairs Index, Experienced TPM₁₀ Emissions & Afro-Caribbean Children	O _G	325	795 (±282)	0.98	0.02*
	O _H	380	951 (±310)	1.00	0.00*
	O _{GH}	353	873 (±246)	1.00	0.00*
	O _S (count)	14	4 (±3)	0.00*	1.00

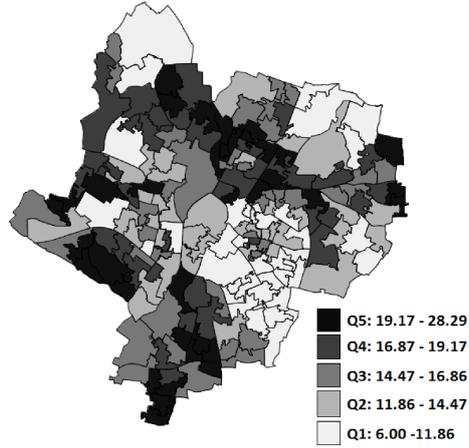
* P≤0.05

APPENDIX F3: Trivariate boundary overlap analysis of community created transport emission levels (from private modes) with deprivation, residential TPM₁₀ exposures and a final social parameter

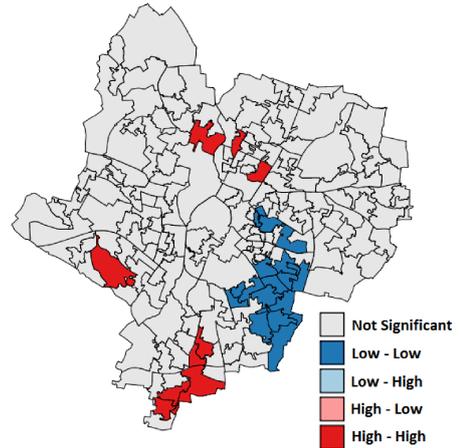


APPENDIX F4: Contour plots, exploring Leicester’s ‘Double Burden’ in relation to levels of employment and then community created transport emissions (from personal modes)

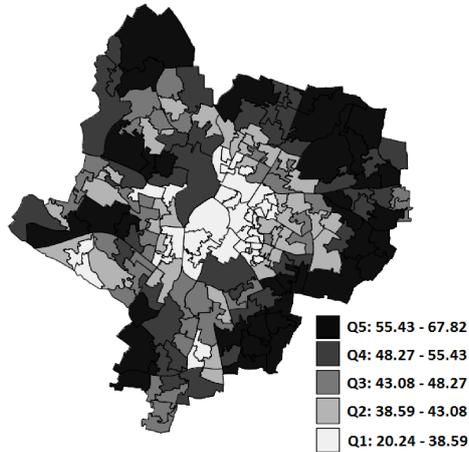
% Commutes Via Public Transport (Bus, Minibus, Coach)



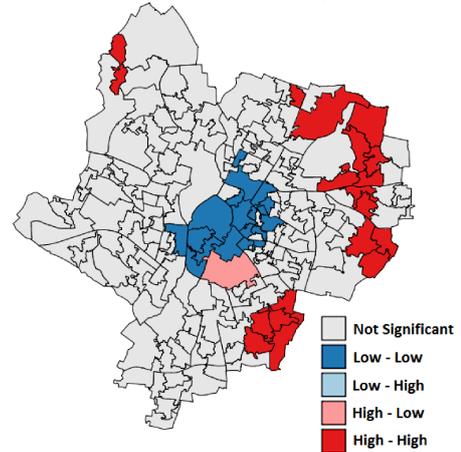
Univariate Local Moran's I:
Commutes Via Public Transport [R² = 0.47]



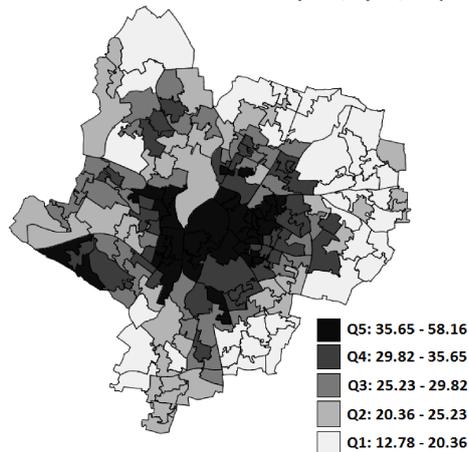
% Commutes Via Personal Transport (Car, Van)



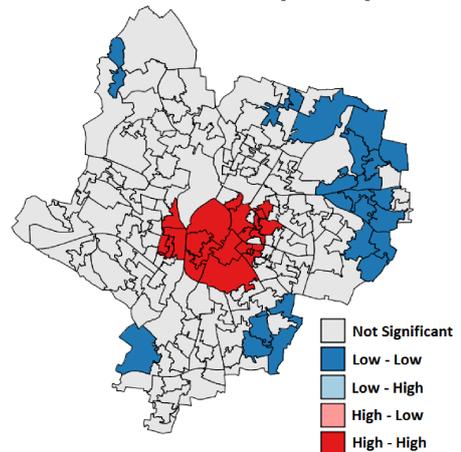
Univariate Local Moran's I:
Commutes Via Personal Transport [R² = 0.71]



% Commutes Via 'Green' Modes (Foot, Cycle, Carpool)



Univariate Local Moran's I:
Commutes Via 'Green' Modes [R² = 0.77]



APPENDIX F5: Local Moran's I cluster and outlier analysis, representing the uptake of specific modes of transportation (%) by communities on their daily commute to work

	Bivariate: Local Moran's Statistic			
	R ²	Pearson's R	I Value	Z-Score
Public Transport	0.47	0.68	0.46	10.36
Personal Transport	0.71	0.84	0.64	14.77
Green Modes	0.77	0.88	0.72	16.27

APPENDIX F6: Summary of the Local Moran's statistical analysis [Appendix F5], describing the spatial uptake of individual transportation forms (%) by community residents on their daily commute

	Bivariate: Local Moran's Statistic			
	R ²	Pearson's R	I Value	Z-Score
Total	0.40	0.63	0.43	9.75
Public Transport	0.05	0.21	0.09	2.08
Personal Transport	0.50	0.71	0.48	10.93
Green Modes	0.28	0.53	0.33	7.68

APPENDIX F7: Summary of the Local Moran's statistical analysis [Figure 7.6], describing the spatial structures of those commute distances travelled per employed person (km/PEP) in total and by individual transportation forms