# Understanding the Sources, Composition, and Health Risks of $PM_{2.5}$ in Indoor Environments

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# Abstract

Humans spend the majority of their time indoors, yet research into inhalable exposures has largely focused on outdoor or occupational settings. Comparatively little monitoring has been conducted in indoor residential environments. This thesis investigates the composition and concentrations of particulate matter in residential indoor environments, with particular focus on compounds of known toxicological relevance, including phthalates and polycyclic aromatic hydrocarbons (PAHs).

A $PM_{2.5}$ sampling method was developed for use in occupied real-home environments without disrupting occupant activity. This approach enabled realistic measurement of indoor particle composition while maintaining the natural conditions of the sampling environment. Collected samples were analysed to quantify target analytes, compare chemical composition, and assess toxicity using toxic equivalency approaches. Results showed substantial variability between households, influenced by occupant behaviour, location, and socioeconomic factors. Benzo[a]pyrene-equivalent analysis of PAHs indicated that all households exceeded WHO guideline values. Phthalates, particularly dibutyl phthalate and bis(2-ethylhexyl) phthalate (DEHP), were detected in nearly all homes, with DEHP contributing most to overall toxicity potential. These findings highlight that while mass concentration is a useful exposure indicator, chemical composition is critical in understanding associated health risks.

To complement residential sampling, controlled cooking experiments were conducted in a controlled kitchen environment. These experiments examined $PM_{2.5}$ and volatile organic compound (VOC) emissions from common household cooking activities. GC×GC-ToFMS analysis showed that meat-containing dishes tended to emit higher concentrations of lipid-derived compounds, while vegetarian dishes containing dairy-derived products showed higher emissions of unsaturated fatty aldehydes. Lipid-derived compounds such as fatty acids and sterols are less volatile and tend to partition into the particulate phase, contributing to elevated $PM_{2.5}$ levels following cooking. In contrast, unsaturated fatty aldehydes are formed through thermal oxidation of fatty acids, are more chemically reactive, and have been linked to respiratory irritation.

Importantly, these compounds are not exclusive to either meat or vegetarian cooking. Their formation depends largely on ingredient composition, particularly fat type and content, as well as cooking conditions. Differences between recipes were greater than differences between meat and vegetarian versions of the same dish, suggesting that ingredient composition plays a more significant role than meal type in emission profiles and associated health risks.

This work contributes to a broader understanding of indoor particulate matter composition, variability, and potential health risks, highlighting the need for continued research into indoor emission sources and improved exposure assessment in residential environments.

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# Author's Declaration

I declare that this thesis is a presentation of original work and that I am the sole author. This work has not previously been presented for a degree or other qualification at this University or elsewhere. All sources are acknowledged as references, and all collaborators have been acknowledged where applicable.

# Chapter 1: Introduction

# 1 Introduction

Humans spend up to 90 percent of their time in indoor environments, but research into inhalable exposures has largely focused on outdoor urban and industrial sources and settings[1, 2]. Comparatively little monitoring has occurred in indoor, residential environments[1]. This is not to say that indoor pollutants will inherently cause more adverse health effects, but further investigation into this field must be completed in order to have a more accurate estimation of individuals' exposures over their lifetimes. In addition, many of the regulations relating to pollutant exposure are based on ambient outdoor concentrations; as individuals spend most of their time indoors in residential, school, or office settings, this may lead to overexposure to certain pollutants, some of which can be higher indoors than at ambient levels[3]. Despite the proportion of time that humans spend indoors, there are only a few countries (Finland, Portugal, Slovenia, Lithuania) which regulate indoor air quality outside of industrial settings[4].

The concentration of an indoor pollutant is dependent upon the relationship between the volume of air in the enclosed space, the generation or release rate of the particular pollutant, the removal rate (i.e. via settling or reaction), the rate of air exchange with the outdoor environment, and the concentration of pollutants in the outdoor environment [3]. Actual levels of exposure to humans is more difficult to quantify as exposure is heavily impacted by personal behaviours [5, 6]. Studies undertaken by the US Environmental Protection Agency (EPA) have shown that personal exposures to many pollutants can exceed measurements taken at ambient levels when high particle-emitting events take place [7]. Particle-emitting events may be continuous (infiltration from outdoors, heating systems), or intermittent (cleaning, cooking, burning wood or candles).

A number of problems arise when attempting to sample in indoor environments as many of the instruments developed for outdoor use are not suitable for indoor use[3]. This is due to the cost, size, noise, and the amount of air that these instruments displace. In addition, many of these instruments intended for indoor use are designed to take long term measurements over several hours or days. This provides good estimates for long term exposures, but may prevent the identification of short-term, acute exposures[3]. This review will focus on particulate matter as an indoor pollutant. Particulate matter consists of liquid droplets and solid particles suspended in the air.

## 1.1 Existing Policies and Regulations

Ambient air pollution is subject to regulations in many countries; these include legally enforceable standards for particulate matter, nitrogen dioxide, ozone, and other pollutants. However, indoor air is often regulated only indirectly through building codes, ventilation requirements, or occupational exposure standards. In the United Kingdom, the 2019 *Clean Air Strategy* acknowledges indoor air pollution as a public health concern and encourages efforts to reduce emissions from domestic sources; however, it does not establish specific concentration limits for pollutants in residential environments[8].

It is important to distinguish between residential or public indoor environments and occupational or industrial settings. In industrial and workplace environments, indoor air quality is subject to extensive monitoring and regulation through occupational health and safety legislation. As an example, the UK's Control of Substances Hazardous to Health (COSHH) Regulations and the US Occupational Safety and Health Administration (OSHA) standards specify occupational exposure limits (OELs) for a wide range of inhalable pollutants[9, 10]. These regulations require employers to monitor workplace environments, assess risks to employees, and implement mitigation measures such as ventilation, substitution, or the use of personal protective equipment when concentrations exceed prescribed limits. Similar policies exist in the European Union, where workplace exposure limits (WELs) are implemented through directives such as the EU Chemical Agents Directive (98/24/EC) and the Carcinogens, Mutagens and Reprotoxic Substances Directive (2004/37/EC)[11, 12].

In contrast, the majority of non-industrial indoor environments lack such enforced standards. The UK Air Quality Expert Group (AQEG) has emphasized that despite individuals spending approximately 80–90% of their time indoors, systematic monitoring and regulation of indoor air quality remains limited [13]. Further guidance from UK-specific bodies reinforces the importance of considering indoor exposures. The Committee on the Medical Effects of Air Pollutants (COMEAP) has estimated that long-term exposure to $PM_{2.5}$ contributes to tens of thousands of attributable deaths annually in the UK, primarily through cardiovascular and respiratory disease pathways [14]. While these estimates are based largely on ambient concentrations, they are highly relevant to indoor environments given that the majority of exposure occurs indoors. Unlike outdoor air, where pollutant concentrations are routinely measured through national networks, there is no equivalent framework for residential or public indoor environments[4]. The absence of monitoring contributes to knowledge gaps and does not allow for the accurate assessment of human exposure across indoor environments.

The World Health Organization (WHO) has issued several guidance documents aimed at closing this gap. The WHO Guidelines for Indoor Air Quality: Selected Pollutants and the WHO Global Air Quality Guidelines provide reference concentrations for key pollutants, including particulate matter ($PM_{2.5}$ and $PM_{10}$), nitrogen dioxide, carbon monoxide, and formaldehyde, among others[15, 16]. Although these values are not legally binding, they serve as benchmarks for policymakers to develop or revise national indoor air quality standards.

In practice, indoor pollutant concentrations are influenced by a combination of both indoor and outdoor sources. Activities such as cooking, heating, and cleaning contribute to elevated pollutant levels indoors, often exceeding outdoor concentrations, particularly in energy-efficient, airtight buildings where ventilation is limited[3]. As a result, usage of outdoor air quality metrics may underestimate total human exposure, given that the majority of exposure occurs indoors. Vulnerable populations, including children, the elderly, and individuals with chronic respiratory or cardiovascular conditions, are especially susceptible to the effects of elevated indoor pollutant concentrations[17].

As understanding of indoor emission sources grows, the development of future policies will likely depend on improved monitoring technologies, standardized exposure metrics, and closer collaboration between environmental, health, and occupational safety agencies.

## 1.2 Particle Size and Lung Deposition

Airborne particles can be separated into three different size modes: nucleation, accumulation, and coarse[18]. Nucleation, which is the smallest size mode, includes particles formed through source condensation processes or via atmospheric chemical reactions. When these particles coagulate or condensate, they form the accumulation mode, where they have the potential to remain in the atmosphere for a long time[18]. When particles have a diameter of $>2.5$ $\mu$m, they are part of the coarse mode[19]. These are often formed through mechanical processes, and can be emitted directly from a source (primary emissions) or formed via secondary reactions in the atmosphere[19].

In atmospheric chemistry, it is accepted that in terms of number concentration, the majority of airborne particles fall within the ultrafine range, with diameters of less than 100 nm. In contrast, larger particles make up the greatest proportion of total particle mass[20]. The behaviour of particles (both in the air and once inhaled) is largely dependent upon their physical characteristics, of which size is one of the most significant[21]. Particle size deter-

mines how long particles remain airborne, how they are transported within the respiratory tract, and where they are ultimately deposited[21].

Fine particulate matter (PM2.5, particles with an aerodynamic diameter of less than 2.5 µm) can penetrate deeply into the respiratory system, depositing in the alveoli and in some cases entering the bloodstream via the pulmonary epithelium[22]. In contrast, coarse particles (PM10, 2.5–10 µm) are primarily deposited in the upper airways and tracheobronchial regions, while ultrafine particles (PM0.1, <100 nm) have the greatest potential for intrusion into other systems within the body, including access to the cardiovascular and central nervous systems[22]. Figure 1 depicts this phenomenon. This size-dependent deposition behaviour highlights why PM2.5 and smaller fractions are often the focus of epidemiological and toxicological studies.

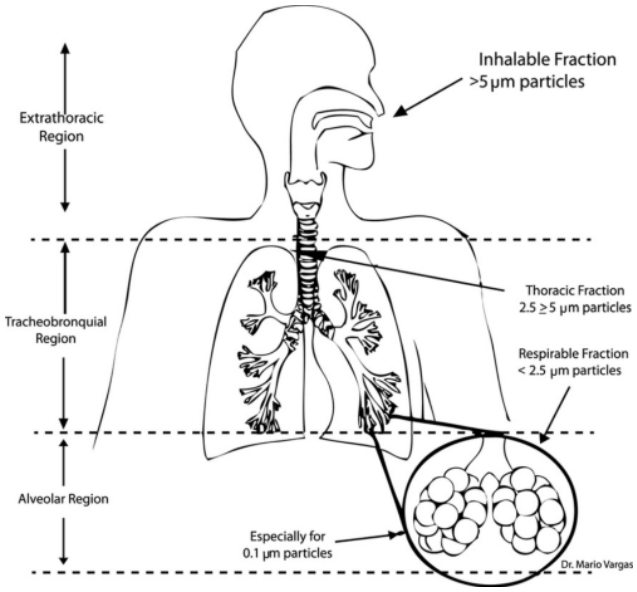



**Figure 1:** A figure showing the lung deposition of particles of varying size. Source: Sierra-Vargas et.al, *Air pollution: Impact and prevention*[23]

The deposition of inhaled particles occurs through several physical mechanisms, including inertial impaction, sedimentation, and diffusion[24]. Inertial impaction is dominant for larger, coarse particles, which tend to deviate from airflow streamlines and deposit in the nasal cavity and pharynx. Gravitational settling (sedimentation) affects mid-sized particles, typically in the tracheobronchial region, while Brownian diffusion allows ultrafine particles to reach and deposit within the alveolar regions[24].

Beyond physical deposition, the toxicological impact of particulate matter depends on its chemical composition and surface area. Particles can cause adverse health effects through

several mechanisms, primarily involving oxidative stress and inflammation[25]. Following deposition in the respiratory tract, reactive components such as transition metals, PAHs, and organic carbon species can result in the generation of reactive oxygen species[25]. This can lead to oxidative damage to cellular membranes, DNA, and proteins. Persistent oxidative stress may contribute to the development of chronic respiratory and cardiovascular diseases as well as carcinogenesis[25].

Ultrafine particles, due to their high surface area–to–mass ratio, exhibit enhanced reactivity compared to larger particles[26]. Their surfaces can adsorb toxic chemicals and biological agents, acting as carriers that facilitate transport into deeper lung tissue and potentially into systemic circulation[26]. These particles can thus provoke inflammatory responses beyond the lungs, including endothelial dysfunction and oxidative damage in the cardiovascular system[26]. These mechanisms, when combined with chronic exposure, may explain the strong epidemiological links observed between fine particulate matter and increased rates of morbidity and mortality.

Some research suggests that improving residential and indoor environments represents one of the most cost-effective approaches for reducing the overall population exposure burden [27]. Annual costs of environmentally attributable childhood illness including asthma, cancer, and lead poisoning are estimated to be 54.9 billion USD per annum. While not all of this is attributable to indoor environmental exposures, their contribution is thought to be significant [28]. However, more detailed investigations are required, particularly for indoor settings such as schools, elderly care facilities, and workplaces, where individuals spend substantial portions of their time and where exposure to ultrafine and fine particulate matter may be prolonged[27].

## 1.3 Chemical and Biological Components of Particulate Matter

Particulate matter (PM) is a mixture of solid and liquid particles suspended in air, comprising a wide range of chemical and biological components originating from both indoor and outdoor sources. The composition of $PM_{2.5}$ is highly variable, and understanding the chemical and biological constituents of PM is essential for interpreting exposure pathways and associated health effects [3].

Chemically, $PM_{2.5}$ typically consists of organic compounds, inorganic ions, trace metals, and elemental carbon. Organic matter can account for a substantial fraction of fine particulate mass and includes a wide variety of compounds such as PAHs, oxygenated organics, and

semi-volatile organic compounds (SVOCs). Many of these compounds are produced through incomplete combustion processes, including cooking, smoking, and outdoor vehicular emissions that infiltrate indoor environments [3, 29]. Inorganic components, such as sulfates, nitrates, and ammonium, are often derived from secondary atmospheric processes outdoors but can contribute significantly to indoor PM through infiltration [30]. Trace metals, including iron, zinc, and lead, may originate from both outdoor sources (e.g. traffic emissions) and indoor activities such as cooking and abrasion of building materials [31].

Biological components of indoor particulate matter include bacteria, fungal spores, pollen, endotoxins, and fragments of biological origin. Biological particles can be emitted directly from occupants (e.g., skin cells), pets, or resuspended dust, and are strongly influenced by occupancy, ventilation, and hygiene practices [32].

The relative contribution of these components varies depending on indoor activities and external influences. Cooking has been identified as one of the dominant indoor sources of $PM_{2.5}$, generating both organic aerosols and ultrafine particles, with smoking as the other dominant source of indoor PM [33]. In addition, cleaning activities and occupant movement within the home can resuspend settled dust, increasing concentrations of both inorganic and biological particles [34].

Infiltration of ambient air introduces particles from traffic emissions, industrial activities, and pollution events, which also contribute to the composition of indoor PM [35].

Table 1 summarises the major chemical and biological components of indoor particulate matter, along with their primary sources and typical associated activities.

**Table 1:** Major components of indoor particulate matter and their sources

| **Component Type** | **Examples** | **Sources/Activities** |
|---|---|---|
| Organic compounds | PAHs, VOCs | Cooking, smoking, combustion |
| Inorganic ions | Sulfate, nitrate, ammonium | Outdoor infiltration, secondary aerosols |
| Trace metals | Iron, zinc, lead | Traffic emissions, cooking, dust |
| Elemental carbon | Soot | Combustion, outdoor pollution |
| Biological particles | Bacteria, spores, pollen | Occupants, pets, resuspension |
| Dust/resuspended particles | Dead skin cells | Cleaning, movement, airflow |

Overall, the composition of indoor PM reflects a dynamic balance between indoor emissions, occupant behaviour, and intrusion from outdoors. This complexity presents challenges for both measurement and source attribution, necessitating advanced analytical techniques capable of resolving a wide range of chemical species.

## 1.4 Sources and Composition

The composition of outdoor particulate matter is well-characterised, with combustion-related activities such as vehicle emissions and industrial processes being the main sources of $PM_{2.5}$ in outdoor environments[22]. In contrast, in indoor environments, many studies have found cooking and smoking to be the largest contributors to indoor particulate matter concentrations, while the use of cleaning products has a comparatively lesser effect[33].

### 1.4.1 Indoor Sources

Several studies have identified cooking activity as the one of the most significant sources of particulate pollution in indoor environments[36]. As cooking and eating meals are an important aspect of human cultural and societal norms, the majority of humans will be exposed to cooking-related respiratory risk. Studies of cooking emissions have been conducted in both real-world and controlled environments[36]. In studies carried out in a controlled environments, cooking emissions are largely determined by the fuel type used and the ingredients being cooked[37, 38]. In real-world settings, emissions are also influenced by factors such as room and kitchen arrangement, building materials, outdoor/indoor air exchange, and cooking methodology[39].

Real-world emissions are further affected by kitchen layout, building ventilation, window opening, and air exchange with adjacent spaces[40]. For example, in a survey of Canadian homes, Sun et al. found that many occupants used ventilation inconsistently; fewer than two-thirds used any ventilation device during cooking episodes[40].

Particle emissions vary by cooking method (e.g. frying, roasting, grilling, boiling, steaming), ingredient composition, fuel types, temperatures, and the presence of ventilation and/or extraction fans. Frying was found to increase total particle concentration by a factor of 6-10 when compared to other cooking methods[41]. See and Balasubramaniam's controlled environment studies found that oil-based methods such as pan-frying, stir-frying, and deep-frying resulted in an increase in particles concentrations by a factor of 2-24 when compared to water-based methods such as steaming and boiling, with cooking temperature and food

amount and type being kept constant across the different cooking types[42]. Amongst these methods, deep frying produced the greatest particle concentrations. The difference in particle concentrations can be attributed to the high-temperature heating of cooking oil, which generates fatty acids[42]. The increased emission is attributed to high-temperature breakdown of lipids and volatilization of organic compounds[42].

Ventilation is among the most effective measures for reducing exposure to cooking emissions, but its efficacy is highly dependent on system design, location, and user behavior[40]. There are two main types of mechanical ventilation systems: vented and unvented. Vented range hoods extract air to outside the home, while unvented range hoods pull air through a filter before recirculating it into the home. Opening a window or door can also be a method of ventilation. In a study by Sun et al., it was found that two thirds of participants did not utilise any methods of ventilation while cooking[40]. In another by Nagda et al., only 38 percent of participants used the range hood frequently at dinner, 14 percent at lunch, and 19 percent at breakfast[43].

Routine cleaning activities such as vacuuming, mopping, dusting, or spraying of cleaning products can lead to resuspension of settled particles or the formation of secondary particles[44]. For example, the use of bleach or terpene-based cleaning agents may generate secondary aerosols; Wang et. al. observed particle formation from terpenes and bleach emissions, and Wong et. al. observed increases in chlorine particles after bleach cleaning[44, 45].

Building materials (and flame retardants contained within building materials) are another important source of particulates in indoor environments. A large number of chemicals found indoors are present due to paints, wood preservatives, varnishes, and solvents[46]. Berglund (1997) provides discussion of how indoor furnishings and building components serve as low-level emission sources[46]. As these materials deteriorate, emissions may increase. Additionally, human activities such as sitting on furniture, walking around, drawing curtains, etc. can also lead to the resuspension of particles into indoor air[47].

Changes in building design over the past few decades to improve energy efficiency have led to the construction of more airtight homes; widespread use of synthetic materials have reduced air leakage but increased the potential for pollutant accumulation indoors. While these changes have led to reduced energy prices and greater comfort, they have also resulted in environments where particulate matter is often more readily produced and built up to higher

concentrations. With the enhanced airtightness of residential buildings, indoor concentrations can exceed outdoor levels if internal sources are present[3, 30]. As such, changes in building design are not a source of indoor particulate matter per se, but nevertheless can result in elevated levels of indoor particulate matter as a consequence.

Cigarette smoking is a significant source of indoor particulate matter and pollution. The prevalence of cigarette smoking has decreased in recent decades; nevertheless, it remains a large contributor of adverse health effects associated with indoor environments[48]. More recently, vaping and e-cigarette aerosol emissions are emerging as indoor sources, but because device composition is highly variable and regulation is nascent, robust data remain scarce.

Personal care products and fragranced candles represent potential sources of fine particulate matter in indoor environments. Candle combustion releases soot and ultrafine particles, as well as compounds such as formaldehyde, acrolein, and naphthalene[49].

Additionally, many personal care items (e.g. perfumes, lotions, sunscreen) emit volatile organic compounds and monoterpenes, which may undergo rapid oxidation indoors and form secondary aerosols in the fine-particle range.[50]. Because occupants typically apply these products and light candles within the home environment, these behaviours contribute to overall indoor $PM_{2.5}$ exposure and should therefore be incorporated into source assessments in real-home settings.

In the UK specifically, key indoor sources consistently identified in literature include cooking, cleaning activities, smoking, and emissions from building materials and consumer products [3, 30, 51]. Cooking is widely reported as the dominant episodic source of fine and ultrafine particles, particularly during high-temperature processes such as frying, while cleaning and occupant activities contribute through both primary emissions and resuspension of settled dust. Outdoor air pollution also makes a significant contribution to indoor PM via infiltration and via soil adhering to footwear, then becoming resuspended into the air [52].

However, despite a relatively robust understanding of PM sources, there remains a notable scarcity of detailed measurements of PM chemical composition in UK residential settings [53]. Existing studies have often focused on mass concentrations ($PM_{2.5}$ and $PM_{10}$) rather than speciated composition. As such, the current state of the science in the UK is well-established source attribution but comparatively limited data on the detailed chemical composition of indoor particulate matter.

### 1.4.2 Indoor and Outdoor Air Exchange

It is important to distinguish between the sources of indoor particulate matter, because particles originating outdoors differ in concentration, composition, toxicity, and mitigation approaches compared to particles generated indoors[54]. Intrusion of outdoor pollutants is an important contributor to indoor air pollution; however, with the introduction of measures to reduce ambient pollution levels, indoor sources are becoming larger contributors to personal exposures[35].

Beko et. al. found that in a study of 60 non-smoking residents of Copenhagen, indoor activity accounted for 50 percent of the residents' daily personal exposure; amongst the indoor environments studied, residences comprised the biggest contribution to exposure and the associated mortality burden[55]. This underscores that even in areas with good outdoor air quality, indoor sources can be significant.

### 1.4.3 PAHs (Polycyclic Aromatic Hydrocarbons)

Polycyclic aromatic hydrocarbons (PAHs) are organic compounds composed of multiple fused aromatic rings, formed primarily by the incomplete combustion of carbon-containing materials, and are present in both indoor and outdoor environments[56, 57]. Indoors, key emission sources include cooking (especially frying or grilling), tobacco smoke, incense or candle burning, heating appliances, and infiltration from ambient air[56, 58]. Because PAHs span a range of volatilities, lower molecular-weight PAHs often partition to the gas phase, while higher molecular-weight PAHs adhere to particles, which affects their transport and deposition characteristics[56, 59].

Many PAHs, and especially heavier ones such as benzo[a]pyrene (BaP), have been recognized as carcinogenic in nature[56, 60]. Epidemiological and experimental studies have linked indoor PAH exposure to DNA adduct formation, oxidative stress, and increased lung cancer risk[60]. Some assessments suggest that indoor PAH inhalation risks in urban settings may exceed acceptable cancer risk thresholds (e.g. $>1 \times 10^{-6}$)[59].

Because PAHs are often co-emitted with particulate matter in combustion processes (e.g., cooking or smoking), combining PM measurements with PAH speciation strengthens source apportionment and toxicity assessments. In indoor air studies, BaP is frequently used as a marker to represent the degree of carcinogenicity of PAH mixtures[56]. Monitoring indoor PAHs is crucial for understanding chemical exposure and for informing public health policies

in residential settings.

### 1.4.4 Phthalates

Phthalates are ubiquitous in many consumer products, and are used to create flexibility and durability in plastics, electronics, flooring, toys, and furniture. In addition, they are also used as solvents and stabilisers in cosmetic and fragrance products[61]. Historically, dibutyl phthalate (DBP), dimethyl phthalate (DMP) and diethyl phthalate (DEP) were the most commonly used phthalates used for cosmetic purposes[62]. Based on a 2010 FDA survey of cosmetics, use of DBP and DMP has fallen dramatically, and DEP is the only phthalate that is still commonly used for cosmetic purposes[62].

While oral exposure via ingestion remains a primary route for many phthalates, inhalation exposure may contribute significantly, particularly for volatile or semi-volatile phthalates and in environments with poor ventilation or high emissions. Toddlers and infants may be especially vulnerable due to hand-to-mouth behaviors and higher breathing rates relative to body weight, and prenatal exposure is also a concern[61, 63].

## 1.5 Indoor Air Quality Measurement Techniques

Many techniques for sampling particulate matter indoors derive from those used outdoors, but without complications such as precipitation or large fluctuations in temperature and humidity[64, 65]. Nevertheless, indoor sampling introduces its own set of challenges. Instruments must be quieter, more compact, and less intrusive.

When measuring outdoors, particle mass and particle count are the two standard metrics[66]. Other key metrics include size distributions and chemical composition, as smaller particles can penetrate further into the lungs.

A diverse range of sensors and samplers exist for measuring particulate matter. Gravimetric methods are a widely used standard: filters are weighed before and after sampling, and the mass difference is used to estimate particulate mass[4]. Active sampling uses pumps to draw air through filters, often coupled with size-selective inlets such as cyclones or impactors to separate particle size classes[4]. Cascade impactors, which cascade through multiple stages of decreasing cutoffs before a final filter, allow mass measurements of discrete size fractions[67].

In contrast, passive samplers rely on diffusion, deposition, or other passive transport

mechanisms to collect particles. These are often favored in personal exposure or long-term deployments because they are quiet and do not require power supply[68]. However, they are more sensitive to ambient airflow, particle size, and deposition biases, and typically require longer sampling durations to accumulate measurable masses[69].

Optical methods estimate particulate concentration via light scattering or absorption. As particles pass through a laser or light beam, they scatter or absorb light, which is detected and converted to mass or particle number via calibration[66]. Optical sensors can provide real-time data and avoid issues like filter evaporation or bounce. However, they rely on assumptions about particle size, density, and refractive index, so they cannot measure true mass without correction[66].

Some researchers argue that particle number may better reflect health risk than particle mass, because it emphasizes the smaller particle fraction that reaches deep into the lungs[22]. Mass measurements, in contrast, are often dominated by larger particles or agglomerates. Optical sensor methods and particle counters are commonly used to obtain number concentrations[70]. Particle surface area is another metric for toxicity that is gaining in popularity, and has shown potential to be a stronger dose metric than particle mass to assess pulmonary inflammation caused by nanoparticles in particular[71].

In recent years, low-cost sensors have been investigated as a potential new avenue of indoor air quality monitoring. Low-cost sensors allow researchers reduce the effects of observational effect when investigating residential environments, as occupants are less likely to be disrupted, either by the researchers or by a larger, noisier sampler. In unoccupied environments, intrusion by researchers can introduce new contaminants and resuspend PM. Low-cost sensor deployment also allows for the use of a greater number of sensors, due to the decrease in cost per unit. Studies have shown that many low-cost sensors perform modestly well in laboratory or field comparisons, but their performance is often variable and dependent on calibration, humidity, particle type, and environmental conditions[72, 73]. For example, a one-year evaluation of three low-cost $PM_{2.5}$ monitors in a non-smoking residence found that while they tracked trends, absolute concentrations sometimes deviated significantly from reference instruments[74].

In the UK, DEFRA and other regulatory bodies employ a set of approved instruments that have been demonstrated to be equivalent to the European PM reference method. These instruments are expensive, large, and noisy, but allow for a high level of accuracy and preci-

sion. These methods are the tapered element oscillating microbalance (TEOM), a gravimetric method which depends upon the changing oscillating frequency of a microbalance with an air filter attached; beta ray attenuation (BAM), which uses the beta ray absorption of particles collected on a filter to measure particle mass; the FIDAS method, which utilises optical methods to count particles in varying size ranges, then extrapolates the data to estimate particle mass; and the Partisol, a gravimetric sampler which draws air through a size-selective inlet and sequentially size-selecting filters.[75, 76].

The choice of measurement technique and monitoring equipment will always be an attempt to balance accuracy, temporal resolution, intrusiveness, noise, and cost. In indoor air studies, combining gravimetric, optical, and number/size metrics often gives the most comprehensive view of particulate exposures, but this may not always be viable due to cost and/or space limitations.

## 1.6 Health Impacts

Indoor air pollution has been estimated to account for approximately 91.5 million disability-adjusted life years (DALYs) globally, with the heaviest burdens borne by low-income countries[77]. Exposure to particulate matter has been linked to various illnesses such as but not limited to cancer, pulmonary inflammation, cardiovascular disease, and coronary arteriosclerosis[30]. Long-term exposure (over years or decades) is associated with increased total, cardiovascular, and infant mortality. The strongest epidemiological associations have been found for $PM_{2.5}$, followed by $PM_{10}$[78, 79].

The pyramid of adverse effects from air pollution illustrates how health impacts are distributed in a population, and is shown in Figure 2

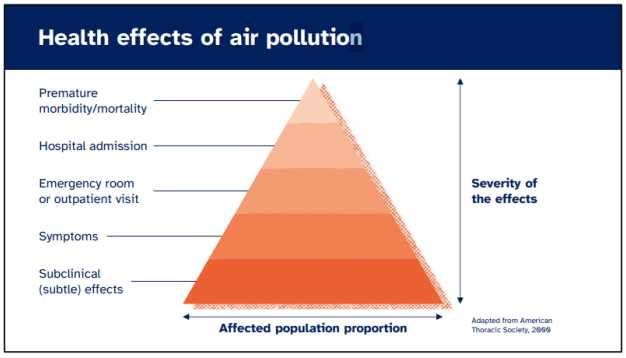



**Figure 2:** The pyramid of health effects associated with ambient air pollution. Source: World Health Organization. *Health effects of air pollution: a general overview*[80]

At the base are subtle health changes such as irritation of airways, reduced lung function, and inflammatory responses that affect many individuals in a population. Moving up the pyramid, the number of affected individuals shrinks but the severity of the effects increase, e.g. from exacerbation of asthma and cardiovascular symptoms to admission to hospital. At the apex of the pyramid are the rarest but most severe outcomes, including premature mortality. The pyramid shows how the human population sits on a continuum of risk due to exposure, from mild subclinical effects to serious illness and death.

$PM_{2.5}$ is considered of high importance due to its consistently reported associations with cardiovascular mortality in large cohort studies, as evidenced in the updated American Heart Association (AHA) statement on particulate air pollution and cardiovascular disease[26]. In particular, it was concluded that: exposure to particulate matter of diameter <2.5 um over a few hours to weeks has the potential to trigger cardiovascular related mortality as well as nonfatal events; exposure to $PM_{2.5}$ for longer periods of time (i.e. months or years) will increase these risks; life expectancy of those who are chronically exposed is decreased by several months to a few years; reductions in overall PM levels were associated with decreases in cardiovascular mortality; and credible biological mechanisms were identified that lend biological plausibility to the findings[26]. The Air Pollution and Health: A European Approach (APHEA and APHEA-2) found that PM in air was significantly associated with the number of deaths from all causes, as well as cardiovascular and respiratory mortality[81]. In all-cause and cardiovascular mortality, the effects were larger on the first and second days of an exposure event, but persisted for longer[81]. In contrast, for mortality, the lagged effects are more pronounced and long-lasting[81]. In addition, the International Agency for Research on Cancer (IARC) has recognised $PM_{2.5}$ as a Group 1 carcinogen[82].

Recent cohort studies reinforce these links. For example, a large national cohort in China reported elevated mortality risk from cardio-metabolic, respiratory, and cancer causes per incremental increase in long-term $PM_{2.5}$ exposure[83]. Similarly, a study from the United States found that a 10 $\mu g/m^3$ increase in average $PM_{2.5}$ was associated with about a 24% rise in cerebrovascular mortality, 23% increase in IHD mortality, and 8% increase in incident myocardial infarction[84].

The incidence of asthma and allergic respiratory illnesses has roughly doubled in developed countries over the past three decades. Bornehag et. al. have theorised that this increase is due to declining air quality in residential environments, due to high energy prices and energy saving campaigns causing individuals to reduce natural ventilation in their homes in an effort

to conserve heat[**Bornehag2005**].

Due to its small size, $PM_{2.5}$ has the ability to penetrate more deeply into lung tissue. Particulate matter deposited in the alveoli can cause local inflammation, which can in turn elicit a systemic inflammatory state[22]. These characteristics are believed to cause many of the downstream cardiovascular and metabolic health effects observed in epidemiological studies[22].

An individual's susceptibility to adverse health effects caused by particulate matter is reliant upon the concentration of the particulate matter in the air, the individual's personal sensitivity to that compound, the duration and frequency of their exposure, and the individual's personal state of health[85]. Children in particular often experience adverse health effects at lower concentrations due to their smaller stature, though the elderly and those with chronic illness are also at higher risk. Gender, atopy, and psychological factors have also been found to affect susceptibility[27]. In a follow-up analysis carried out on the Adventist Health Pollution Study (AHSMOG), it was found that fatal coronary heart disease was significantly associated with $PM_{2.5}$ exposure among women but not men [86]. Along with the results found in the Women's Health Initiative Observational Study and the Nurses' Health Study, this suggests that women are potentially at special risk due to exposure to particulate matter[26, 86]. However, further study is needed to determine whether this is a recurring pattern.

Studies of particle deposition characteristics have also shown that pulmonary deposition of inhaled particles may differ by gender. Overall deposition between men and women is relatively comparable, or slightly higher in women depending on breathing pattern; however, deposition was found be more localised within the lung in women[87]. Kim and Hu (1998) suggest that this comparative increase in regional deposition may result in a greater health risk in women exposed to PM[88].

A less quantifiable health outcome linked to indoor environments is Sick Building Syndrome (SBS). Symptoms include mucosal irritation, headache, fatigue, and respiratory discomfort, which tend to worsen with prolonged occupancy[6]. Epidemiological reviews suggest that women report SBS symptoms more frequently than men in identical indoor settings[89]. Building-Related Illness (BRI), by contrast, refers to specific pathologies (e.g. hypersensitivity pneumonitis, Legionnaire's disease) tied to known indoor exposures (e.g. mold, VOCs, formaldehyde)[89].

Although exposure to PM has been strongly linked to poor health outcomes, there has more recently been a push to identify the specific compounds that are more likely to be a threat to public health, thereby giving regulators more specific sources to target. A review by Stanek et. al. found that while there was some evidence for links between certain ambient PM components and specific health outcomes, studies differed in their conclusions as to which groups of PM sources lead to which health outcomes[90]. As more studies investigate these links, it is possible that there will be more agreement in the future within the scientific and health communities.

### 1.6.1 Vulnerable Populations

Certain groups are more susceptible to the adverse health effects associated with particulate matter exposure, due to physiological, developmental, or behavioural factors[17]. Among these, children, the elderly, and individuals with pre-existing health conditions represent the most at-risk populations[17].

Children experience greater vulnerability to airborne pollutants due to the ongoing development of their respiratory and immune systems. They also have higher ventilation rates relative to their body weight and spend more time indoors, increasing their inhalation dose of fine and ultrafine particles[91, 92]. Epidemiological studies have linked chronic exposure to $PM_{2.5}$ in children to reduced lung function growth, increased respiratory infections, and heightened risk of developing asthma[91]. Prenatal exposure to fine particles has also been associated with adverse birth outcomes, including low birth weight and preterm birth[93]. Black carbon particles have been found on the fetal side of human placentas, suggesting that ambient particulates could bebeing transported to the fetus; this may provide evidence for the deleterious effects of exposure to air pollution from early life onwards[94].

Older adults face increased susceptibility due to age-related declines in pulmonary clearance and cardiovascular resilience[26, 95]. Exposure to particulate matter can exacerbate existing conditions such as chronic obstructive pulmonary disease (COPD), ischemic heart disease, and hypertension, leading to increased hospital admissions and mortality rates among this demographic[96]. Systemic inflammation and oxidative stress are hypothesised to be key mechanisms linking PM exposure to cardiovascular morbidity in older populations[97].

Individuals with pre-existing respiratory or cardiovascular conditions are particularly sensitive to even small increases in particulate matter concentrations[26]. Short-term exposure events, such as those caused by cooking or heating, can trigger acute respiratory distress,

chest tightness, or arrhythmia in these individuals[26, 98]. Similarly, people with metabolic disorders, including diabetes, have demonstrated heightened inflammatory responses and impaired vascular function following exposure to $PM_{2.5}$[99].

Socioeconomic status also plays a crucial role in exposure inequality. Lower-income households are often located near major roads or industrial areas and may lack adequate ventilation or filtration systems, compounding health disparities[100, 101]. Consequently, environmental justice perspectives are increasingly being incorporated into air quality research and policy development to address disproportionate exposure burdens among vulnerable and marginalised populations[102].

## 1.7 Measuring Exposure

The measurement of human exposure to airborne pollutants is a critical step in understanding associated health risks. In indoor environments, exposure is often quantified through the concentration of particulate matter and the time individuals spend in those environments. However, the relationship between concentration and exposure is not always straightforward, as human behaviour, ventilation, and building characteristics influence both pollutant accumulation and personal contact rates with contaminated air[103].

Exposure assessments typically combine measurements of ambient concentrations with estimates of inhalation rates, occupancy patterns, and microenvironmental variability[103]. Indoor monitoring campaigns often use time-resolved $PM_{2.5}$ data to identify peaks corresponding to activities such as cooking, cleaning, or smoking, while background levels are recorded to represent quiescent conditions[103]. Integrating these measurements over time provides a more complete estimate of personal exposure, particularly when combined with information about occupant movement and duration of stay within each environment.

In residential and occupational settings, exposure can be expressed as an average daily dose or cumulative lifetime dose. These calculations combine concentration data with physiological factors such as breathing rate, body weight, and duration of exposure. For example, the U.S. Environmental Protection Agency (EPA) provides standard exposure factors that are widely used in risk assessments[103]. Such data allow for comparisons across demographic groups, highlighting vulnerable populations who may experience greater exposures due to age, occupation, or health status.

In the UK, COMEAP provides independent advice to the government on the health

impacts of air pollution. COMEAP has highlighted that both short and long term exposure to particulate matter, particularly $PM_{2.5}$, are associated with increased mortality and morbidity, and emphasises that there is no clear safe threshold for exposure [14].

In addition, the Air Quality Expert Group (AQEG) have recognised that indoor environments represent a major component of total exposure, particularly in the UK where housing and ventilation practices differ from those in North America and Asia. UK homes are often more airtight due to energy efficiency measures, which can reduce infiltration of outdoor air but also cause the accumulation of indoor generated pollutants [13]. As such, assumptions from other regions may not fully capture the circumstances of indoor air pollution in UK residential environments.

Beyond total PM concentration, source apportionment and chemical characterization provide insights into which specific compounds contribute most significantly to human exposure. Certain compounds, such as phthalates or PAHs, are associated with higher toxicological concern[15, 104]. Their presence in indoor particulate matter may be small compared to the concentrations of other compounds, but they can dominate health risk assessments due to their carcinogenic or endocrine-disrupting properties[15, 104].

### 1.7.1 Measuring the Health Risks of PAH and Phthalate Exposure

Polycyclic aromatic hydrocarbons (PAHs) and phthalates are among the most studied semi-volatile organic compounds (SVOCs) in indoor environments, due to their ubiquity and toxicological significance. Both compound groups exist in the gas and particle phases, and their distribution between these phases depends on temperature, particle composition, and volatility[15]. In indoor settings, they are typically measured from particulate matter samples collected on filters, often using low- or medium-volume air samplers designed for integrated sampling over several hours to days.

Toxic equivalency factors (TEFs) are a tool used to estimate carcinogenic potential of exposures. These factors, first proposed by Nisbet and LaGoy in 1982, express the toxicity of each PAH relative to benzo[a]pyrene (BaP), which serves as the reference compound due to its well-established carcinogenicity[104]. The resulting BaP-equivalent concentration ($BaP_{eq}$) is a metric that accounts for differences in toxic potency across multiple PAH species, allowing comparison between samples and between environments[104].

In measuring phthalate exposure, bis (2-ethylhexyl) phthalate (DEHP) is often used as a

marker compound because of its widespread presence and relatively high toxicological relevance[105]. Its risk assessment commonly relies on the use of reference doses or cancer slope factors, which translate exposure estimates into quantitative health risk metrics. The DEHP slope factor of 0.014 (mg $kg^{-1}$ $d^{-1})^{-1}$, for instance, is often used to estimate incremental cancer risk in exposure studies[105].

While absolute concentrations provide an indication of pollutant abundance, toxicity-weighted measures such as $BaP_{eq}$ for PAHs and cancer slope factor–based doses for phthalates offer a more direct link between exposure and health risk.

## 1.8 Comprehensive Two-Dimensional Gas Chromatography Coupled with Time-of-Flight Mass Spectrometry

Comprehensive two-dimensional gas chromatography coupled with time-of-flight mass spectrometry (GC×GC–ToF-MS) is an analytical technique designed to improve the separation and identification of complex mixtures of organic compounds. Conventional one-dimensional gas chromatography (1D-GC) separates compounds using a single capillary column, where analytes are separated based on their volatility and affinity with the stationary phase[106]. While this approach is effective for simple mixtures, it can struggle when analysing environmental samples that contain hundreds or thousands of compounds with similar structural properties[107]. Co-elution of compounds in 1D-GC can make it difficult to resolve individual analytes [108].

GC×GC addresses this limitation by introducing a second separation step that operates using a column with different chemical selectivity. In this system, analytes first pass through the primary column, where they are separated based on volatility or polarity depending on the stationary phase used. Fractions of the effluent are then periodically trapped and re-injected into a second, shorter column via a modulator [106, 109]. This process occurs continuously throughout the chromatographic run, producing a two-dimensional separation in which compounds are separated according to two distinct chemical properties[106]. As a result, compounds that may co-elute in the first column may be separated in the second dimension, significantly increasing chromatographic resolution. In the setup used throughout this thesis, the primary column separated analytes based on their volatility, and the second based on their polarity.

The modulation process is a key component of GC×GC systems. During modulation,

small portions of the effluent from the first column are transferred into the second column in narrow pulses via a stop-ang-go system caused by hot and cold jets of air hitting the column and controlling the rate at which the effluent reaches the second column [109]. This results in a series of short, highly concentrated peaks entering the second column, which improves peak sharpness and enhances sensitivity [106]. The result is a chromatogram where compounds are organised according to two retention dimensions, visualised as a two-dimensional contour plot.

Detection in this system is achieved using time-of-flight mass spectrometry (ToF-MS). ToF-MS instruments measure the mass-to-charge ratio of ions based on the time it takes for them to travel through a flight tube after ionisation. Because all ions are detected simultaneously, ToF-MS can achieve rapid spectral acquisition rates [110]. The fast acquisition speed of ToF-MS allows full mass spectra to be collected across narrow peaks, allowing for accurate compound identification[110].

The combination of comprehensive two-dimensional separation and high-speed mass spectrometric detection makes GC×GC–ToF-MS particularly well suited for the analysis of complex environmental samples such as those collected for this thesis. Indoor air contains a wide range of organic compounds; GC×GC–ToF-MS enables improved resolution of structurally similar compounds such as PAHs and their derivatives, which may overlap in conventional GC analyses. The increased peak capacity and enhanced sensitivity of this technique allow both targeted and non-targeted analyses to be performed within the same dataset, allowing for the identification and quantitation of targeted compounds as well as the exploration of untargeted components of indoor particulate matter.

## 1.9 Literature Search Strategy

The literature review was conducted using a flexible and exploratory search strategy, whereby initial searches were carried out using Google Scholar, employing combinations of key terms including “indoor particulate matter”, “$PM_{2.5}$”, “indoor air quality”, “PAHs in indoor environments”, “phthalates in indoor environments”, and “cooking emissions”.

Search results were screened based on title and abstract to identify studies relevant to indoor environments, particulate matter composition, and human exposure. Particular emphasis was placed on articles reporting health-related assessments. Where relevant papers were identified, their reference lists and citing articles were also examined (backward and forward citation tracking) to identify additional key studies.

Studies were generally included if they investigated indoor particulate matter or indoor air quality in residential or comparable environments, investigated or quantified health risks due to exposure to an indoor pollutant, and/or provided information regarding specific compound classes and their toxicity.

Studies were excluded if they focused on volatile compounds as they are not within the scope of this project, or if they were not accessible through the University of York's journal subscriptions.

## 1.10 Conclusions and Project Aims

Although there have been rising numbers of studies focusing on the generation, distribution, and longevity of indoor particulate matter, it is apparent that it is still a field which needs to be further explored. As most academic focus thus far has been on outdoor environments, many of the existing monitoring methods will need to be modified for use indoors.

As there is still debate regarding the best metric for estimating toxicity and risk to human health, further investigation is needed regarding the efficacy of measuring particle mass, particle number, and particle surface area. It is possible that these factors, in addition to composition, combine with each other to form unique toxicity profiles.

Due to the inherent variation present across indoor environments depending on occupant number, behaviour, location, and furnishings, there will be significant challenges to building understanding of particle composition in indoor environments. Many researchers are focusing on laboratory cooking experiments, as cooking has been found to be the most significant source of indoor particulate matter; however, these experiments are difficult to compare due to different approaches to study design and protocols, and due to the varied nature of cuisines across cultures. Particle emissions from cleaning supplies are similarly varied, and due to their relatively low concentrations in indoor environments, are challenging to quantify and characterise. With advances in technology and research, it is likely that in the years to come, our knowledge of indoor particulate matter and its risks to human health will expand greatly.

The aim of this study is to investigate the composition, concentrations, and potential health risks of indoor particulate matter in real home environments, with particular emphasis on compounds with known health risks such as PAHs and phthalates.

This project seeks to:

- Quantify concentrations of particulate matter in residential indoor environments under real-home conditions.
- Characterise the chemical composition of collected particles to aid source identification.
- Evaluate the toxicological significance of observed concentrations of compounds with known health effects.
- Carry out an exploratory non-targeted analysis of cooking emissions in a controlled environment. This was initially outside the scope of this study, but due to a global helium shortage causing delays in the original study, experiments were added to compare emissions across different popular recipes and between vegetarian and non-vegetarian ingredients.

Through these aims, the project intends to contribute to the broader understanding of human exposure to indoor particulate matter and to identify key factors influencing variation in exposure and health risks. This study is novel in combining detailed chemical characterisation with real-world residential sampling across multiple homes, providing an integrated assessment of indoor particulate matter that captures both environmental variability and human exposure, and using an interdisciplinary approach encompassing environmental monitoring, chemical analysis, and toxicological relevance.

By identifying key sources and measuring exposure to harmful compounds, this study provides evidence that can inform future research and the development of strategies to reduce indoor air pollution and its associated health risks.

# Chapter 2: Development of a method to collect and characterise particulate matter in occupied indoor homes

# 2 Development of a method to collect and characterise particulate matter in occupied indoor homes

## 2.1 Background

The INGENIOUS (UnderstandING the sourcEs, transformatioNs and fates of IndOor air pollUtantS) project is a multi-disciplinary collaboration between several universities in the UK and Born in Bradford (BiB), which conducts a cohort study following the health of children and families over a period of 30 years. INGENIOUS aims to broaden understanding of the composition and concentration of indoor air pollutants vary and how this exposure affects people in their homes. It also aims to investigate these unknowns and deliver useful interventions to limit exposure. Its project structure is outlined in Figure 3

The INGENIOUS team seeks to quantify and identify:

- The composition and concentrations of air pollutants within indoor spaces, such as those from cooking and cleaning.
- How air pollutants react chemically and transform over time, including when different air pollutants mix.
- How air pollutants from indoor air sources affect outdoor air quality and vice versa.
- How different household behaviours affect the production of and exposure to air pollutants, and how this influences health outcomes and inequalities.
- Which behaviour change interventions are most effective at reducing exposure to indoor air pollution.
- Which recommendations should be taken forward as policy solutions.

[111]

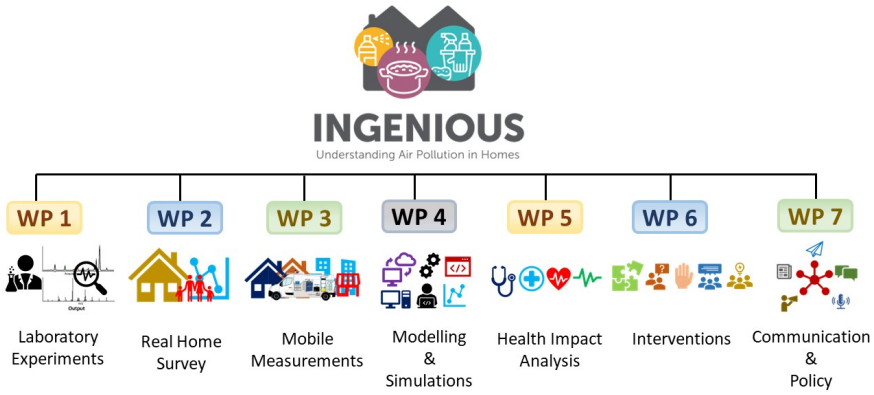



**Figure 3:** Structure of the INGENIOUS project

Work Package 2 of the wider INGENIOUS project was focused on collecting air quality data from real-home environments. This was done in several phases, whereby several different samplers were introduced over a two-week period. This project specifically was set to come in at the end of the overall sampling period, and collected samples over the last 72 hours in participants' homes. Figure 4 shows the deployment breakdown for the different samplers across WP2 homes. The focus of this thesis is the tip of the pyramid, where the aim was to obtain speciated particulate matter data.

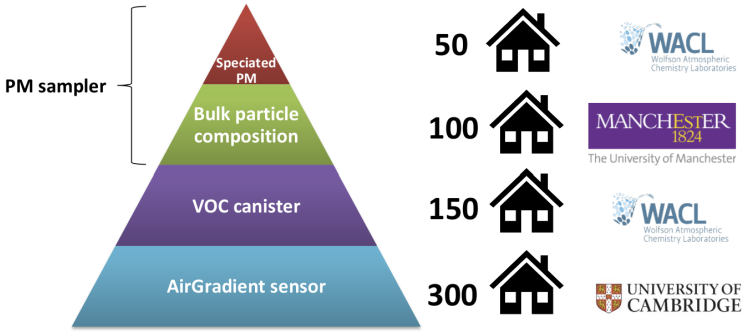



**Figure 4:** Number of houses for each sampler type

## 2.2 Sampler selection and modification

Because the sampling would take place in an indoor, residential environment, there were three major areas of concern that needed to be considered in the selection of an appropriate PM sampling instrument:

- Participant safety: Was it safe to operate over an extended period of time? Was it stable, and what was the potential for it to cause physical harm to a participant?
- Participant comfort: Will the noise from the instrument be overwhelming or irritating to participants? If so, this could cause participants to change their behaviour, e.g. avoiding the kitchen or its proximal areas, leaving the house for prolonged period of time, etc., which could result in samples that are not representative of the household. It could also result in the sampler being turned off prematurely. In addition, there were concerns about the size of the instrument, as kitchen counter top space was likely to be limited, and the cost of the instrument to run given the cost-of-living crisis that was sweeping the UK at the time of the experiment.
- Reliability of the sampler: Will the sampler stay on for the entire 72-hour period, and will it maintain its set flow rate throughout the sampling period? Would the instrument overheat or catch fire?

Before indoor sampling was allowed to commence, NHS ethics approval for the project needed to be obtained, as the project involved human subjects and access to NHS medical records. This meant that the sampler and procedure had to meet high standards for the safety and comfort of the participants. As a consequence, the decision was made to select an industry-manufactured sampler as opposed to one created in-house, as it would have undergone extensive safety testing as part of the product development process.

In summary, what was needed was:

- A sampler that could be relied upon to keep to a set flow rate for 72 hours. The flow rate required would vary from instrument to instrument depending on its cutpoint, so this would be determined after the instrument was selected. The 72-hour sampling time was pre-determined by the wider INGENIOUS project.
- The selected sampler would use PTFE (polytetrafluoroethylene) and SPE (solid-phase extraction) filters, as outlined in the method developed by Stewart et. al. [112].

### 2.2.1 The Microvol 1100

Initially, the Microvol 1100 (ACOEM) was selected as the sampler to use for this project as it was advertised as a low-flow sampler which was designed for both indoor and outdoor sampling of particulate matter. It was also promoted as having low energy consumption and

quiet operation. Weighing 3.75 kg and with dimensions of 30 x 17 x 17 cm, it was determined to be a good weight and size for deployment purposes [113].

**Figure 5:** The Microvol 1100

However, as testing commenced with this sampler, it became clear that there were several factors which would make the Microvol 1100 very difficult to use for the purposes of the study. The device was fairly unstable and easily toppled. The filter holder was located near the top of the sampler, and the filter holder component was easily detachable from the rest of the device. It was thought that the shape and design were not particularly rugged and any attention from children or pets in the household could cause damage to the sampling head, and its aforementioned instability would have posed a potential safety hazard to participants. In addition, the buttons which controlled the settings and operation of the device were easily accessible and there was no way to lock the device; if participants accidentally or purposely pressed any of the buttons, operation would have been compromised. A cage or cover would have needed to be constructed to cover the buttons and prevent tampering.

Despite being designed for indoor sampling, the device was still too loud to be placed within participants' homes for the planned period of time. When operated in a laboratory environment, its noise emissions were tolerable but in a series of tests conducted in a quiet,

residential environment, the sound was intolerable to residents of the domiciles. These tests were carried out by members of the supervisory team, other PhD students, and the author of this thesis–samplers were to collect samples over the weekend, and all samplers were turned off within a few hours of operation. As such, a soundproofing enclosure was necessary to reduce the overall noise emissions. The shape of the Microvol 1100 meant that the soundproofing enclosure would be complex to produce. Additionally, the sampler caused significant vibrations on the surface it was placed on. Budgetary constraints limited the amount that could be spent on custom enclosures. As such, it was determined that other samplers would be explored.

### 2.2.2 The Minivol TAS

The Minivol Tactical Air Sampler (TAS) (AirMetrics) is a low-flow pump designed for both outdoor and indoor particulate sampling, with programmable flow rates ranging from 1-10 L/min. It is shown in Figure 6a. It can operate from both AC and DC power sources; when using DC power, it can draw from an internal battery pack which holds roughly 24 hours' worth of power. It records the total time it has ever been in operation, in hours, with an Elapsed Time Totalizer, pictured in Figure 6


**(a)** The Minivol TAS

**(b)** The interior and control panel of the Minivol TAS

**Figure 6:** The interior and exterior of the Minivol TAS

It was both heavier (4.5 kg) and bulkier (25.4 x 53.3 x 17.8 cm) than the the Microvol 1100, but much more stable and robust [114]. Most significantly, it produced noise at higher levels than the Microvol. However, its rectangular shape was deemed more easily built around for the construction of a soundproofing enclosure. The sampler also came with an easy-to-use transportable case that made deployment of the instrument to homes much simpler. It was determined that the Minovol TAS would be the sampler of choice for the real-home sampling of $PM_{2.5}$ for the INGENIOUS project; following a testing phase, five additional devices were purchased for a total of six samplers to be deployed semi-simultaneously.

### 2.2.3 Soundproofing the Sampler

It was determined that custom soundproofing enclosures would need to be built for the Minivol TAS to make the noise levels emitted bearable to participants. A Class II sound level meter which complied with IEC 61772-1 was purchased and used to measure the sound emitted from the instrument. At point-blank range, the sound level was around 60 dBA (decibels, A-weighted), which is equivalent to the sound levels produced by normal conversation

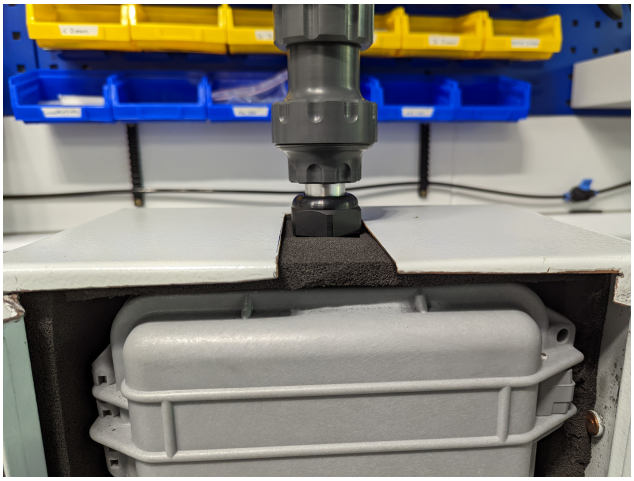
from 1 metre away.

Electrical housing boxes (Schneider Electric NSYS3D3320P, 300 mm, 300 mm, 200 mm, IP66) were purchased to form the base of the soundproofing enclosure. The size of the boxes were chosen based on the idea that while soundproofing was necessary, it was also vital that the size of the box would be kept minimal to conserve space in small kitchens. Cutouts were made by the Chemistry Department Machine Shop to accommodate the filter holder and inlet of the device. Soundproofing foams of several thicknesses and composition were purchased, as well as a pad of anti-vibration foam. Different combinations and placements of the foams were used to line the walls, floor, and door of the enclosure. Between each version of placements, sound levels were measured.

It was eventually determined that the Dodo Soundstopper v2 foam by itself was the best choice to reduce noise while leaving enough room for the sampler within the soundproofing enclosure. This soundproofing foam is comprised of two layers of sound absorbing foam with a layer of soundproofing mass coupled vinyl between them, and was designed for use in vehicles and around pumps or generators. Of the foams purchased, it was also the thinnest and heaviest.

The foams were glued onto the walls and floor of the electric box. The door was not able to be insulated due to lack of space; the door could not close with a layer of insulation glued to it. Figure 7 shows how the sampler sits within the soundproofing enclosure.


**(a)** The Minivol TAS, placed inside the soundproofing enclosure



**(b)** The opening in the top of the enclosure, showing where an additional layer of foam has been placed to reduce noise escaping

**Figure 7:** The soundproofing enclosure

As seen above in 7b, a layer of foam had to be placed under the cutout made to accommodate the sampler inlet, as the cutouts allowed a moderate amount of sound to escape. At

the end of the soundproofing enclosure building process, the noise levels emitted at point-blank range had decreased from 60 dBA to 50 dBA, a ten-fold reduction. 50 dBA is roughly equivalent to the sound of a refrigerator or heavy rainfall, while 60 dBA is roughly equivalent to the sound level of a conversation.

Given the budgetary constraints and the placement of the sampler within the kitchen, this was deemed acceptable to move forward with this prototype.

However, two major concessions were made to the original sampling strategy: due to the noise produced by the instrument when two types of filters (polytetrafluoroethylene (PTFE) above and SPE (solid phase extraction) below) were present in the filter holder, the decision was made to eliminate the SPE filter. This meant that the focus of the PM collection moved from particulate matter and semi-volatile compounds to particulate matter only. In addition, the manual for the Minivol TAS states that to ensure a cutpoint of particles 2.5 microns or less in diameter, a flow rate of 5 L/min must be maintained. This was not achievable given the sound levels emitted from the sampling device (around 60 dBA with both filters installed) and time and budgetary constraints; as a result, the flow rate was dropped to 3 L/min, with a cutpoint of 3.2 microns. The equation for the calculation is shown in Equation 1. As PM2.5 and PM3.2 have similar health effects based on their ability to intrude further into the lungs, this was deemed acceptable and preferable to the participants turning off the instrument or changing their behaviour.

$$C_{act} = \sqrt{\frac{Q_{design}}{Q_{act}} \times C_{design}}$$

Where:
$C_{act}$ =Actual cutpoint at set flow
$C_{design}$ =Cutpoint at designed flow rate
$Q_{design}$ =Design flow rate (5 L/min)
$Q_{act}$ =Actual sampler flow rate

[H] (Equation 1)

At a community engagement event prior to the deployment of the samplers, members of the Bradford community were shown an early version of the soundproofing enclosure with the sampler in operation. At this event, they were asked for their opinions on improvement and whether they personally would agree to have the sampler in their homes for 72 hours. During this event, information was gathered regarding potential barriers to their participation in the study. Noise and cost were determined to be the biggest possible barriers to participation. Several children at this event expressed how "boring" they thought the sampler looked; the smiley face scientist was later added to confer a warmer and friendlier aspect to the sampler

housing. Following this engagement event, further changes were made to the enclosure in order to achieve the 50 dBA noise levels of the final version of the enclosure. The final version of the soundproofing enclosure is shown in Figure 8.

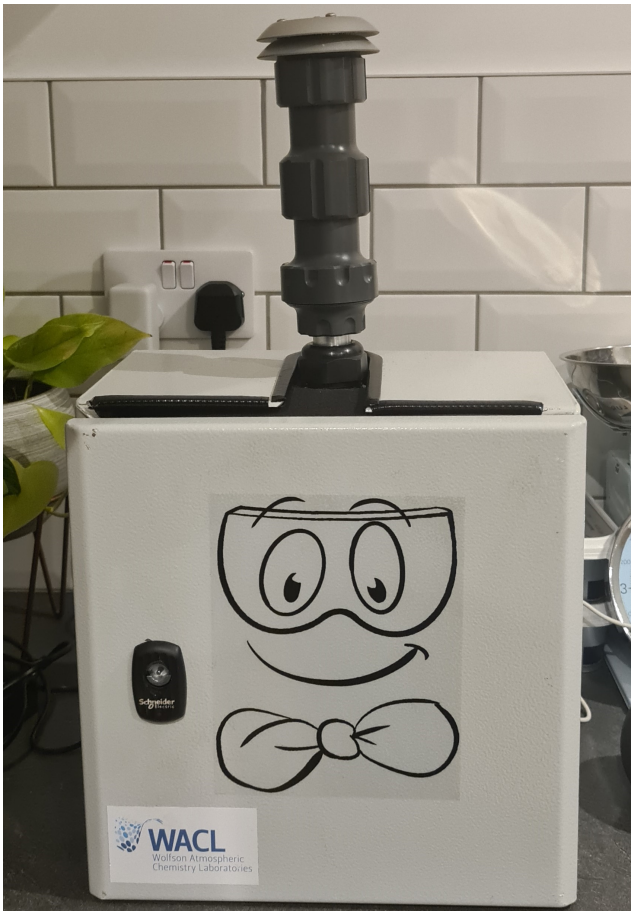



**Figure 8:** The final product

The noise of the sampler within its housing was deemed to be acceptable, and the cost to run the sampler for the duration of the sampling procedure was found to be only £0.06 (this value was based on readings from an energy usage meter, multiplied by the hours run and the cost of electricity at the time).

## 2.3 Filter selection

Initially, the aim was to target both semi-volatile gases using a solid phase extraction disk, combined with a PTFE filter to collect the particulate matter using a previously established methodology in Stewart et al. However, due to the noise produced by the sampler when trying to draw air through the two filters, the SPE filter was eliminated. The PTFE filters

initially purchased (Whatman, $PM_{2.5}$ PTFE Membrane Filter, 46.2 mm with support ring, 7592-104) were chosen due to their being specifically designed for $PM_{2.5}$ sampling.

When sampling commenced, it became clear that there was some contamination present on the filters, seen in Figure 9. At concentrations of collected material commonly seen when carrying out extended sampling outdoors, this would not matter, as the peak signals from other compounds would have been stronger. However, due to the low flow rates used in indoor sampling, the contamination overwhelmed the chromatographic plots. As such, sampling halted for a while to run some tests in order to achieve a cleaner filter, whether through washing, pre-baking, or simply purchasing a different brand/model of filter. It was found that the filters purchased varied greatly in cleanliness, which was why this contamination had not been caught earlier on.

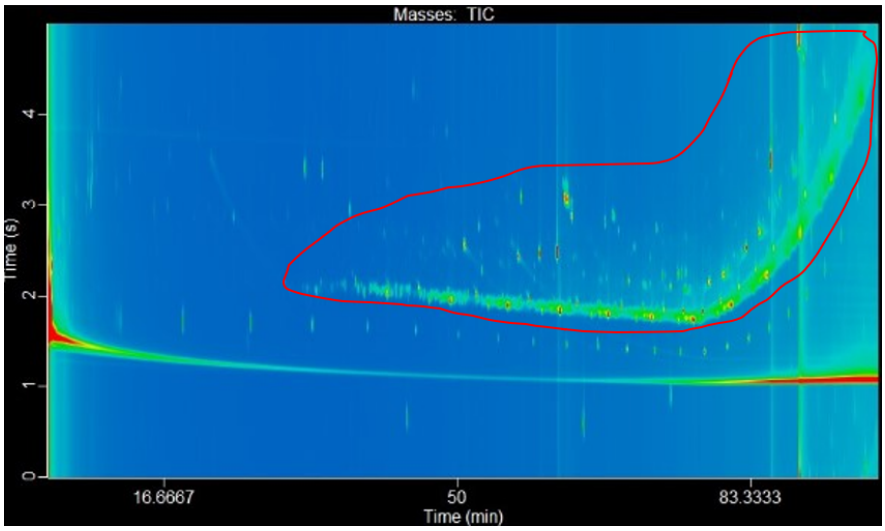
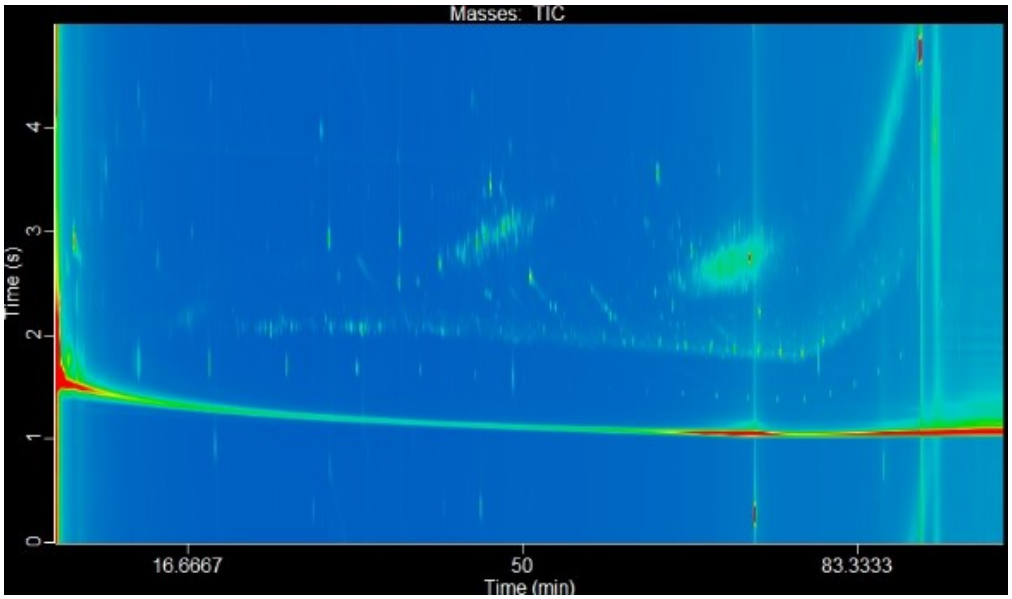


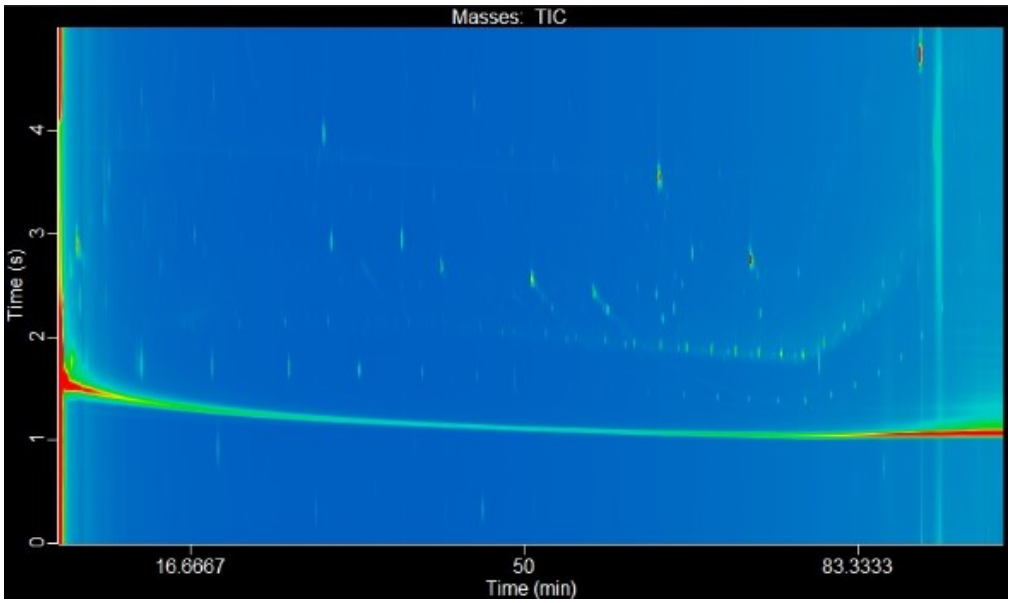

**Figure 9:** Old filter blank. The red circled section shows the areas of excessive contamination, where some target analytes were getting lost. In addition, this contamination prevented the untargeted analysis.

In consultation with INGENIOUS colleagues at the University of Manchester, it was found that they were pre-baking their filters at 200°C for 24 hours. After a series of tests (comparing filters straight out of package, washed with methanol and acetone, and pre-baked) with 3 different brands of filters (Whatman, Fisher Scientific, Mitex), it was determined that the Mitex filters post-pre-baking were the cleanest of all available options. As such, a new brand of filters were purchased, and pre-baked, after which sampling recommenced. Specifically, the new brand and model of filters used were the Mitex Membrane Filter (5.0 $\mu$g pore size, hydrophobic PTFE, 47 mm, LSWP04700). Figures 10a and 10b show the Mitex filters before

and after pre-baking respectively.




**(a)** Mitex filter, not pre-baked




**(b)** Mitex filter, pre-baked. Contamination is still rpesent, but greatly reduced.

**Figure 10:** 2D plots comparing the cleanliness of Mitex filters in their original state and post pre-baking

## 2.4 The Sampling Process

### 2.4.1 Sampling Activities

The decision was made to carry out the sampling in participants' kitchens, as cooking activities produce the majority of indoor particulate matter. The sampler was placed on kitchen counter tops whenever possible, and on a high stool when this was not possible. The timeline for the deployment of all WP2 samplers is shown in Figure 34; the Minivol TAS in its enclosure was deployed on Day 11 of 14.

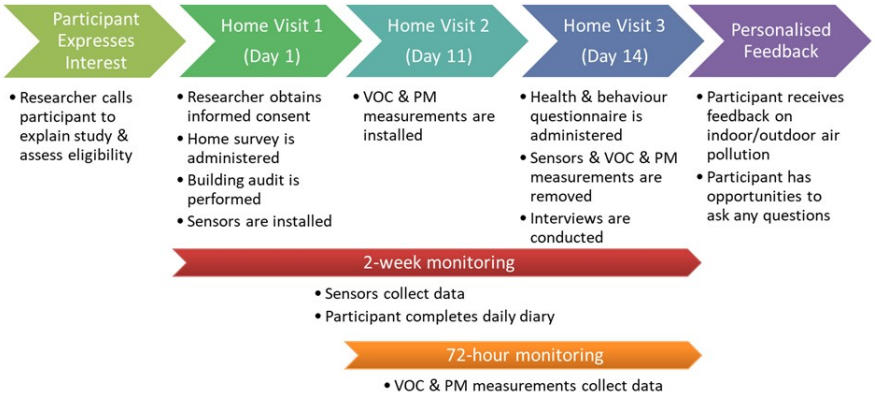



**Figure 11:** WP2 timeline

The physical sampling was carried out by research assistants at BiB, who would visit participants' homes at various intervals during the sampling period. A Standard Operating Procedure (SOP) was written by me for the research assistants at BIB, detailing instructions for installing filters, setting up and removing the samplers, and storing the filters until they could be collected and brought back to the lab. After sample collection, the filters were placed in pre-baked foil pockets (pre-baked at 550 degrees Celsius for 5 hours), individually stored in sealable plastic bags, and frozen in the Bradford Royal Infirmary's labs. This SOP is outlined in the section below. Training sessions for the research assistants were held in Bradford; the sampler and soundproofing enclosure were brought into the training facility, and set-up and removal were completed several times by each research assistant. The start and end dates and times were recorded, as was the "Total Elapsed Time" on the sampling instrument itself, which detailed the total amount of time the sampler had ever been on. The difference between the two start and end times was compared to ensure that the sample had indeed been on and running for the entire sampling period.

Every 4-6 weeks, Yunqi Shao (post-doctoral research fellow, University of Manchester) and I travelled to Bradford to collect filters and conduct maintenance activities on the samplers.

This entailed disassembling the filter holder and impactor system and reapplying vacuum grease to the impactor plates. Every 3 months, the impactor system was disassembled and washed with soap and warm water, as per manufacturer recommendations. Filter blanks (new filter, placed inside non-operating sampler for 10 minutes) were collected at these times. The samples were transported in freezer bags with built-in ice packs, and placed in the laboratory freezer at the University (at -20 °C) prior to analysis.

## 2.5 Standard Operating Procedure

The Standard Operating Procedure was developed as a reference material for the BiB community team to have on hand and refer to when deploying and collecting samplers and filters, and was written to be as clear and easy-to-use as possible. This ensured uniformity of procedure across all homes sampled.

### 2.5.1 Setting up filters

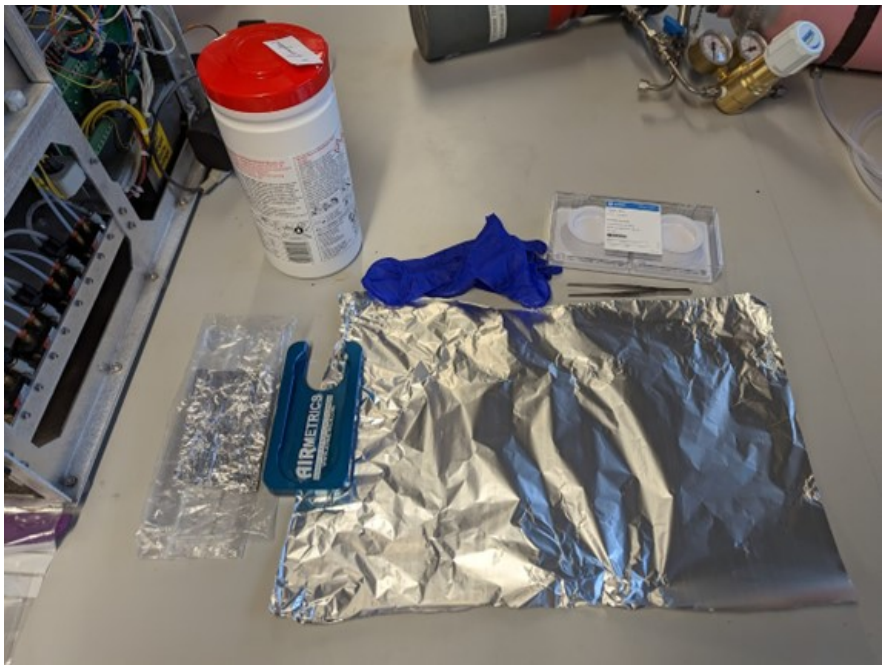



**Figure 12:** All the items needed for filter setup

1. Put on protective gloves and set down a sheet of aluminium foil to use as a work surface.
2. Wipe down foil and tweezers with an alcohol wipe.

3. Open filter case using the filter cassette separator and wipe with alcohol wipe. To use the casette separator, place the filter holder on the left side, then slide to the right.

**Figure 13:** Casette separator

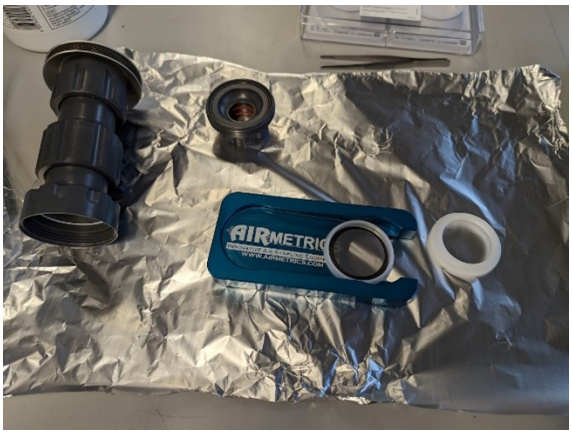


**Figure 14:** Casette separator in use

4. Using tweezers to hold the outside plastic ring on the filter, transfer a filter to the holder, keeping the serial number facing up. These filters have a tendency to stick together, so make sure that you only get one.

**Figure 15:** Placing a filter into the casette

**Figure 16:** Closed and loaded casette

5. Close filter holder

6. Place filter holder in the impactor base, and screw the base onto the whole impactor. The metal part of the filter holder should be on the bottom, so that the filter is exposed.

**Figure 17:** Casette and filter placed in the casette holder

**Figure 18:** Assembled impactor tower

7. Attach the impactor to the sampler.

**Figure 19:** Attaching the impactor tower to the sampler

### 2.5.2 Setting up sampler

1. Open the enclosure with a plastic key

**Figure 20:** Enclosure key

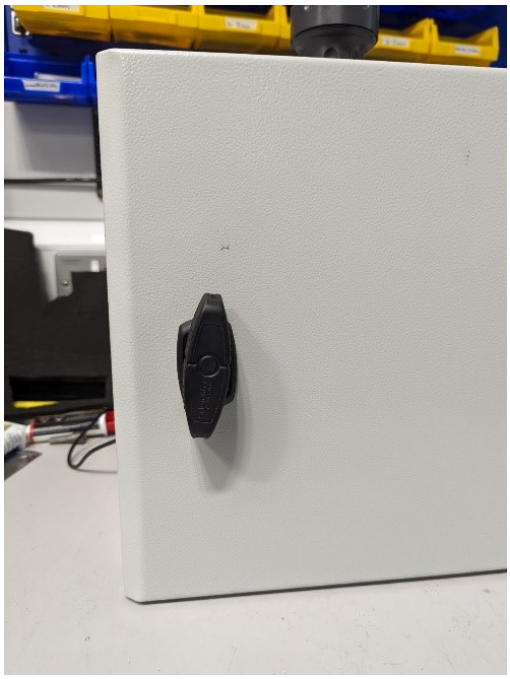


**Figure 21:** Enclosure key in use

2. Feed power cable through the opening on the left side of the enclosure. Plug into power source.

**Figure 22:** Feeding power cable through opening in enclosure

3. Attach power cable to the sampler

**Figure 23:** Connecting power cable to the sampler

4. Open sampler box, and push "Auto/On/Off" button twice


**Figure 24:** Auto/on/off button circled in red

5. Adjust flow rate to 3 L/min by turning the knob labelled "Flow Volume". When adjusting flow rate, turn the knob in gradual increments and wait for the red lights to go off before turning knob again.

6. Once flow rate is set and steady, note the starting time on the "Elapsed Time" display. Also note down the actual time. Close the sampler door.

7. Place sampler back in the box and slide 1 piece of soundproofing material on top. The material will have a cutout for the impactor.

**Figure 25:** Sampler placed inside enclosure

**Figure 26:** Soundproofing foam placed to cover the opening on top of the enclosure

8. Close and secure enclosure door.

9. Place tape over the cord, just outside where it comes out of the opening for it on the left side. This will help ensure that if someone steps on the cord, the sampler will not come unplugged.

### 2.5.3 Removing and storing filters

1. Put on gloves, and set down a sheet of aluminium foil to use as a work surface. Wipe down foil and tweezers with alcohol wipe.

2. Open the enclosure door with black plastic key.

3. Take the sampler out of the box, and open the sampler door. Do not unplug the power supply yet.

4. Note down the ending time on the "Elapsed Time" display.

5. Turn off sampler by pressing the "On/Auto/Off" button once.

6. Take the impactor off the sampler, and take it back to your work surface. Unscrew the base of the impactor to reveal the filter holder.

7. Open the filter holder using the cassette separator. Using tweezers, remove the filter, taking care to only hold the plastic ring on the outside.

8. Place filter inside pre-baked foil pocket.

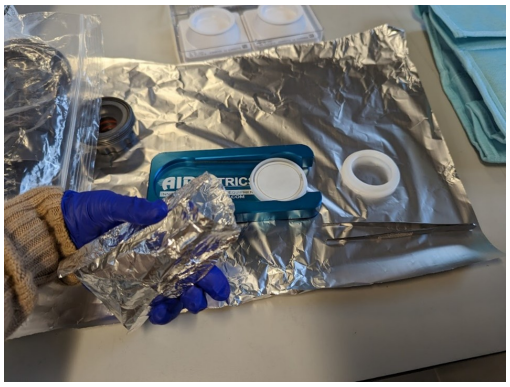


**Figure 27:** Opening a pre-baked foil pocket

9. Fold top of filter pocket down twice, enclosing the filter.

**Figure 28:** Enclosing the filter in the foil pocket

10. Seal the foil inside a plastic bag. Label the bag with House ID and sampling dates.

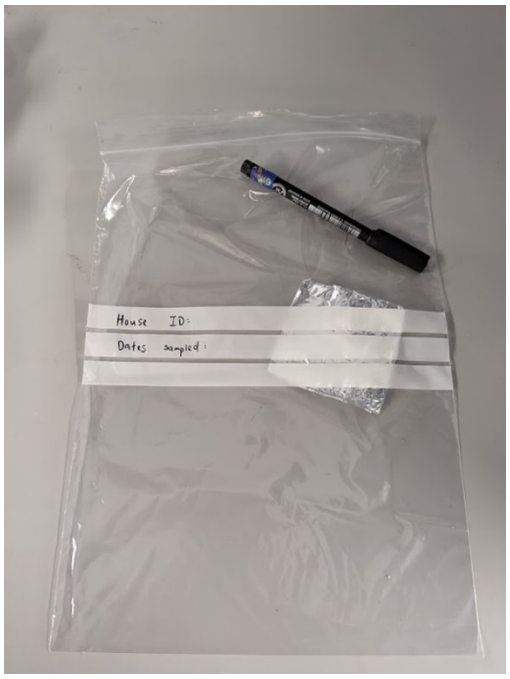



**Figure 29:** Labelling the plastic bags

11. Place the bags inside the freezable sample carriers.


**Figure 30:** Freezable bag for samples

12. Upon return to BiB office, move samples from the freezer bags to the freezer.

## 2.6 Filter sample preparation

In order to extract the organic materials from the $PM_{2.5}$ samples, an accelerated solvent extraction approach was used. Extractions were completed using the ASE-EXTREVA, manufactured by Thermofisher Scientific, (Oven temperature 80 ∘C, flow rate 0.65 ml/min, extraction time 15 min) and using ethyl acetate as a solvent (hypergrade for LC-MS LiChrosolv, purity  99.8 percent, Merck). Filters were spiked with 25 $\mu$l of an internal standard mixture containing six deuterated compounds (EPA 8270 Internal Standard Mix, 6 compounds at 2000 $\mu$g/ml) at a concentration of 20 $\mu$g/ml, for a concentration of 1.0 $\mu$g/ml in solution. The internal standard was diluted using ethyl acetate (hypergrade for LC-MS LiChrosolv, purity $\geq$ 99.8 percent, Merck).

**Figure 31:** Two filters collected from INGENIOUS homes

Samples were extracted into ethyl acetate (EtOAC). Initially this extraction procedure was followed by evaporation under an $N_2$ blowdown in an ice bath. This was extremely labour intensive and more importantly, the final volume of the samples were difficult to control. As such, after extraction, the samples were evaporated to dryness using vacuum evaporation (Mivac) and reconstituted using 500 $\mu$l of EtOAC (300 $\mu$l reconstitution, 2 washes of 100 $\mu$l).

Samples were injected using two-dimensional gas chromatography linked to a time-of-flight mass spectrometer. Samples were injected splitless (1 $\mu$l). The primary column was a RXI-5SilMS (Restek, 30m $\times$ 0.25 $\mu$m $\times$ 0.25mm) connected to a second column of RXI-17SilMS (Restek, 0.25$\mu$m $\times$ 0.25mm, 0.17m primary GC oven, 0.1m modulator, 1.42m secondary

oven, 0.31m transfer line) with a helium flow of 1.4mL/min. The primary oven was held at 40 °C for 1 min, then ramped at 3 °C/min to 322 °C, whereupon it was held for 3 mins. The secondary oven was held at 62 °C for 1 min, then ramped at 3.2 degrees/min to 190 degrees, whereupon it was held for 19.5 mins. The modulator temperature offset (°C, relative to the secondary oven temperature) was 15°C, as per the recommended settings from the manufacturer. The inlet was held at 280 °C, and the transferline at 350 °C. Between each sample, a blank run was performed with ethyl acetate to ensure that there was no carryover between samples. These blanks were run in 1D only, at 320°C for 30 minutes.

## 2.7 Comprehensive Two-Dimensional Gas Chromatography coupled to Time of Flight Mass Spectrometry (GC × GC-ToFMS)

Two-dimensional gas chromatography (GC×GC) is an analytical technique that exceeds the capabilities of traditional one-dimensional gas chromatography (1D GC) by introducing a second separation dimension. 1D GC methods often struggle when use to separate highly complex mixtures, where many analytes may co-elute or overlap in retention time. GC×GC addresses this limitation by combining two columns of differing selectivity, thereby greatly increasing the separation of compounds.

The system operates by connecting two columns in a series, each coated with a stationary phase of different selectivity; in this study, the first column separated analytes based on volatility, while the second column provided separation based on polarity. A modulator, positioned between the two columns, periodically collects effluent from the first dimension and re-injects it into the second column in narrow pulses by using a stop-and-go system of hot and cold pulses of air. This modulation process compresses and focuses peaks, enabling further resolution in the second dimension.

GC×GC produces a two-dimensional separation space, with retention time in the first dimension plotted on the $x$-axis and retention time in the second dimension plotted on the $y$-axis. Each analyte is represented by a spot or band within this space.

When coupled with time-of-flight mass spectrometry (ToF-MS), GC×GC, comprehensive spectral information can be acquired for every resolved peak, allowing compound identification through library matching (min value for match=700/1000); in these studies, the NIST library was used to identify the spectra of individual peaks. Further description of GC×GC and ToF-MS can be found in Chaper 1.

## 2.8 Evaluation of GC×GC–ToF-MS Method

The use of GC×GC–ToF-MS in this study was chosen based on the complexity of indoor particulate matter and the need to resolve structurally similar semi-volatile organic compounds. For example, compound classes such as polycyclic aromatic hydrocarbons (PAHs)may exhibit similar properties, including comparable volatilities and overlapping mass spectral features, making them prone to co-elution in conventional one-dimensional gas chromatography. The additional separation dimension provided by GC×GC was therefore considered necessary to achieve sufficient chromatographic resolution for confident compound identification.

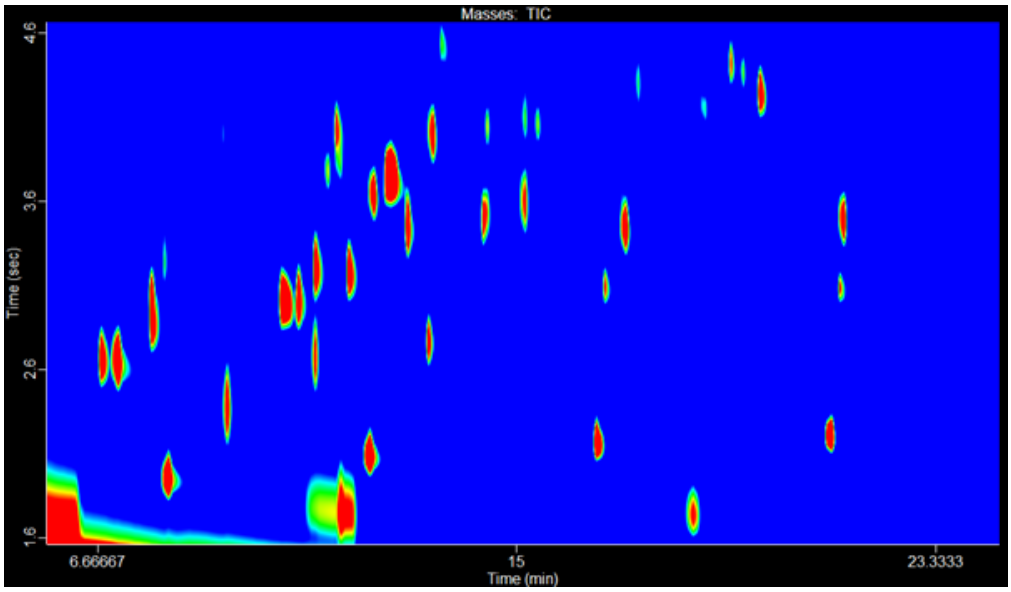



**Figure 32:** Section of a GC×GC chromatogram illustrating separation in the second dimension. Peaks that share similar first-dimension retention times (x-axis) but different second-dimension retention times (y-axis) would co-elute in one-dimensional GC, demonstrating the improved separation of GC×GC.

As shown in Figure 32, compounds that are not resolved in the first dimension can be separated in the second dimension due to differences in polarity. The chromatogram produced by GC×GC also aids in compound classification, as analytes of similar chemical classes tend to co-elute, and thus are closer together in the two-dimensional plot.

The settings employed in this study were selected to prioritise separation while maintaining a practical analysis time. The primary column enabled separation based predominantly on volatility, while the secondary column introduced polarity-based separation.

However, the increased data complexity associated with GC×GC–ToF-MS introduced significant challenges in data processing, peak identification, and quantitation. While the

software was capable of handling large datasets, it was often restrictive in functionality, requiring substantial manual intervention. Peak assignment and verification were frequently performed manually, and additional calculations were required outside of the software environment. As a result, data processing was both time-consuming and labour-intensive, and it is acknowledged that a simpler analytical approach (such as one-dimensional GC) may have been sufficient for many of the target analytes.

Overall, GC×GC–ToF-MS was considered fit for the analysis of indoor particulate matter in this study, as it enabled improved resolution and identification of target analytes compared to conventional GC methods. However, the method should be viewed as a compromise between analytical performance and practical constraints.

### 2.8.1 Choosing Targeted Analytes

Analysis of the data was completed using the pre-installed LECO ChromaTOF software, which was designed to be used with the GC × GC-ToFMS (LECO Pegasus BT 4D). For the targeted analysis component of this project, 102 target analytes were chosen based on pesumed prevalence in home environments, toxicity to humans, and accessibility on the market. Compound classes of the final selected analytes include polycyclic aromatic hydrocarbons, alkanes, phthalates, and aldehydes, all of which are compounds commonly found in homes based on existing literature [115, 116]. Following the first few samples being taken in home environments, several other prevalent compounds of interest (octocrylene, cholesterol, and indoor air fragrances) were added to the list of calibration standards, for a total of 142. Several of these compounds are too volatile to be found using the methods outlined in the sampling procedure. Ultimately, 130 target analytes were selected. Initially, fatty acid methyl esters were included, but these were not found in home environments and their inclusion took up significant time in the data processing method; thus, they were eliminated.

The details of the standards used in the making of the reference mixture are shown in the table below:

### 2.8.2 Injection and Mass Spectrometry Methods

Injections were performed using a Gerstel MPS2 Autosampler. The 10 $\mu$l syringe was prewashed prior to each sample injection with 3 rounds of 1 $\mu$l of two solvents–firstly with DCM, then followed by EtOAC. There were pre- and post-injection delays of 0.5 sec. The injection rate was set at 70 $\mu$l/sec. 1 sample pre-wash was conducted per sample, and the fill volume

**Table 2:** List of Reference Standards

| Name | Manufacturer | Concentration ($\mu$g/ml) | Solvent |
|---|---|---|---|
| EPA 8270 Megamix | Restek | 1000 | DCM |
| C7-C40 Saturated Alkanes | Supelco | 1000 | Hexane |
| 48 Component Indoor Air | Supelco | 1000 | Methanol |
| Stearic acid | Supelco | Neat | N/A |
| Cholesterol | Supelco | 10000 | Chloroform |
| Octocrylene | Supelco | Neat | N/A |

of the syringe syringe was set at 3 $\mu$l. The injection volume of each sample was set at 1 $\mu$l, at a fill speed of 0.5 $\mu$l/sec. After each sample injection, the syringe was post-washed with again with both wash solvents at 2 rounds of 1 $\mu$l of each solvent.

The mass spectrometry method was set with a 210-second acquisition delay, and the acquisition rate was set to 200 spectra/second. The mass range (mu) was 40-500, and the maximum sensitivity setting was used for the detector mode. The extraction frequency was set at 30 kHz.

### 2.8.3 Data Analysis

Table 3 shows the list of target analytes, their display masses, the the correlation coefficients of the calibration method. The LECO ChromaTOF software uses a library search function to identify peaks based on their spectra. The search function utilised the National Institute of Science and Technology (NIST) mass spectral library to compare peaks found in the samples to those in its database. The minimum molecular weight allowed for the search results was set to 45 so as to eliminate the basic gases. The minimum similarity for matches was set to 500 (out of a possible range of 0-999) and a minimum similarity before a hit is assigned was set to 700 (out of a possible range of 0-999).

This tool is fairly accurate; however, each peak needed to be individually identified and assigned as a target analyte. To begin this process, each hit for an analyte of interest had to be manually checked and its peak compared against the spectra in the NIST library. Sometimes, multiple analytes of similar structure and chemical formula eluted at such similar times that the software would misidentify the analyte; to overcome this, data from the NIST library and analysis of the chemical structure of the analytes were used to determine patterns in elution order.

Once this process was completed, a table of retention times and the display masses of

the target analytes was created, and is seen the table below. The display masses refer to the extracted ion that is used for the peak area determination of a particular analyte.

**Table 3:** Retention times, display masses, and calibration correlation coefficients for targeted analytes

| Analyte | 1st Dim Time (min) | 2nd Dim Time (s) | Masses | Corr. Coefficient | LOD | LOQ |
|---|---|---|---|---|---|---|
| n-Hexane | 3.58334 | 4.005 | XIC(57.09±0.03) | 0.97571 | na | na |
| N-Nitrosodimethylamine | 4.16672 | 2.815 | XIC(74.05±0.04) | 0.99025 | 0.9411 | 2.852 |
| Pyridine | 4.25006 | 2.410 | XIC(79.05±0.04) | 0.99129 | 0.8537 | 2.587 |
| Toluene | 4.58342 | 2.125 | XIC(91.06±0.05) | 0.97474 | 0.8997 | 2.726 |
| Octane | 5.25014 | 1.710 | XIC(43.08±0.02) | 0.94050 | 0.8559 | 2.594 |
| Ethylbenzene | 6.83360 | 2.620 | XIC(91.06±0.05) | 0.95448 | 0.8077 | 2.448 |
| p-Xylene | 7.16696 | 2.600 | XIC(91.06±0.05) | 0.96101 | 1.032 | 3.129 |
| Styrene | 7.83368 | 3.020 | XIC(104.06±0.05) | 0.97582 | 1.627 | 4.932 |
| o-Xylene | 7.91702 | 2.835 | XIC(91.06±0.05) | 0.97508 | 1.084 | 3.284 |
| Nonane | 8.16704 | 1.915 | XIC(43.07±0.02) | 0.96188 | 1.52 | 4.607 |
| $\alpha$-Pinene | 9.33380 | 2.330 | XIC(93.07±0.05) | 0.98816 | | |
| Benzene, 1-ethyl-3-methyl- | 10.41720 | 2.975 | XIC(105.07±0.05) | 0.88231 | 0.5147 | 1.56 |
| Benzene, 1-ethyl-4-methyl- | 10.58390 | 2.955 | XIC(105.07±0.05) | 0.97812 | 1.105 | 3.349 |
| Mesitylene | 10.83390 | 2.975 | XIC(105.07±0.05) | 0.98723 | 0.8396 | 2.544 |
| $\beta$-Pinene | 11.08390 | 2.625 | XIC(93.07±0.05) | 0.99330 | | |
| Phenol | 11.33400 | 3.750 | XIC(94.04±0.05) | 0.99577 | 0.7442 | 2.255 |
| Aniline | 11.33400 | 4.585 | XIC(93.06±0.05) | NA | 0.3888 | 1.178 |
| Phenol, 2-chloro- | 11.58400 | 3.790 | XIC(128.01±0.06) | 0.99620 | 0.598 | 1.812 |
| Bis(2-chloroethyl) ether | 11.58400 | 3.975 | XIC(93.01±0.05) | 0.98761 | 0.6375 | 1.932 |
| Benzene, 1,2,3-trimethyl- | 11.83400 | 3.125 | XIC(105.07±0.05) | 0.97980 | 1.139 | 3.452 |
| Decane | 12.25070 | 2.045 | XIC(43.08±0.02) | 0.95803 | 0.8119 | 2.46 |
| Benzene, 1,3-dichloro- | 12.25070 | 3.615 | XIC(145.98±0.07) | 0.98665 | 0.6991 | 2.119 |
| 1,4-Dichlorobenzene-D4 | 12.58410 | 3.700 | XIC(150.01±0.08) | NA | na | na |
| Benzene, 1,4-dichloro- | 12.66740 | 3.725 | XIC(145.98±0.07) | 0.97827 | 0.8932 | 2.707 |
| Benzene, 1,2,4-trimethyl- | 13.00080 | 3.390 | XIC(105.07±0.05) | 0.98005 | 1.301 | 3.943 |
| D-Limonene | 13.41750 | 2.715 | XIC(68.07±0.03) | 0.98196 | 1.016 | 3.08 |
| Benzene, 1,2-dichloro- | 13.41750 | 3.975 | XIC(145.98±0.07) | 0.97541 | 0.6479 | 1.963 |
| Benzyl alcohol | 13.66750 | 4.505 | XIC(79.06±0.04) | 0.99863 | 0.6944 | 2.104 |
| Bis(2-chloroisopropyl) ether | 14.50090 | 3.460 | XIC(45.05±0.02) | 0.97294 | 1.067 | 3.235 |

*Continued on next page*

| Analyte | 1st Dim Time (min) | 2nd Dim Time (s) | Masses | Corr. Coefficient | LOD | LOQ |
|---|---|---|---|---|---|---|
| Phenol, 2-methyl- | 14.58420 | 3.995 | XIC(108.06±0.05) | 0.99879 | 0.6223 | 1.886 |
| 1-Propanamine, N-nitroso-N-propyl- | 15.25090 | 4.065 | XIC(70.08±0.04) | 0.99268 | 0.6968 | 2.111 |
| Phenol, 3-methyl- | 15.58430 | 4.010 | XIC(107.05±0.05) | 0.99956 | 0.7619 | 2.309 |
| Benzene, nitro- | 16.00100 | 0.100 | XIC(77.05±0.04) | 0.99730 | 0.6173 | 1.871 |
| Undecane | 16.75110 | 2.125 | XIC(57.09±0.03) | 0.96136 | 1.039 | 3.148 |
| Nonanal | 16.91770 | 3.050 | XIC(57.08±0.03) | 0.99176 | 0.5099 | 1.545 |
| Benzene, 1,2,4,5-tetramethyl- | 17.33440 | 3.390 | XIC(119.09±0.06) | 0.96789 | 0.9214 | 2.792 |
| Isophorone | 17.58450 | 4.265 | XIC(82.05±0.04) | 0.99831 | 0.5049 | 1.53 |
| Phenol, 2-nitro- | 17.91780 | 4.865 | XIC(139.04±0.07) | 0.99922 | 0.6533 | 1.98 |
| Phenol, 2,4-dimethyl- | 18.91790 | 4.105 | XIC(107.06±0.05) | 0.99941 | 0.3582 | 1.085 |
| Methane, bis(2-chloroethoxy)- | 19.41790 | 4.395 | XIC(93.01±0.05) | 0.98374 | 0.7356 | 2.229 |
| Phenol, 2,4-dichloro- | 19.66800 | 4.310 | XIC(161.97±0.08) | 0.99959 | 0.6485 | 1.965 |
| Benzene, 1,2,3-trichloro- | 20.00130 | 4.205 | XIC(179.94±0.09) | 0.98537 | 0.6989 | 2.118 |
| Naphthalene-D8 | 20.25130 | 4.850 | XIC(136.12±0.07) | NA | na | na |
| Naphthalene | 20.33470 | 4.890 | XIC(128.07±0.06) | 0.99059 | 0.817 | 2.476 |
| Dodecane | 21.41810 | 2.165 | XIC(57.09±0.03) | 0.96203 | 0.532 | 1.612 |
| Decanal | 21.58480 | 3.045 | XIC(57.07±0.03) | 0.96702 | 1.129 | 3.42 |
| 1,3-Butadiene, 1,1,2,3,4,4-hexachloro- | 21.66810 | 3.440 | XIC(224.86±0.11) | 0.98718 | 0.4493 | 1.362 |
| p-Chloroaniline | 22.50150 | 0.040 | XIC(127.03±0.06) | NA | na | na |
| Phenol, 4-chloro-3-methyl- | 25.33510 | 4.485 | XIC(107.06±0.05) | 0.99966 | 0.5314 | 1.61 |
| Naphthalene, 2-methyl- | 25.33510 | 4.740 | XIC(142.08±0.07) | 0.99701 | 0.6598 | 1.999 |
| Tridecane | 25.91850 | 2.180 | XIC(57.09±0.03) | 0.95726 | 0.6504 | 1.971 |
| Naphthalene, 1-methyl- | 26.00180 | 4.965 | XIC(142.08±0.07) | 0.99733 | 0.1766 | 0.5351 |
| Hexachlorocyclopentadiene | 26.75190 | 3.720 | XIC(236.86±0.12) | 0.99443 | 0.1661 | 0.5033 |
| Phenol, 2,4,6-trichloro- | 27.83530 | 4.585 | XIC(195.94±0.10) | 0.99792 | 0.7593 | 2.301 |
| Phenol, 2,4,5-trichloro- | 28.08530 | 4.550 | XIC(195.94±0.10) | 0.99887 | 0.6721 | 2.037 |
| Naphthalene, 2-chloro- | 28.91870 | 0.050 | XIC(162.03±0.08) | 0.99528 | 0.6159 | 1.866 |
| o-Nitroaniline | 30.16880 | 1.645 | XIC(65.05±0.03) | 0.99927 | 0.5661 | 1.715 |
| Tetradecane | 30.25210 | 2.185 | XIC(57.09±0.03) | 0.96535 | 0.6823 | 2.067 |

*Continued on next page*

| Analyte | 1st Dim Time (min) | 2nd Dim Time (s) | Masses | Corr. Coefficient | LOD | LOQ |
|---|---|---|---|---|---|---|
| Benzene, 1,4-dinitro- | 31.25220 | 2.030 | XIC(75.03±0.04) | 0.99927 | 0.6954 | 2.107 |
| Acenaphthylene | 31.83560 | 0.685 | XIC(152.07±0.08) | 0.99868 | 0.5229 | 1.585 |
| Benzene, 1,3-dinitro- | 31.83560 | 1.830 | XIC(76.04±0.04) | 0.99964 | 0.5859 | 1.776 |
| Dimethyl phthalate | 32.08560 | 0.645 | XIC(163.05±0.08) | 0.99986 | 0.4911 | 1.488 |
| Benzene, 2-methyl-1,3-dinitro- | 32.16900 | 1.470 | XIC(165.04±0.08) | 0.99969 | 0.6176 | 1.871 |
| Benzene, 1,2-dinitro- | 32.41900 | 2.270 | XIC(50.04±0.03) | 0.99998 | 0.5391 | 1.634 |
| Acenaphthene-d10 | 33.00240 | 0.455 | XIC(164.15±0.08) | NA | na | na |
| Acenaphthene | 33.25240 | 0.480 | XIC(153.08±0.08) | 0.99383 | 0.6104 | 1.85 |
| m-Nitroaniline | 33.66910 | 1.790 | XIC(65.05±0.03) | 0.98315 | 0.2317 | 0.7021 |
| Pentadecane | 34.33580 | 2.180 | XIC(57.09±0.03) | 0.96625 | 0.8514 | 2.58 |
| Dibenzofuran | 34.58580 | 0.285 | XIC(168.06±0.08) | 0.99124 | 0.6791 | 2.058 |
| Benzene, 1-methyl-2,4-dinitro- | 35.16920 | 1.210 | XIC(165.04±0.08) | 0.99957 | 0.5942 | 1.801 |
| Phenol, 2,3,5,6-tetrachloro- | 35.58590 | 4.925 | XIC(231.90±0.12) | 0.99407 | na | na |
| Phenol, 2,3,4,6-tetrachloro- | 35.91930 | 4.985 | XIC(231.90±0.12) | 0.99727 | na | na |
| Phenol, 4-nitro- | 36.16930 | 0.335 | XIC(65.06±0.03) | 0.99927 | na | na |
| Fluorene | 37.16940 | 0.435 | XIC(166.08±0.08) | 0.99712 | 0.7379 | 2.236 |
| Diethyl Phthalate | 37.58610 | 0.025 | XIC(149.03±0.07) | 0.99872 | 0.6164 | 1.868 |
| Benzene, 1-chloro-4-phenoxy- | 37.66940 | 4.810 | XIC(204.04±0.10) | 0.99877 | 0.5744 | 1.741 |
| Hexadecane | 38.16940 | 2.185 | XIC(57.09±0.03) | 0.98791 | 0.6906 | 2.093 |
| p-Nitroaniline | 38.16940 | 2.575 | XIC(65.05±0.03) | NA | 1.956 | 5.927 |
| Diphenylamine | 38.75280 | 0.600 | XIC(169.10±0.08) | 0.99938 | 0.5124 | 1.553 |
| Azobenzene | 38.83620 | 0.065 | XIC(77.04±0.04) | 0.99165 | 0.696 | 2.109 |
| Benzene, hexachloro- | 41.25300 | 4.870 | XIC(283.82±0.14) | 0.99232 | 0.6049 | 1.833 |
| Benzene, 1-bromo-4-phenoxy- | 41.33640 | 0.065 | XIC(248.00±0.12) | 0.99456 | 0.5797 | 1.757 |
| Heptadecane | 41.91970 | 2.160 | XIC(57.09±0.03) | 0.98215 | 0.714 | 2.163 |
| Phenol, pentachloro- | 43.41990 | 0.075 | XIC(265.86±0.13) | 0.98251 | na | na |
| Phenanthrene-D10 | 44.08660 | 0.915 | XIC(188.15±0.09) | NA | na | na |
| Phenanthrene | 44.25330 | 0.930 | XIC(178.09±0.09) | 0.99645 | 0.5797 | 1.757 |

*Continued on next page*

| Analyte | 1st Dim Time (min) | 2nd Dim Time (s) | Masses | Corr. Coefficient | LOD | LOQ |
|---|---|---|---|---|---|---|
| Anthracene | 44.67000 | 0.815 | XIC(178.09±0.09) | 0.99980 | 0.4837 | 1.466 |
| Octadecane | 45.42000 | 2.125 | XIC(57.09±0.03) | 0.97950 | 0.7416 | 2.247 |
| Carbazole | 46.33680 | 1.475 | XIC(167.08±0.08) | 0.99992 | 0.5404 | 1.638 |
| Nonadecane | 48.75360 | 2.090 | XIC(57.09±0.03) | 0.98801 | 0.7212 | 2.185 |
| Dibutyl phthalate | 50.25370 | 3.940 | XIC(149.03±0.07) | 0.99361 | 0.562 | 1.703 |
| Eicosane | 51.92050 | 2.055 | XIC(57.09±0.03) | 0.98314 | 0.7229 | 2.191 |
| Fluoranthene | 53.25400 | 0.810 | XIC(202.09±0.10) | 0.99968 | 0.5397 | 1.635 |
| Pyrene | 54.75410 | 1.135 | XIC(202.09±0.10) | 0.99952 | 0.6221 | 1.885 |
| Heneicosane | 55.00410 | 2.025 | XIC(57.09±0.03) | 0.99031 | 0.6929 | 2.1 |
| Docosane | 57.92100 | 1.995 | XIC(57.09±0.03) | 0.97483 | 0.7165 | 2.171 |
| Tricosane | 60.67120 | 1.980 | XIC(57.09±0.03) | 0.97582 | 0.7106 | 2.153 |
| Benzyl butyl phthalate | 61.42130 | 4.430 | XIC(91.06±0.05) | 0.99890 | 0.4899 | 1.484 |
| Hexanedioic acid, bis(2-ethylhexyl) ester | 63.00480 | 2.480 | XIC(129.06±0.06) | 0.98785 | 0.7732 | 2.343 |
| Tetracosane | 63.42150 | 1.950 | XIC(57.09±0.03) | 0.97651 | 0.684 | 2.073 |
| Benz[a]anthracene | 64.08820 | 0.700 | XIC(228.11±0.11) | 0.99951 | 0.5031 | 1.525 |
| Chrysene-D12 | 64.17150 | 0.850 | XIC(240.18±0.12) | NA | na | na |
| Chrysene | 64.33820 | 0.870 | XIC(228.10±0.11) | 0.99556 | 0.1579 | 0.4785 |
| Pentacosane | 66.00500 | 1.925 | XIC(57.09±0.03) | 0.97488 | 0.7066 | 2.141 |
| Bis(2-ethylhexyl) phthalate | 66.58840 | 2.895 | XIC(149.03±0.07) | 0.98626 | 0.6168 | 1.869 |
| Hexacosane | 68.50520 | 1.910 | XIC(57.09±0.03) | 0.97939 | 0.7002 | 2.122 |
| Octocrylene | 69.33860 | 3.745 | XIC(70.09±0.04) | 0.99965 | 0.4254 | 1.289 |
| Heptacosane | 70.92210 | 1.895 | XIC(57.09±0.03) | 0.98512 | 0.3768 | 1.142 |
| Di-n-octyl phthalate | 71.25540 | 2.880 | XIC(149.03±0.07) | 0.99053 | 0.4694 | 1.422 |
| Benzo[b]fluoranthene | 71.75550 | 0.710 | XIC(252.11±0.13) | 0.99867 | 0.1406 | 0.4262 |
| Benzo[k]fluoranthene | 71.92210 | 0.705 | XIC(252.11±0.13) | 0.99831 | 0.1258 | 0.3811 |
| Octacosane | 73.17220 | 1.885 | XIC(57.08±0.03) | 0.95715 | 0.8632 | 2.616 |
| Benzo[a]pyrene | 73.67230 | 1.140 | XIC(252.11±0.13) | 0.99904 | 0.126 | 0.382 |
| Perylene-D12 | 74.08900 | 1.220 | XIC(264.18±0.13) | NA | na | na |
| Nonacosane | 75.42240 | 1.870 | XIC(57.09±0.03) | 0.97791 | 0.6675 | 2.023 |
| Triacontane | 77.58930 | 1.865 | XIC(57.08±0.03) | 0.97603 | 0.914 | 2.77 |
| Hentriacontane | 79.67280 | 1.965 | XIC(57.08±0.03) | 0.99098 | 0.05497 | 0.1666 |
| Dibenz[a,h]anthracene | 80.75620 | 1.945 | XIC(278.12±0.14) | 0.99178 | 0.1117 | 0.3385 |

*Continued on next page*

| Analyte | 1st Dim Time (min) | 2nd Dim Time (s) | Masses | Corr. Coefficient | LOD | LOQ |
|---|---|---|---|---|---|---|
| Benzo[ghi]perylene | 81.67290 | 3.175 | XIC(276.11±0.14) | 0.99037 | 0.1004 | 0.3044 |
| Dotriacontane | 81.75630 | 2.120 | XIC(57.09±0.03) | 0.99576 | 0.139 | 0.4211 |
| Tritriacontane | 83.67310 | 2.315 | XIC(57.09±0.03) | 0.99588 | 0.1242 | 0.3764 |
| Tetratriacontane | 85.58990 | 2.550 | XIC(57.09±0.03) | 0.99083 | 0.2189 | 0.6632 |
| Pentatriacontane | 87.42340 | 2.825 | XIC(57.09±0.03) | 0.98754 | 0.3231 | 0.9792 |
| Hexatriacontane | 89.25690 | 3.140 | XIC(57.09±0.03) | 0.99039 | 0.4022 | 1.219 |
| Heptatriacontane | 91.00700 | 3.535 | XIC(57.09±0.03) | 0.98537 | 0.4626 | 1.402 |
| Octatriacontane | 92.67380 | 3.980 | XIC(57.09±0.03) | 0.97611 | na | na |
| Nonatriacontane | 94.34060 | 4.525 | XIC(57.09±0.03) | 0.97363 | na | na |

To quantify the target analytes, the software required a number of preliminary steps, and which needed to be repeated every time that the second column had been changed. During the injection of the calibration curve, the data processing method was set only to identify the peaks. First, the target analytes had to be individually identified and assigned as an analyte, preferably in the sampler at the middle point of the calibration curve. In this case, the middle point was 0.5 $\mu$g/ml. This process was done manually each time the calibration range was injected–this would occur roughly every 10 samples, or whenever the second column snapped and needed replacement. The analyte identification process needed to be repeated regularly to account for changes in the column retention times as the column degraded.

These analytes were added to a Target Analyte Finding (TAF) method, which was used on all samples of the calibration curve. Following this, a Quantitation Method was created, whereby the analytes' concentrations were identified and internal standards identified. The software then generated plots and linear equations for each target analyte, which would be used to allow the software to quantify target analytes in real-home samples.

### 2.8.4 Calibration and Quantitation

A eight-point calibration curve was created with concentrations 0.01 $\mu$g/ml, 0.05 $\mu$g/ml, 0.1 $\mu$g/ml, 0.25 $\mu$g/ml, 0,5 $\mu$g/ml, 1.0 $\mu$g/ml, 1.5 $\mu$g/ml and 2.5 $\mu$g/ml in ethyl acetate. Each point in the calibration curve was also spiked with a six-compound deuterated internal standard (EPA 8270 Semivolatile Internal Standard Reference Mix, Supelco), specifically with 25 $\mu$l at 20 $\mu$g/ml for a final concentration in solution of 1.0 $\mu$g/ml. Once injected into the 2DGC, an internal standard had to be selected for each compound. Phenanthrene-D10 or Perylene D-12 were the two most commonly used internal standards, due to their reliability of recovery and their suitability for use (based on improving the correlation coefficient value when used) with the particular analyte.

Samples were analysed in batches, with quality control measures implemented throughout each analytical sequence. Each sample was injected at least twice, and the mean of the duplicate injections was used for quantitation. This approach was used to minimise the influence of short-term instrumental variability and injection efficiency, thereby reducing the contribution of instrument-related uncertainty to the reported concentrations.

Between injections, solvent blanks (ethyl acetate) were run at the maximum oven temperature of the method to minimise carryover and ensure that residual compounds from previous

injections did not affect subsequent samples.

Calibration standards were re-injected regularly throughout each batch to monitor instrument stability and account for potential drift. Specifically, calibration standards were analysed approximately every ten samples, or at the start of each new liquid nitrogen tank used for modulation, whichever occurred more frequently. As liquid nitrogen was replenished on a weekly basis, this limited analytical batches, and calibration was repeated at the beginning of each batch to ensure consistent modulation performance and retention behaviour.

Figure 33 shows the full reference standard of 130 species on a 2D plot, at a concentration of 1.0 $\mu$g/ml; it is a total ion chromatogram, showing a summary across all masses. To give a brief overview of how to interpret a 2D plot: the x-axis shows the separation in the 1st dimension in units of mins; as time increases, volatility decreases. The y-axis shows the separation in the second dimension in seconds; as time increases, polarity increases. The colours show ion abundance, scaled to the highest peak.

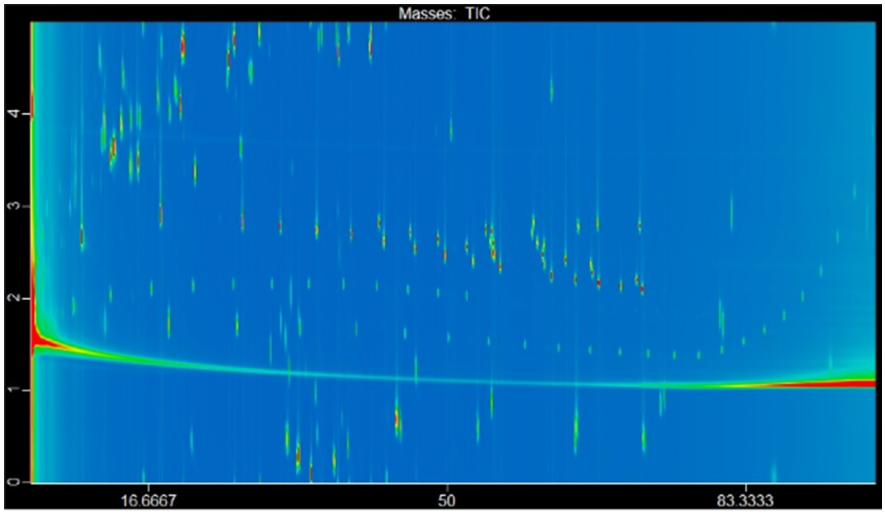



**Figure 33:** Full reference standard at 1.0 $\mu$g/ml

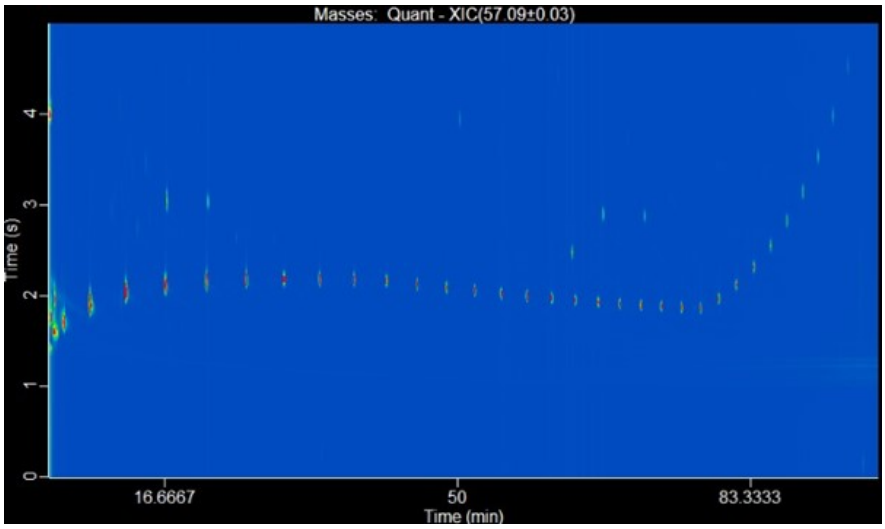



**Figure 34:** Filtering by mass 57, displaying C7-C39 alkanes

Figure 34, in contrast, shows the data filtered by a mass of 57. At this mass, alkanes are shown. This type of data filtration was very useful in investigating the presence and prevalence of certain compound classes, for example alkanes and PAHs.

Table 3 shows the correlation coefficients obtained after the software had generated calibration curves from the injections of the reference standards. The 6 internal standards are marked "NA", as they were defined manually and form the basis of the other standards' calculated correlation coefficients. As seen in the table, all target analytes have correlation coefficient values >0.9000, with a majority having values >0.9900. These correlation coefficient values showed that the calibration curves were of good quality.

A retention time deviation was set for each analyte, and was set at 0.2 mins (6 seconds) in the first dimension and for 0.5 seconds in the second dimension. The match threshold was set at 700 (range of 0-999).

Blank samples were analysed alongside environmental samples and were spiked with internal standard and injected using the same process as the INGENIOUS samples. Blank correction was completed post-acquisition using R, whereby the concentration of each analyte found in the blank was subtracted from the matching analytes found in the real-home samples. The median blank value was used as the basis for subtraction to minimise the influence of outliers or sporadic contamination.

### 2.8.5 Recovery Testing

Recoveries were assessed using spiked standards ($n = 4$), but those values were inconsistent with concentrations observed in real samples. Results of recovery testing are shown in Table 4; of 130 targeted analytes, 52 were detected. We determined that recovery correction was not reliable, as spiking a reference compound onto a filter in liquid form does not replicate aerosol dynamics in real air: compounds may be taken up irreversibly onto particle surfaces or absorbed into a liquid or glassy/amorphous phase, and this may not correspond to volatility-based estimates of gas–particle partitioning. Discrepancies of this nature between spiked-filter recoveries and sampling-derived recoveries have been documented in particulate sampling studies, where spiked recoveries primarily reflect extraction efficiency and can vary widely between compounds rather than representing real sampling behaviour [117, 118]. Importantly, measured concentrations of compound classes such as PAHs and phthalates were consistent with ranges reported in previous studies of indoor environments, providing independent validation of the analytical approach despite low apparent recoveries. Given the inconsistency and lack of physical representativeness, recovery correction was not applied in this study.

**Table 4:** Recovery testing values for target analytes

| Analyte | Median Recovery (%) | n detected |
|---|---|---|
| Decane | 0.15 | 2 |
| D-Limonene | 0.22 | 1 |
| Dodecane | 0.23 | 4 |
| Tetradecane | 0.26 | 4 |
| Fluorene | 0.42 | 1 |
| Tridecane | 0.45 | 4 |
| Toluene | 0.51 | 4 |
| Undecane | 0.90 | 2 |
| Acenaphthylene | 1.13 | 3 |
| Hexadecane | 1.79 | 4 |
| Benzene, 1-methyl-2,4-dinitro- | 2.59 | 2 |
| Pentadecane | 2.66 | 2 |
| Heptadecane | 3.18 | 3 |
| Benzene, hexachloro- | 3.61 | 4 |
| Benzene, 1-bromo-4-phenoxy- | 4.38 | 2 |
| Diphenylamine | 4.42 | 4 |

| | | |
|---|---|---|
| Docosane | 5.40 | 2 |
| Phenol, 2,3,5,6-tetrachloro- | 5.68 | 1 |
| Diethyl Phthalate | 6.68 | 3 |
| Anthracene | 7.24 | 1 |
| Phenol, 2,3,4,6-tetrachloro- | 9.79 | 1 |
| Phenanthrene | 11.09 | 3 |
| Octadecane | 16.38 | 4 |
| Decanal | 18.13 | 4 |
| Hexacosane | 20.47 | 1 |
| Nonanal | 22.48 | 4 |
| Indeno[1,2,3-cd]pyrene | 37.14 | 3 |
| Nonadecane | 41.58 | 4 |
| Dibenz[a,h]anthracene | 41.66 | 2 |
| Pyrene | 47.48 | 4 |
| Octocrylene | 47.54 | 4 |
| Fluoranthene | 48.11 | 4 |
| Cholesterol | 48.67 | 4 |
| Benzo[a]pyrene | 49.09 | 2 |
| Dibutyl phthalate | 51.29 | 3 |
| Heptacosane | 52.00 | 4 |
| Benzo[ghi]perylene | 52.93 | 4 |
| Benzo[b]fluoranthene | 57.17 | 4 |
| Eicosane | 57.94 | 4 |
| Heneicosane | 64.74 | 4 |
| Benzo[k]fluoranthene | 65.43 | 4 |
| Benzyl butyl phthalate | 66.77 | 4 |
| Hexanedioic acid, bis(2-ethylhexyl) ester | 68.03 | 2 |
| Di-n-octyl phthalate | 69.36 | 4 |
| Tetracosane | 69.59 | 4 |
| Bis(2-ethylhexyl) phthalate | 71.65 | 4 |
| Hentriacontane | 79.86 | 4 |
| Octacosane | 80.71 | 4 |
| Benz[a]anthracene | 81.26 | 4 |
| 1,3-Butadiene, 1,1,2,3,4,4-hexachloro- | 0.00 | 0 |
| 1-Propanamine, N-nitroso-N-propyl- | 0.00 | 0 |
| Acenaphthene | 0.00 | 0 |

| | | |
|---|---|---|
| Aniline | 0.00 | 0 |
| Azobenzene | 0.00 | 0 |
| Benzene, 1,2,3-trichloro- | 0.00 | 0 |
| Benzene, 1,2,3-trimethyl- | 0.00 | 0 |
| Benzene, 1,2,4,5-tetramethyl- | 0.00 | 0 |
| Benzene, 1,2,4-trimethyl- | 0.00 | 0 |
| Benzene, 1,2-dichloro- | 0.00 | 0 |
| Benzene, 1,2-dinitro- | 0.00 | 0 |
| Benzene, 1,3-dichloro- | 0.00 | 0 |
| Benzene, 1,3-dinitro- | 0.00 | 0 |
| Benzene, 1,4-dichloro- | 0.00 | 0 |
| Benzene, 1,4-dinitro- | 0.00 | 0 |
| Benzene, 1-chloro-4-phenoxy- | 0.00 | 0 |
| Benzene, 1-ethyl-3-methyl- | 0.00 | 0 |
| Benzene, 1-ethyl-4-methyl- | 0.00 | 0 |
| Benzene, 2-methyl-1,3-dinitro- | 0.00 | 0 |
| Benzene, nitro- | 0.00 | 0 |
| Benzyl alcohol | 0.00 | 0 |
| Bis(2-chloroethyl) ether | 0.00 | 0 |
| Bis(2-chloroisopropyl) ether | 0.00 | 0 |
| Carbazole | 0.00 | 0 |
| Chrysene | 0.00 | 0 |
| Dibenzofuran | 0.00 | 0 |
| Dimethyl phthalate | 0.00 | 0 |
| Dotriacontane | 0.00 | 0 |
| Ethylbenzene | 0.00 | 0 |
| Heptatriacontane | 0.00 | 0 |
| Hexachlorocyclopentadiene | 0.00 | 0 |
| Hexatriacontane | 0.00 | 0 |
| Isophorone | 0.00 | 0 |
| Mesitylene | 0.00 | 0 |
| Methane, bis(2-chloroethoxy)- | 0.00 | 0 |
| N-Nitrosodimethylamine | 0.00 | 0 |
| Naphthalene | 0.00 | 0 |
| Naphthalene, 1-methyl- | 0.00 | 0 |
| Naphthalene, 2-chloro- | 0.00 | 0 |

| | | |
|---|---|---|
| Naphthalene, 2-methyl- | 0.00 | 0 |
| Nonacosane | 0.00 | 0 |
| Nonane | 0.00 | 0 |
| Nonatriacontane | 0.00 | 0 |
| Octane | 0.00 | 0 |
| Octatriacontane | 0.00 | 0 |
| Pentacosane | 0.00 | 0 |
| Pentatriacontane | 0.00 | 0 |
| Phenol | 0.00 | 0 |
| Phenol, 2,4,5-trichloro- | 0.00 | 0 |
| Phenol, 2,4,6-trichloro- | 0.00 | 0 |
| Phenol, 2,4-dichloro- | 0.00 | 0 |
| Phenol, 2,4-dimethyl- | 0.00 | 0 |
| Phenol, 2-chloro- | 0.00 | 0 |
| Phenol, 2-methyl- | 0.00 | 0 |
| Phenol, 2-nitro- | 0.00 | 0 |
| Phenol, 3-methyl- | 0.00 | 0 |
| Phenol, 4-chloro-3-methyl- | 0.00 | 0 |
| Phenol, 4-nitro- | 0.00 | 0 |
| Phenol, pentachloro- | 0.00 | 0 |
| Pyridine | 0.00 | 0 |
| Styrene | 0.00 | 0 |
| Tetratriacontane | 0.00 | 0 |
| Triacontane | 0.00 | 0 |
| Tricosane | 0.00 | 0 |
| Tritriacontane | 0.00 | 0 |
| m-Nitroaniline | 0.00 | 0 |
| n-Hexane | 0.00 | 0 |
| o-Nitroaniline | 0.00 | 0 |
| o-Xylene | 0.00 | 0 |
| p-Chloroaniline | 0.00 | 0 |
| p-Nitroaniline | 0.00 | 0 |
| p-Xylene | 0.00 | 0 |
| $\alpha$-Pinene | 0.00 | 0 |
| $\beta$-Pinene | 0.00 | 0 |

During recovery testing, measured concentrations of individual target compounds were consistent with values reported in the literature for indoor environments, providing support for the analytical approach despite low recoveries observed in spike experiments. Benzo[a]pyrene (BaP), a well-established marker of PAHs, was typically present at low concentrations, with a median of 2.1 ng $m^{-3}$ and a maximum of 5.76 ng $m^{-3}$. This is consistent with (and higher than) reported indoor BaP concentrations, where median values ranged from 0.09 ng $m^{-3}$ to 0.65 ng $m^{-3}$ range in residential environments, depending on season and residents' smoking status [119]. This supports the decision to not apply recovery correction.

In contrast, dibenz[a,h]anthracene was more consistently detected, with a median concentration of 10.6 ng $m^{-3}$ and values ranging up to 41.8 ng $m^{-3}$, placing the measured concentrations within the range of reported indoor values [120].

Phthalates were present at substantially higher concentrations, consistent with their known emission from indoor materials and consumer products. Dibutyl phthalate exhibited a median concentration of 228.6 ng $m^{-3}$, with values up to 483.8 ng $m^{-3}$, while bis(2-ethylhexyl) phthalate (DEHP) showed a median of 104.8 ng $m^{-3}$ and a maximum of 358.3 ng $m^{-3}$. These concentrations are well within the ranges reported in indoor air studies, where DBP and DEHP are commonly detected at tens to several hundred ng $m^{-3}$ [121].

A total of 90 unique analytes were measured in the real-home samples; in contrast, recovery experiments detected fewer than half of the target analytes, with approximately 60% of compounds not recovered in spike tests. This discrepancy indicates that spike-based recoveries substantially underestimate the effective detection of analytes under real sampling conditions.

The agreement between measured concentrations and literature-reported ranges for specific compounds indicates that the analytical method captures environmentally realistic concentrations. This supports the conclusion that the low and variable recoveries obtained from spiked filter experiments are not representative of real sampling processes, and that applying recovery corrections derived from such experiments would likely introduce bias rather than improve accuracy.

## 2.9 Conclusions

The aim of this chapter was to find and/or create a sampling methodology which would suit the needs of both the INGENIOUS project and of the families participating in this study. By using the Minivol TAS, a sampler that was mass-manufactured and which had gone through all commercially required testing, the participants' safety was prioritised; the modifications made via the soundproofing enclosures reduced noise to a comfortable level (50 dBA, which is equivalent to the noise caused by a refrigerator and a ten-fold decrease from initial levels). This was important not only from an ethical and safety standpoint, but also to ensure that participants did not change their behaviours overmuch with the goal of obtaining samples that reflected their real-life behaviours as much as was possible. Additionally, the cost efficiency of the sampler ensured that no undue financial burden would be placed upon the families participating in this study.

The development of a Standard Operating Procedure and training of research assistants ensured that the samplers were deployed as uniformly as possible, and that the samples were safely collected and stored until further analysis could be completed. Public engagement events were carried out in order to gauge community response and to resolve potential concerns before the deployment period. Importantly, zero samplers were turned off or moved by participants, showing that the soundproofing measures and deployment strategy (in terms of how and where samplers were placed) were successful. The method development outlined in this chapter made it possible for particulate matter sampling to be conducted in residential environments while causing the least possible disruption to study participants.

# Chapter 3: INGENIOUS: Characterising and Quantifying Particulate Matter in Real-Home Environments in Bradford, UK

# 3 INGENIOUS: Characterising and Quantifying Particulate Matter in Real-Home Environments in Bradford, UK

The collection of samples and procurement of raw data were outlined in the preceding chapter. This chapter presents the outcomes of detailed analysis of the INGENIOUS WP2, dataset derived from real-home environments in Bradford, UK. Emphasis is placed on quantifying and characterising indoor particulate matter ($PM_{2.5}$) and its chemical composition.

The chapter is structured to provide a comprehensive overview of the findings across all 34 sampled homes, including statistics of pollutant concentrations, source attribution analysis, and predicted health outcomes.

## 3.1 Experimental Section

### 3.1.1 The Cohort

Bradford is the fifth-largest city in the UK, with a population of approximately 560,200 and notable for its high ethnic diversity—32% of residents identify as Asian, predominantly South Asian of Pakistani descent [122]. According to the 2021 Census for England and Wales, around 57% of households in the Bradford district experience deprivation in at least one area: education, employment, health, or housing, compared to the national average of 52% [122]. Between 2021 and 2023, outdoor annual $PM_{2.5}$ levels ranged from 7.1 to 8.4 $\mu g/m^3$; the WHO air quality guideline (AQG) states that annual average concentrations of $PM_{2.5}$ should not exceed 5 $\mu g/m^3$, though it is important to note that this is an ambitious health-based guideline [15, 122, 123]. Rates of respiratory illness in Bradford are also above the national average, with 7.4% of residents diagnosed with asthma, compared to 6.5% across the country [122].

The INGENIOUS cohort was comprised of 315 households, recruited from the 12,453 Born in Bradford (BiB) families. The Born in Bradford families were recruited between 2007 and 2011, and consisted of 12,453 women with 13,776 pregnancies (recruited at around 28 weeks)[124]. The families recruited for Born in Bradford are socioeconomically and ethnically diverse. 49 and and 40 percent of the BiB cohort is comprised of South Asian and White British families. Based on Index of Multiple Deprivation (IMD) scores, over half (7904 families, 65.4% ) of the families recruited for BiB are in the bottom fifth of deprivation for

England and Wales. Socioeconomic status in Bradford is unevenly distributed across ethnic groups; in the BiB cohort, the White British families could be split into wealthy and non-wealthy categories, but no such distinction could be made for the South Asian families, as there were not enough families in the wealthy category.

For INGENIOUS WP2 households recruited (321 households), 144 households had a BiB-cohort child within that household who was recorded as having an active asthma diagnosis in their primary healthcare records (45%). This is higher than the rates of childhood asthma in both the BiB cohort (in which incidence of childhood asthma is at 14%, (n = 1,807 out of N = 13,044 children)) but the study design aimed to recruit about half of participant families to have a child with asthma in order to better investigate the links between indoor air pollutant exposure and the prevalence of childhood asthma in the Bradford community.

Recruitment was stratified based on child ethnicity (White British, South Asian, or Other), the family's housing tenure (privately owned/mortgaged or rented), and child's asthmatic status (the presence of an active asthma diagnosis recorded in primary care within the past two years).

**Table 5:** Demographics of INGENIOUS cohort

| | | Ethnicity | | |
|---|---|---|---|---|
| | | **South Asian** (45%) | **White (British)** (45%) | **Other** (10%) |
| **Housing tenure** | **Private/mortgaged property** (70%) | N=95 (asthma: N=48; non-asthma: N=47) | N=95 (asthma: N=48; non-asthma: N=47) | N=21 (asthma: N=10; non-asthma: N=11) |
| | **Rented property** (30%) | N=40 (asthma: N=20; non-asthma: N=20) | N=40 (asthma: N=20; non-asthma: N=20) | N=9 (asthma: N=5; non-asthma: N=4) |

### 3.1.2 Average $PM_{2.5}$ Concentration Data Collection

Average $PM_{2.5}$ concentrations were obtained via a collaboration with another INGENIOUS project partner; in this instance, the data obtained resulted from sampling conducted by Dr. Evangelia Chatzidiakou from the University of Cambridge, and was also a part of WP2.

AirGradient ONE (Model I-9PSL) samplers were deployed in participants' homes for a period of 14 days. These are commercially available low-cost samplers, which integrate multiple low-cost environmental monitoring components. These sensors recorded indoor environmental parameters, including particulate matter concentrations ($PM_{1.0}$, $PM_{2.5}$ and $PM_{10.0}$ in $\mu g/m^3$), temperature (°C), relative humidity (%), carbon dioxide (ppm), and total volatile organic compounds (ppb), at five-minute intervals. The analysis conducted for this thesis focuses specifically on $PM_{2.5}$ measurements. Data were transmitted via a secure cellular connection managed by the research team, enabling continuous remote monitoring. Routine calibration and quality assurance procedures were conducted throughout the sampling period. In the event of connectivity issues, participants were contacted promptly in order to minimise data losses. Start and end times for each MiniVol TAS sampling session were recorded, and the corresponding AirGradient data were filtered based on both date and timestamp to ensure temporal alignment between the two datasets. This filtering process ensured that comparisons between sampler outputs reflected the same sampling intervals.

### 3.1.3 Quantitation of Target Analytes

The calibration method for quantifying the target analytes in solution is detailed in the previous chapter. This section outlines the subsequent step, which was the conversion of analyte concentrations from solution (µg/mL) to corresponding concentrations in air ($ng/m^3$), enabling comparison with established indoor air quality thresholds and exposure guidelines.

Firstly, each target analyte was paired with one of the compounds in the internal standard mixture (EPA 8270 Semivolatile Internal Standard Reference Mix, Supelco. The selection was based on which internal standard compound resulted in the most positive effect on each analyte's correlation coefficient, thus correcting for slight differences in volume.

The ChromaTOF software's quantitation function was then used to determine the concentration of each target analyte in the extraction solution (EtOAC), in µg/mL.

Lastly, the concentrations in solution were converted to airborne concentrations using Equation Equation 1: Concentration in Air Equation, which accounts for the volume of air sampled and the volume of solvent used during the extraction process. This conversion allowed for expression of results in units of $mg/m^3$, which was later converted to $ng/m^3$, the standard units for airborne pollutant concentration.

$$Conc.in\ air = \frac{conc.in\ solution\ (\frac{\mu g}{ml}) \times \frac{1\ mg}{1000\mu g} \times 1.7ml \times \frac{1000L}{1m^3}}{total\ air\ volume\ (L)}$$

(Equation 1: Concentration in Air Equation)

## 3.2 Results

### 3.2.1 Chromatography

A key advantage of GC×GC–ToF-MS is its ability to separate overlapping peaks. Even when analytes co-elute, their $m/z$ values can still be isolated and attributed to different compounds. This is particularly valuable in environmental and indoor air chemistry, where mixtures may contain structurally similar compounds such as polycyclic aromatic hydrocarbons (PAHs) or phthalates.

This section will demonstrate the use of GC×GC–ToF-MS in real-home environments, comparing different homes and showing how the use of extracted ion chromatograms can provide more specialised insight into different compound classes. It is important to note that for the purposes of maintaining participant anonymity, each INGENIOUS/BiB household was given a York ID number, which will be used throughout this thesis to describe specific households.

Figure 35 presents a comparison of three GC×GC–ToF-MS total ion chromatograms. 35a shows a household sample (York ID=29), 35b depicts an example of a blank filter, and 35c illustrates the resulting blank-subtracted chromatogram as processed in the software. In terms of the colour scale of the contour plot, all peaks are automatically scaled to the highest peak.

**(a)** Total ion 2D Plot of York ID=29, no blank adjustments made.




**(b)** Total ion 2D plot of a field blank filter. The presence of peaks indicates background contamination originating from the filter material, handling, or the analysis. These features must be accounted for to avoid overestimation of analyte concentrations in samples.




**(c)** Blank-subtracted 2D plot for York ID = 29. Subtraction removes background contamination observed in the blank, allowing clearer visualisation of compounds. Peaks remaining after subtraction represent analytes attributable to the sample, while reduced or absent features indicate contributions from background contamination.

**Figure 35:** Figure showing blank subtraction of a household sample (York ID=29).

An important step in the analysis was the use of blank subtraction; filters and samplers can introduce background signals originating from contaminants present in the material of the filters, or the handling and transport of the samples themselves. By comparing the extracted household samples to blank filters that have undergone the same extraction procedure, it is possible to identify and remove peaks and, later in the analysis process, concentrations of analytes which are attributable to these sources. This process ensures that compound identification and quantification reflect the real-home environments rather than contamination or artifacts introduced by the sampling and/or analytical process. As such, blank subtraction strengthens the validity of the results, particularly when assessing the prevalence of low-concentration compounds within the household environments.

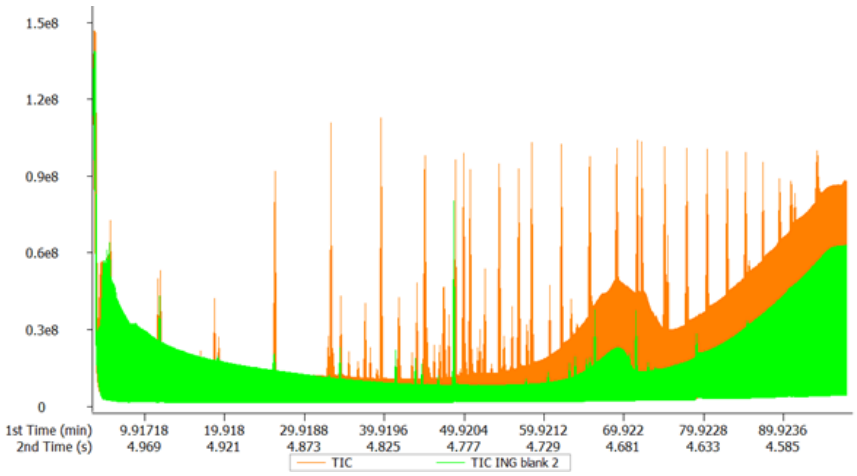



**Figure 36:** Total ion chromatogram showing York ID=29 compared to blank filter. The green represents the blank filter, and the orange represents the real home

Figure 36 presents the total ion chromatograms (TICs) of a household sample (York ID=29, shown in orange) and the corresponding filter blank (shown in green), overlaid for direct comparison. This figure highlights the complex background from the blank sample, which is primarily associated with solvent impurities and column bleed that is often present in GC×GC analysis, and is amplified by the modulator compared to standard 1-dimensional chromatography. By comparing these chromatograms, it becomes possible to distinguish household-derived chemical signals from the background signal visually.

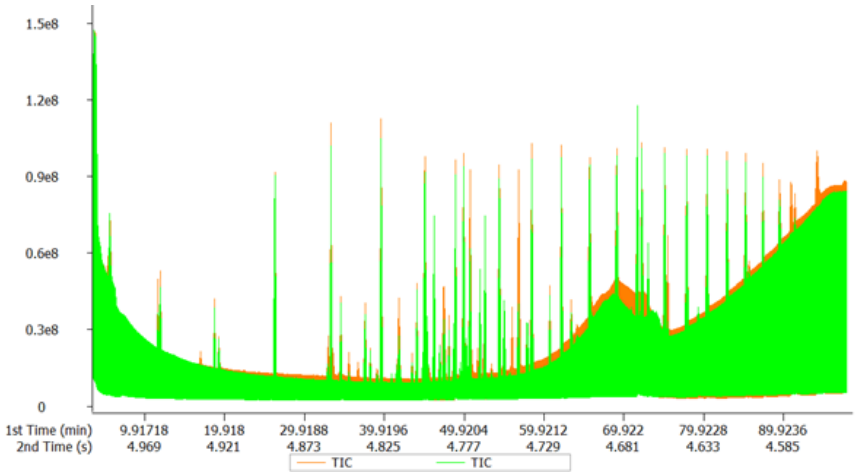



**Figure 37:** Total ion chromatogram comparing two households

Figure 35c shows the TICs of two different households. Differences can be observed in both the location and the height of the peaks, reflecting variation in the chemical composition of $PM_{2.5}$ between households.

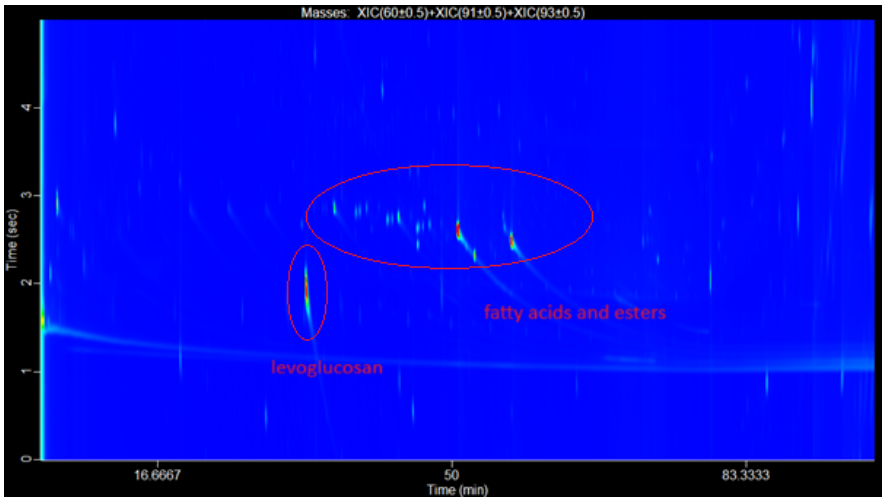
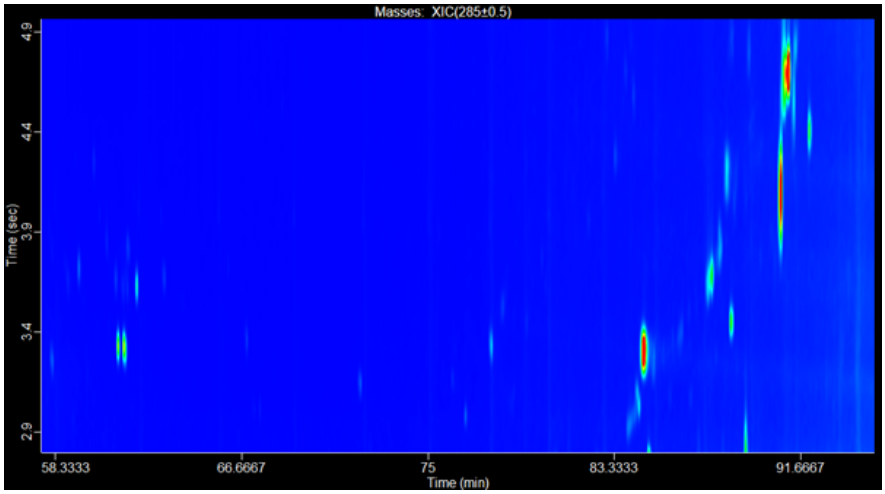


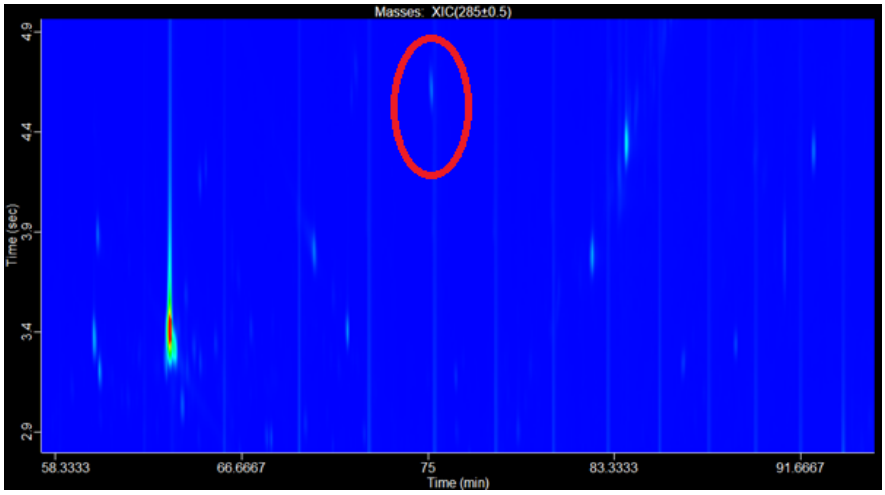

**Figure 38:** Extracted ion 2D plot of York ID=29, targeting fatty acids

In Figure 38, the two-dimensional chromatogram has been filtered to display only masses $m/z$ 60, 91, and 93. This selective filtering was applied to target three major compound classes of interest: fatty acids, aromatics, and terpenes. The circled regions highlight specific compounds and compound families, including levoglucosan, which is a well-known biomass burning marker, and a cluster of fatty acids and esters. These compounds are likely to be a result of the degradation of the trigylcerides and from free fatty acids found in cooking oils used during the preparation of meals within the kitchen.

This approach demonstrates how the use of extracted ion chromatograms allows for the identification of chemically distinct groups within complex indoor air samples, providing more nuanced insights compared to total ion chromatograms alone.




**(a)** York ID=29, no piperine




**(b)** York ID=7, piperine circled

**Figure 39:** Side-by-side comparison of extracted ion 2D plots at mass 285, targeting piperine in different samples.

Piperine ($C_{17}H_{19}NO_3$) is the active compound in black pepper which gives it its distinct flavour. Its presence in indoor air therefore serves as a chemical marker for culinary practices in the household (York ID=7) shown in Figure 39b. By filtering the data at a specific mass-to-charge ratio, in this case $m/z$=285 (its molecular weight), and by comparing the mass spectra obtained in each of the peaks against the NIST database EI mass spectrum of piperine, it was possible to selectively isolate the piperine signal in the 2D plots. This targeted

filtering revealed that one household (York ID = 7) displayed a clear and pronounced piperine peak, showing that pepper was used in cooking activities that occurred during the sampling period. This was not the only household that had a peak for piperine; these two households were chosen merely as an example of the differences visible in the 2D contour plots. The detection of piperine through this method not only highlights the utility of GC×GC–ToF-MS for pinpointing source-specific compounds in complex indoor air mixtures, but also provides direct evidence of lifestyle-related contributions to indoor PM profiles.

In Figure 40, the two-dimensional chromatogram has been filtered to highlight the masses $m/z$ 202, 252, and 278, which correspond to several polycyclic aromatic hydrocarbons (PAHs). These masses are characteristic for mid- to high-molecular-weight PAHs, making them useful for targeted visualization against the complex background of indoor air samples. The labeled peaks indicate specific PAHs of interest, many of which are recognized priority pollutants. This compound class is of particular concern because many PAHs are recognised carcinogens, and will be discussed further in Chapter 4. The continuous horizontal "smear" is attributed to background signal, likely arising from column bleed, and is a common feature in GC×GC plots.

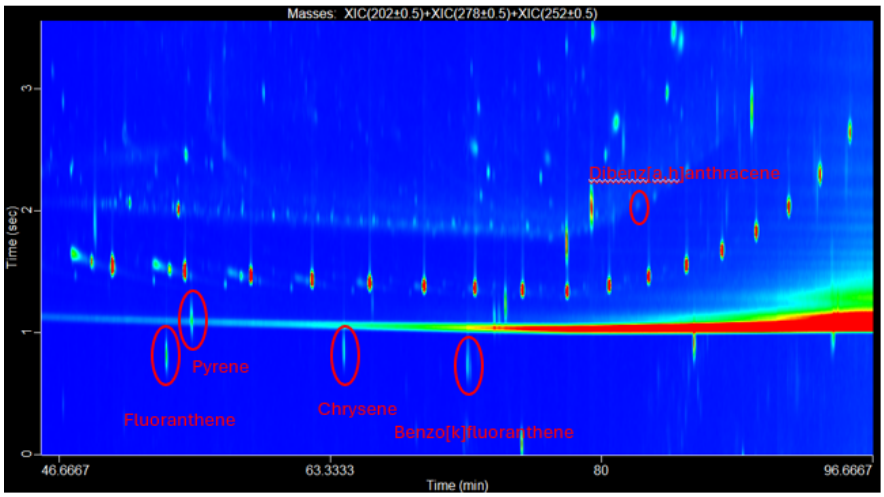



**Figure 40:** Extracted ion 2D plot of York ID=18 for masses 202, 253, and 278, targeting PAHs

Similarly, in Figure 41 below, the two-dimensional chromatogram has been filtered to highlight the masse $m/z$ 149, which targets phthalates. Peaks of interest have been circled and labeled in the figure to indicate the presence of several phthalates. These compounds are of particular concern due to their widespread use as plasticizers, their tendency to leach into indoor environments from consumer products and building materials, and their well-documented endocrine-disrupting properties [125]. By filtering with $m/z$ 149, the analysis

highlights both the sensitivity of the technique and the importance of phthalates as a class of indoor contaminants warranting close attention in exposure assessments.

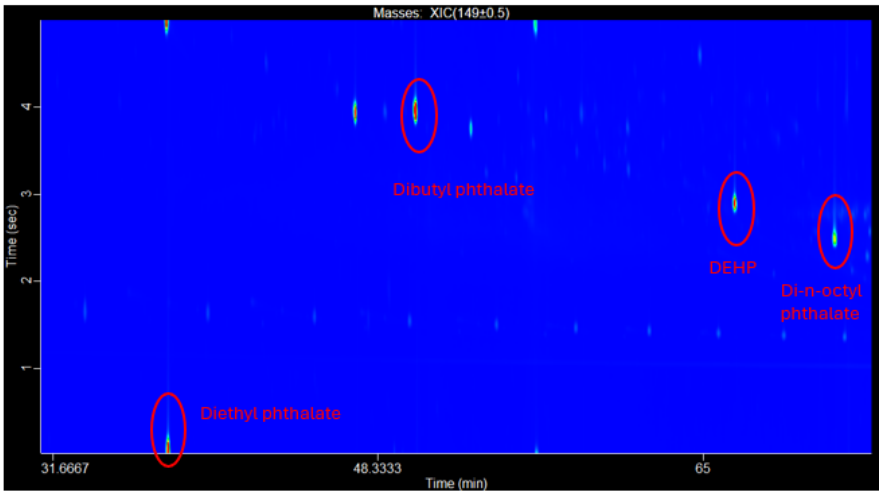



**Figure 41:** Extracted ion 2D plot of York ID=18 for masses 149, targeting phthalates

Overall, this section shows the utility of GC×GC–ToF-MS for the separation and mass-selective filtering of compounds to investigate indoor air chemistry in real-home environments. By utilizing blank subtraction, it was possible to remove background signals from filters, thereby ensuring that compound identifications and quantitations were representative of true household exposures. The use of extracted ion chromatograms provided a targeted means of isolating specific compound classes and source-specific compounds. The volume of data that the GC×GC produces provides a wealth of information about the chemical composition of the $PM_{2.5}$ in the homes. The previous examples highlight the impact of occupant activities on PM composition. However, a full non-target analysis of the samples is beyond the scope of this thesis. Instead, the following sections outline a more targeted approach, concentrating on compounds with known sources or health risks. Future work could expand the non-target analysis; the GC×GC data here will be stored and could act as a virtual composition archive to be re-examined in future studies. These targeted approaches allow for compound identification within complex mixtures as well as providing insight into behavioural differences between households.

### 3.2.2 Concentrations of Target Analytes

To provide an overview of the concentration ranges of all target analytes measured during this study, Figure 42 presents a series of box plots. Each box plot summarises the distribution

of measured concentrations for a given compound, plotted on a logarithmic scale as there was a large variability in values.

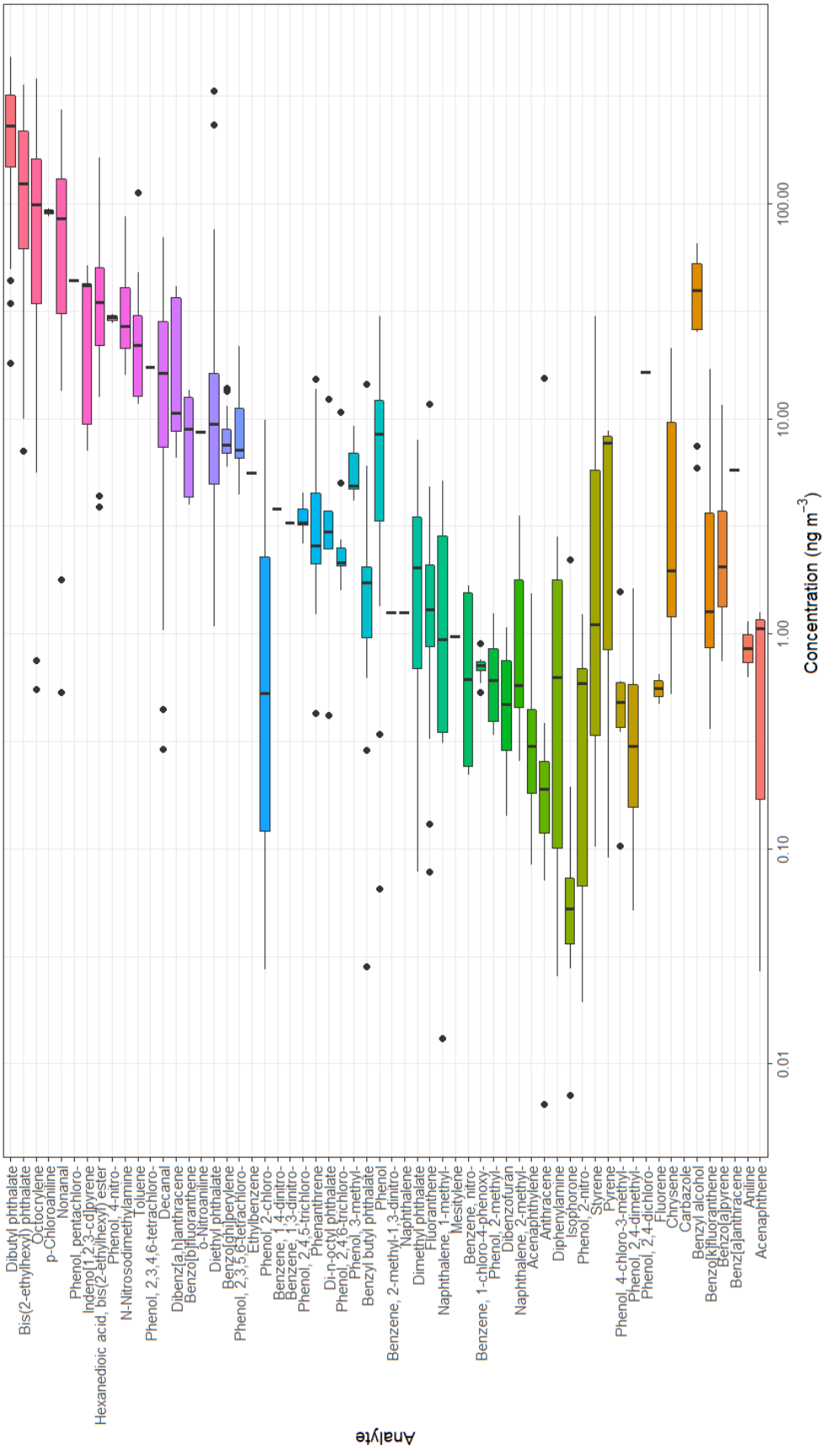



**Figure 42:** Boxplot of concentrations of all analytes found in homes, ordered by median (descending)

Table 6 provides summary statistics of all the target anayltes that were found in homes. It should be noted that all alkanes and cholesterol were excluded from analysis due to extremely improbable results from the quantitation analysis. For the alkanes, this was potentially caused by contamination of the filters from the vacuum grease used for sampler maintenance. The reasons for the improbable cholesterol results remain unknown.

**Table 6:** Summary statistics of compounds detected in ING homes

| Compound | Mean | Median | Min | Max | SD | Homes detected |
|---|---|---|---|---|---|---|
| Acenaphthene | 0.2 | 0.0 | 0.0 | 1.3 | 0.5 | 10 |
| Acenaphthylene | 0.4 | 0.3 | 0.1 | 1.6 | 0.4 | 21 |
| Aniline | 0.1 | 0.0 | 0.0 | 1.1 | 0.4 | 13 |
| Anthracene | 0.8 | 0.1 | 0.0 | 15.4 | 3.1 | 24 |
| Benz[a]anthracene | 0.6 | 0.0 | 0.0 | 5.8 | 1.8 | 10 |
| Benzene, 1,3-dinitro- | 3.3 | 3.3 | 3.3 | 3.3 | NA | 1 |
| Benzene, 1,4-dinitro- | 3.8 | 3.8 | 3.8 | 3.8 | NA | 1 |
| Benzene, 1-chloro-4-phenoxy- | 0.7 | 0.7 | 0.5 | 0.9 | 0.1 | 14 |
| Benzene, 2-methyl-1,3-dinitro- | 1.3 | 1.3 | 1.3 | 1.3 | NA | 1 |
| Benzene, nitro- | 0.9 | 0.9 | 0.2 | 1.7 | 0.8 | 4 |
| Benzo[a]pyrene | 0.5 | 0.0 | 0.0 | 11.7 | 2.1 | 32 |
| Benzo[b]fluoranthene | 8.7 | 8.9 | 4.0 | 13.7 | 4.5 | 5 |
| Benzo[ghi]perylene | 8.4 | 7.5 | 6.0 | 13.9 | 2.2 | 20 |
| Benzo[k]fluoranthene | 0.8 | 0.0 | 0.0 | 17.1 | 3.1 | 31 |
| Benzyl alcohol | 15.0 | 0.0 | 0.0 | 66.2 | 22.9 | 25 |
| Benzyl butyl phthalate | 2.5 | 1.6 | 0.0 | 14.4 | 3.6 | 16 |
| Bis(2-ethylhexyl) phthalate | 129.0 | 104.8 | 0.0 | 358.3 | 111.2 | 29 |
| Carbazole | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 25 |
| Chrysene | 2.1 | 0.0 | 0.0 | 21.5 | 5.4 | 29 |
| Decanal | 19.6 | 15.8 | 0.0 | 70.1 | 17.4 | 28 |
| Di-n-octyl phthalate | 2.8 | 2.5 | 0.0 | 12.3 | 3.9 | 9 |

**Table 6:** Summary statistics of targeted analytes *(continued)*

| Compound | Mean | Median | Min | Max | SD | Homes detected |
|---|---|---|---|---|---|---|
| Dibenz[a,h]anthracene | 18.9 | 10.6 | 6.6 | 41.8 | 13.4 | 32 |
| Dibenzofuran | 0.5 | 0.5 | 0.1 | 1.1 | 0.3 | 12 |
| Dibutyl phthalate | 233.3 | 228.6 | 18.1 | 483.8 | 126.1 | 34 |
| Diethyl phthalate | 29.2 | 7.8 | 0.0 | 334.6 | 68.8 | 33 |
| Dimethyl phthalate | 1.9 | 1.3 | 0.0 | 8.0 | 2.1 | 28 |
| Diphenylamine | 0.6 | 0.1 | 0.0 | 2.8 | 0.9 | 32 |
| Ethylbenzene | 5.6 | 5.6 | 5.6 | 5.6 | NA | 1 |
| Fluoranthene | 1.6 | 1.2 | 0.0 | 11.6 | 2.2 | 32 |
| Fluorene | 0.1 | 0.0 | 0.0 | 0.7 | 0.3 | 9 |
| Hexanedioic acid, bis(2-ethylhexyl) ester | 39.4 | 34.7 | 3.9 | 164.6 | 29.6 | 30 |
| Indeno[1,2,3-cd]pyrene | 30.7 | 41.8 | 7.1 | 51.9 | 17.7 | 14 |
| Isophorone | 0.3 | 0.1 | 0.0 | 2.2 | 0.7 | 9 |
| Mesitylene | 1.0 | 1.0 | 1.0 | 1.0 | NA | 1 |
| N-Nitrosodimethylamine | 39.5 | 27.3 | 16.0 | 87.5 | 32.6 | 4 |
| Naphthalene | 1.3 | 1.3 | 1.3 | 1.3 | NA | 1 |
| Naphthalene, 1-methyl- | 1.9 | 1.1 | 0.0 | 5.2 | 2.0 | 6 |
| Naphthalene, 2-methyl- | 0.9 | 0.5 | 0.0 | 3.6 | 1.1 | 13 |
| Nonanal | 85.5 | 83.5 | 0.0 | 273.8 | 66.4 | 27 |
| Octocrylene | 120.2 | 92.7 | 0.0 | 382.2 | 117.5 | 32 |
| Phenanthrene | 3.8 | 2.5 | 0.0 | 15.2 | 3.5 | 30 |
| Phenol | 5.7 | 1.4 | 0.0 | 30.3 | 7.9 | 27 |
| Phenol, 2,3,4,6-tetrachloro- | 17.4 | 17.4 | 17.4 | 17.4 | NA | 1 |

**Table 6:** Summary statistics of targeted analytes *(continued)*

| Compound | Mean | Median | Min | Max | SD | Homes detected |
|---|---|---|---|---|---|---|
| Phenol, 2,3,5,6-tetrachloro- | 10.6 | 7.1 | 4.5 | 22.0 | 7.1 | 8 |
| Phenol, 2,4,5-trichloro- | 3.5 | 3.3 | 2.6 | 4.6 | 0.7 | 5 |
| Phenol, 2,4,6-trichloro- | 3.2 | 2.1 | 1.6 | 10.7 | 2.7 | 11 |
| Phenol, 2,4-dichloro- | 1.2 | 0.0 | 0.0 | 16.4 | 4.4 | 14 |
| Phenol, 2,4-dimethyl- | 0.2 | 0.0 | 0.0 | 1.6 | 0.4 | 22 |
| Phenol, 2-chloro- | 5.0 | 5.0 | 0.0 | 10.0 | 7.0 | 2 |
| Phenol, 2-methyl- | 0.7 | 0.6 | 0.3 | 1.2 | 0.3 | 9 |
| Phenol, 2-nitro- | 0.3 | 0.0 | 0.0 | 1.2 | 0.4 | 15 |
| Phenol, 3-methyl- | 3.0 | 2.1 | 0.0 | 9.4 | 3.3 | 18 |
| Phenol, 4-chloro-3-methyl- | 0.2 | 0.0 | 0.0 | 1.6 | 0.4 | 15 |
| Phenol, 4-nitro- | 29.4 | 29.4 | 28.1 | 30.8 | 1.4 | 3 |
| Phenol, pentachloro- | 43.9 | 43.9 | 43.9 | 43.9 | NA | 1 |
| Pyrene | 0.8 | 0.0 | 0.0 | 8.9 | 2.6 | 20 |
| Styrene | 3.9 | 0.0 | 0.0 | 30.3 | 10.7 | 8 |
| Toluene | 29.7 | 20.9 | 0.0 | 112.4 | 33.7 | 9 |
| o-Nitroaniline | 8.7 | 8.7 | 8.7 | 8.7 | NA | 1 |
| p-Chloroaniline | 91.6 | 91.6 | 87.6 | 95.6 | 5.6 | 2 |

Figure 43 shows all the target analytes separated into their compound groups.

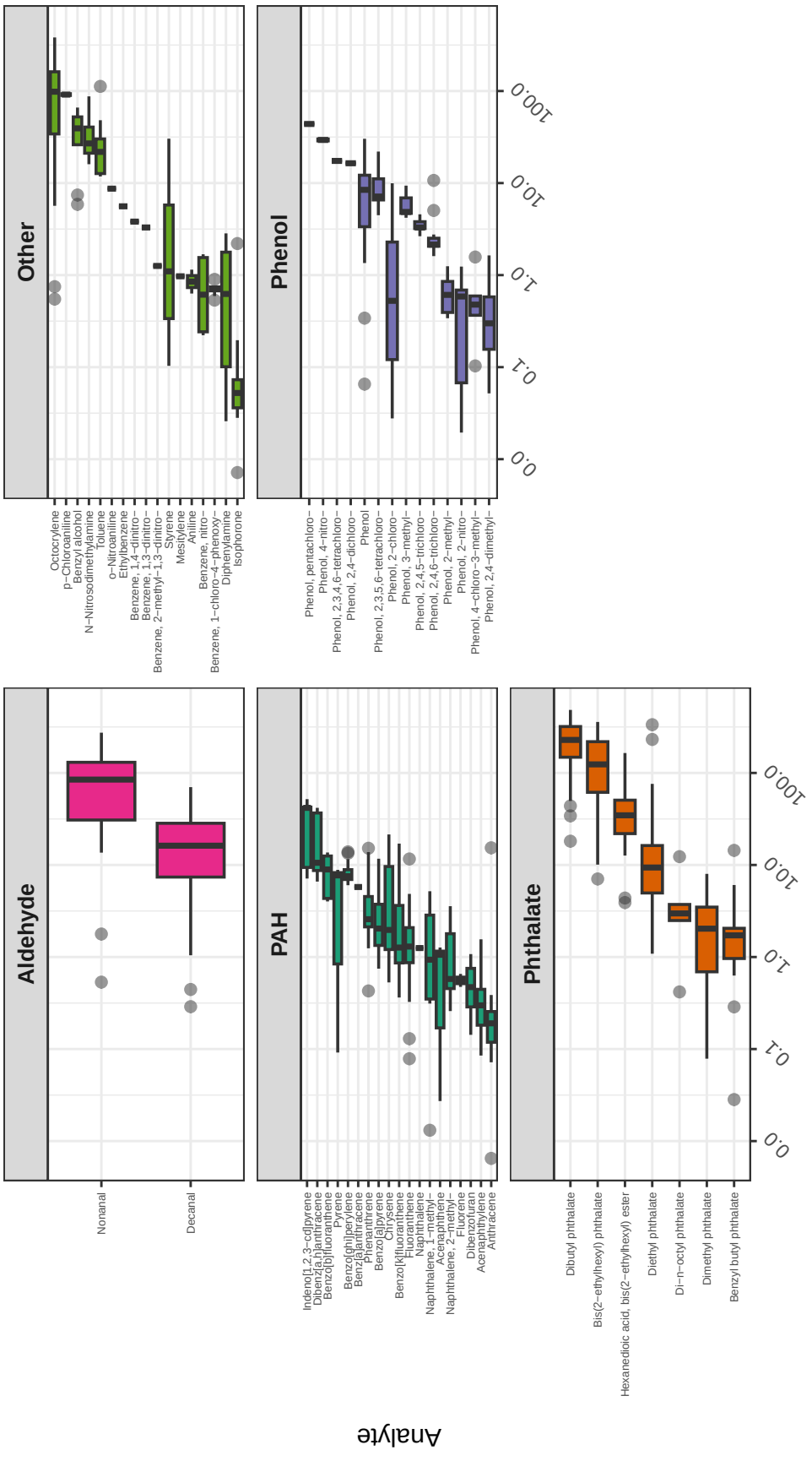



**Figure 43:** Concentration distributions of all target analytes found in homes, faceted by group

Of the compound groups presented in the above figure, PAHs and phthalates will be discussed in greater depth in Chapter 4.

Phenols were selected as target analytes due to their wide range of sources in indoor environments and their potential health effects. Sources in indoor environments include cigarette smoking as well as the degradation of building materials[126, 127]. Amongst the targeted phenols, pentachlorophenol exhibited a high concentration, but was only present in a single housheold. It is a persistent organic pollutant banned under the Stockholm Convention, and was used as a pesticide or disinfectant[128]. In contrast, 2,4-dimethylphenol was among the least abundant compounds but was detected in 22 households, its ubiquitousness suggesting a combustion source.

The aldehydes nonanal and decanal were added to the targeted analytes after observations that they were present in all homes sampled in up to that point. Nonanal and decanal are commonly released during cooking processes; as such, they were added to serve as cooking tracers and to possibly compare the frequency of cooking activities across homes[129]. Nonanal and decanal were both detected across a large number of homes (nonanal n=28, decanal n=28); nonanal exhibited substantially greater variability, with concentrations ranging from 0 to 274 ng $m^{-3}$ compared to 0 to 70 ng $m^{-3}$ for decanal. The higher mean concentration and larger standard deviation observed for nonanal suggest greater heterogeneity in emissions across households.

Lastly, particular attention must be brought to octocrylene. Octocrylene is a compound found primarily in sunscreens, and serves as a UV filter in personal care products[130]. Its detection in nearly all sampled households (n = 32) was unexpected; further literature review revealed that indoor air is a major exposure pathway for organic UV filters. In addition, these compounds have been linked to health effects including endocrine dysfunction, diabestes, and female infertility[131]. Its presence in nearly all homes highlights the pervasive nature of consumer product emissions on indoor air composition.

The statistical summary and the boxplots provide a comprehensive overview of the occurrence and distribution of the targeted compounds across the study homes. The table presents the data, while the visualizations highlight the highly variable nature of many of the distributions. This combined approach illustrates not only which analytes were most frequently detected, but also the degree of variability in exposure between homes. Such variation highlights the complex nature of indoor environments, where both ubiquitous and sporadically

found compounds can contribute to cumulative exposure and its associated health risks.

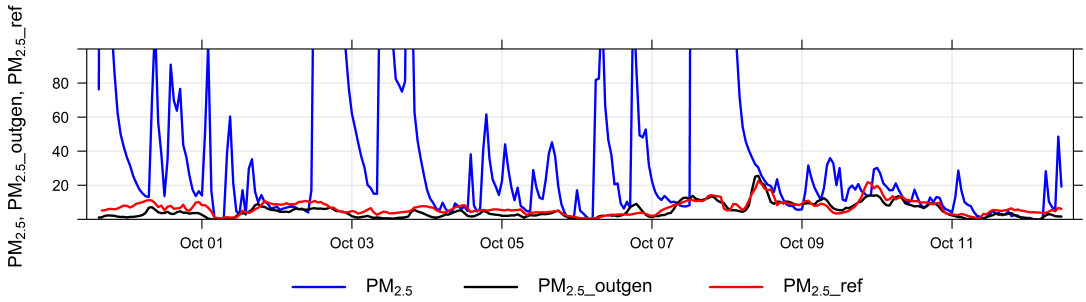
### 3.2.3 $PM_{2.5}$ Analysis: AirGradient Measurements

For the purposes of this study, data collected by the AirGradient samplers were filtered to include only the time periods during which the MiniVol TAS samplers were concurrently deployed in the participating households. Thus, they cannot be directly compared to the recent and upcoming publications on INGENIOUS that use the full dataset. The purpose of this analysis is to allow comparison of the trends in the households where filters were also collected and the results presented here.

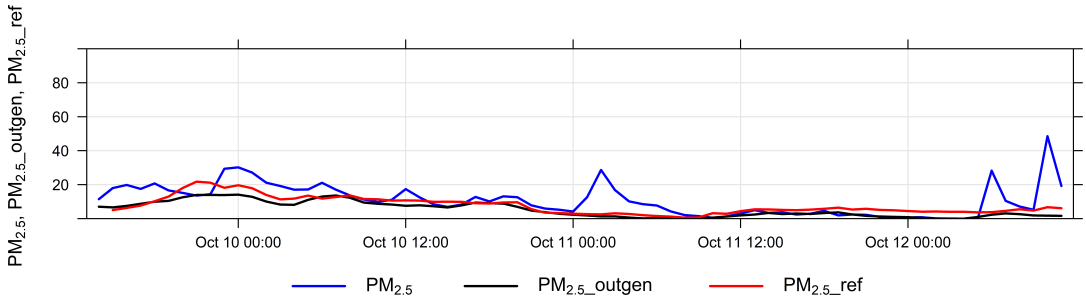
#### Time series plots from AirGradient Sensors

Figures 44, 45, and 46 below are time series plots of $PM_{2.5}$ concentration readings over a) the two-week sampling period that the AirGradient sensors were deployed in homes and b) the three day sampling period where the Minivol TAS was also deployed to collect filter samples. These are shown here in order to illustrate the natural variability that occurs both between different households and between different time periods within the same home. All plots were obtained courtesy of Evangelia Chatzidiakou of the University of Cambridge.

In these figures, the measured $PM_{2.5}$ concentration measurements are indicated by the blue line. The black lines show an estimate of outdoor generated $PM_{2.5}$ and the red shows outdoor $PM_{2.5}$ measured at a background urban reference station. The estimate of outdoor generated $PM_{2.5}$ was obtained by calculating ventilation rates from $CO_2$, calculating PM loss ratio from the indoor/outdoor ratio when indoor concentrations are at a steady state, then using outdoor background, the ventilation rate constant (kvent), and the local loss coefficient (kloss) to solve a mass balance equation. Anything below this line was classified as outdoor generated; this was subtracted from the total measured PM in order to obtain the indoor measurements. The following discussion will focus on the indoor (blue) measurements.



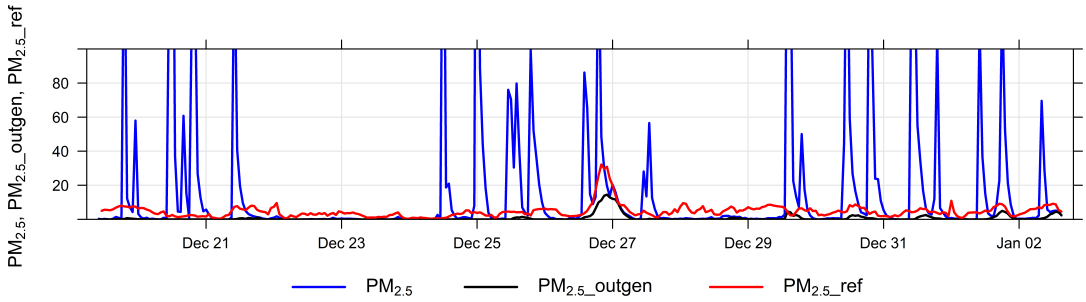

**(a)** Two-week time series of average $PM_{2.5}$ concentrations



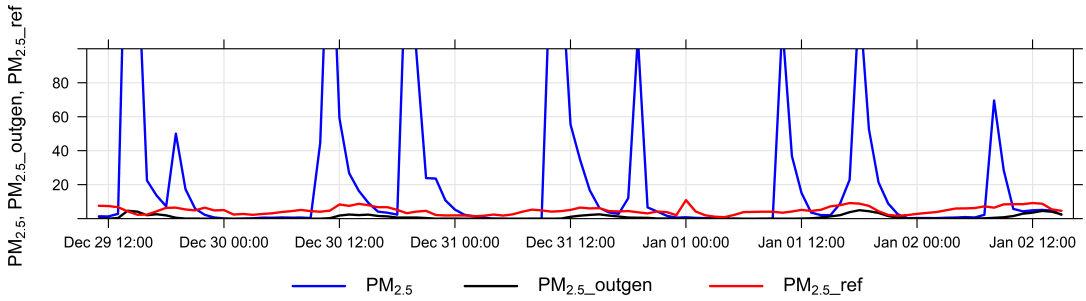

**(b)** Three-day sampling period of average $PM_{2.5}$, when Minivol TAS also deployed

**Figure 44:** Comparison of $PM_{2.5}$ time series over (a) two weeks and (b) three-day sampling period when filter samples were also being taken for York ID=5.

As an example, Figure 44 above shows a two-week sampling period characterised by frequent high spikes and substantial variability in PM2.5 concentrations. However, the corresponding three-day sampling period exhibits relatively low PM2.5 levels, without the high spikes. This underscores the considerable variability and the impact of household activities on the measured PM2.5 depending on the temporal window during which sampling is conducted.



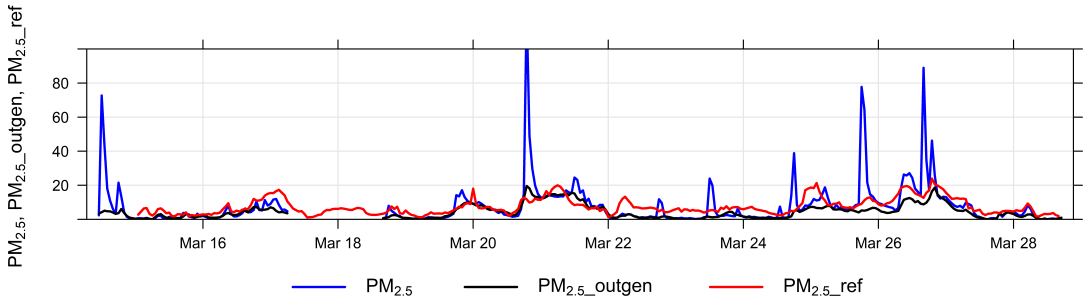

**(a)** Two-week time series of $PM_{2.5}$ concentrations



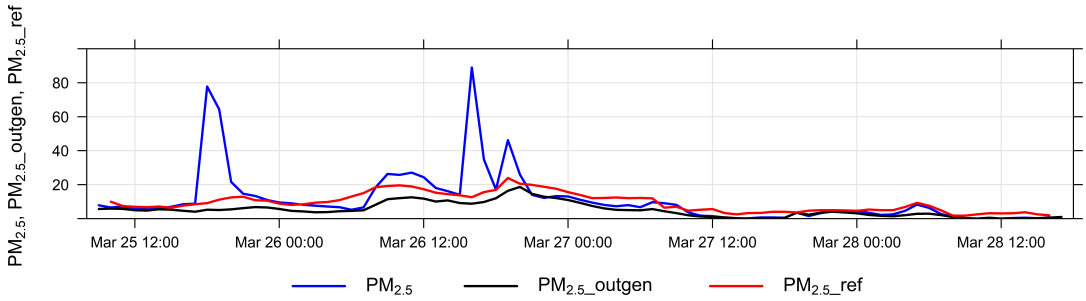

**(b)** Three-day sampling period of $PM_{2.5}$, when Minivol TAS also deployed

**Figure 45:** Comparison of $PM_{2.5}$ time series over (a) two weeks and (b) three-day sampling period when filter samples were also being taken for York ID=16.

In contrast, Figure 45 illustrates a two-week sampling period characterized by sharp, high-concentration spikes, which indicate intermittent activities that generated substantial emissions. It is hypothesised that this is a home where cooking occurs on a near-everyday basis, as the spikes occur multiple times on days where they are found; in contrast, Figure 44 shows far more random timings in terms of spikes. These episodes are interspersed with periods of very low concentrations, some of which were quite prolonged, perhaps indicating days where residents dined out or went on holiday. Notably, the sampling for this home took place over the Christmas and New Year period, and the spikes occur around the holiday dates, likely reflecting periods of increased activity.




**(a)** Two-week time series of $PM_{2.5}$ concentrations




**(b)** Three-day sampling period of $PM_{2.5}$, when Minivol TAS also deployed

**Figure 46:** Comparison of $PM_{2.5}$ time series over (a) two weeks and (b) three-day sampling period when filter samples were also being taken for York ID=29.

Lastly, Figure 46 presents a household with generally low concentrations of $PM_{2.5}$. Only a small number of spikes are observed, occurring sporadically across the two-week sampling period. These spikes vary in amplitude and width, suggesting emission events of differing intensity and duration. The corresponding three-day sampling window reflects this overall pattern, with a few broader peaks interspersed with extended periods of very low concentrations. This indicates that for this household, particle-emitting activities were infrequent and not sustained.

Together, Figures 44, 45, and 46 highlight the substantial temporal variability in indoor $PM_{2.5}$ concentrations across households. The comparison between longer two-week measurements and shorter three-day periods underscores how the timing of sampler deployment influenced observed outcomes. While some households exhibit frequent and pronounced peaks associated with recurrent activities, others display a relatively clean baseline interrupted only by occasional events. These findings emphasize the point that short-term sampling may not fully capture the range of indoor exposures, and that temporal variability must be carefully considered.

Comparison of the chemical composition across York IDs 5, 16, and 29 indicates that while similar compound classes were detected, their relative abundances and profiles differed between households.

York ID=5 showed elevated concentrations of aldehydes such as nonanal, which was used as a cooking tracer. This suggests that indoor activities, particularly cooking, are likely to be a dominant source of emissions in this household, contributing to a relatively consistent background level of particulate matter. The corresponding time series data showed relatively few spikes, indicating that these emissions may occur at a low but consistent/sustained level rather than as distinct episodic events.

York ID=16 exhibited a more complex chemical profile, with both aldehydes and phthalates present at elevated levels. This indicates contributions from both indoor activities and material-based sources. The higher mean $PM_{2.5}$ concentration observed in this household is consistent with the presence of multiple emission sources and potentially more sustained or frequent emission events, which was also reflected in the time series data.

York ID=29 showed comparatively lower concentrations of these more reactive and source-specific compounds, including nonanal. This suggests that while similar emissions may occur, they are either less frequent or of lower intensity. This aligns with the lower mean $PM_{2.5}$ concentration observed and may reflect a household with fewer emission producing events. However, the time series data showed intermittent spikes, indicating that emissions in this household may be more sporadic in nature. These events may not be fully captured in the averaged chemical composition, resulting in a disconnect between the time-resolved and filter measurements.

Overall, these results suggest that differences between households are driven not only by the presence of similar emission sources, but by their relative intensity and frequency.

A summary of the data collected from the AirGradient samplers is shown in the table below. This table shows the minimum, maximum, mean $PM_{2.5}$ mass concentration and standard deviation of the sensor collected during the 3 days sampling period in each household. To approximate the mass loading on each of the filters collected by the Minivol TAS, the average $PM_{2.5}$ concentration (mean) was multiplied by the total volume of air sampled.

**Table 7:** Summary of $PM_{2.5}$ by York ID

| York ID | Start Date | End Date | Total Volume (l) | Mean ($\mu g/m^3$) | Min ($\mu g/m^3$) | Max ($\mu g/m^3$) | Std Dev ($\mu g/m^3$) | Est Mass Loading of $PM_{2.5}$ ($\mu g$) |
|---|---|---|---|---|---|---|---|---|
| 1 | 29/09/2023 | 02/10/2023 | 12996 | 13.7 | 0.0 | 398.0 | 31.5 | 177.8 |
| 2 | 02/10/2023 | 05/10/2023 | 12978 | 6.9 | 0.0 | 167.0 | 14.2 | 89.9 |
| 3 | 06/10/2023 | 09/10/2023 | 13842 | 33.9 | 1.0 | 738.0 | 95.5 | 469.7 |
| 4 | 09/10/2023 | 12/10/2023 | 12996 | 9.6 | 0.0 | 381.0 | 26.9 | 124.7 |
| 5 | 09/10/2023 | 12/10/2023 | 12492 | 10.8 | 0.0 | 253.0 | 13.4 | 134.7 |
| 6 | 20/10/2023 | 23/10/2023 | 12258 | 22.8 | 0.0 | 554.0 | 67.2 | 279.5 |
| 7 | 20/10/2023 | 23/10/2023 | 13104 | 36.6 | 0.0 | 938.0 | 83.7 | 479.5 |
| 8 | 20/10/2023 | 23/10/2023 | 13104 | 20.8 | 0.0 | 159.0 | 26.3 | 272.0 |
| 9 | 27/10/2023 | 30/10/2023 | 13158 | 51.3 | 0.0 | 677.5 | 65.3 | 674.9 |
| 10 | 27/10/2023 | 30/10/2023 | 13158 | 13.4 | 0.0 | 163.0 | 17.2 | 176.5 |
| 11 | 03/11/2023 | 06/11/2023 | 12996 | 10.8 | 0.0 | 316.0 | 19.5 | 140.8 |
| 12 | 07/11/2023 | 10/11/2023 | 12024 | 5.4 | 0.0 | 558.0 | 36.8 | 64.9 |
| 13 | 10/11/2023 | 13/11/2023 | 13014 | 38.9 | 0.0 | 831.5 | 93.9 | 506.6 |
| 14 | 24/11/2023 | 27/11/2023 | 13536 | 24.8 | 0.0 | 687.5 | 55.7 | 335.4 |
| 15 | 24/11/2023 | 27/11/2023 | 12978 | 33.3 | 0.0 | 951.0 | 86.5 | 431.8 |
| 16 | 29/12/2023 | 02/01/2024 | 18126 | 22.7 | 0.0 | 740.0 | 64.6 | 411.4 |
| 17 | 16/01/2024 | 19/01/2024 | 12960 | 25.9 | 0.0 | 964.0 | 89.2 | 335.2 |
| 18 | 19/01/2024 | 22/01/2024 | 13104 | 42.3 | 0.0 | 996.0 | 128.0 | 554.3 |
| 19 | 02/02/2024 | 05/02/2024 | 12996 | 19.2 | 0.0 | 920.0 | 64.0 | 250.1 |
| 20 | 09/02/2024 | 12/02/2024 | 12960 | 34.6 | 0.5 | 855.0 | 64.8 | 448.8 |
| 21 | 16/02/2024 | 19/02/2024 | 13040 | 8.4 | 0.0 | 200.0 | 18.6 | 109.1 |
| 22 | 01/03/2024 | 04/03/2024 | 12978 | 49.6 | 1.0 | 986.0 | 106.3 | 644.3 |
| 23 | 05/03/2024 | 08/03/2024 | 10404 | 44.7 | 11.0 | 586.0 | 48.9 | 465.5 |
| 24 | 12/03/2024 | 15/03/2024 | 14202 | 43.2 | 0.0 | 964.5 | 109.0 | 613.3 |
| 25 | 15/03/2024 | 18/03/2024 | 12996 | 8.3 | 0.0 | 168.0 | 16.9 | 108.5 |
| 26 | 19/03/2024 | 25/03/2024 | 12978 | 16.0 | 0.0 | 157.0 | 15.0 | 207.3 |
| 27 | 22/03/2024 | 25/03/2024 | 13626 | 4.0 | 0.0 | 20.0 | 4.9 | 54.7 |
| 28 | 22/03/2024 | 26/03/2024 | 15480 | 3.6 | 0.0 | 130.0 | 9.5 | 55.9 |
| 29 | 25/03/2024 | 28/03/2024 | 12960 | 11.4 | 0.0 | 379.0 | 23.7 | 147.3 |
| 30 | 25/03/2024 | 28/03/2024 | 12960 | 20.7 | 0.0 | 838.0 | 67.1 | 268.6 |
| 31 | 02/04/2024 | 05/04/2024 | 13014 | 11.7 | 0.0 | 182.5 | 24.2 | 151.6 |
| 32 | 19/04/2024 | 22/04/2024 | 12906 | 0.8 | 0.0 | 16.0 | 1.6 | 10.8 |
| 33 | 23/04/2024 | 26/04/2024 | 14004 | 2.5 | 0.0 | 270.0 | 9.8 | 34.6 |
| 34 | 23/04/2024 | 26/04/2024 | 12942 | 6.4 | 0.0 | 89.0 | 9.9 | 82.6 |

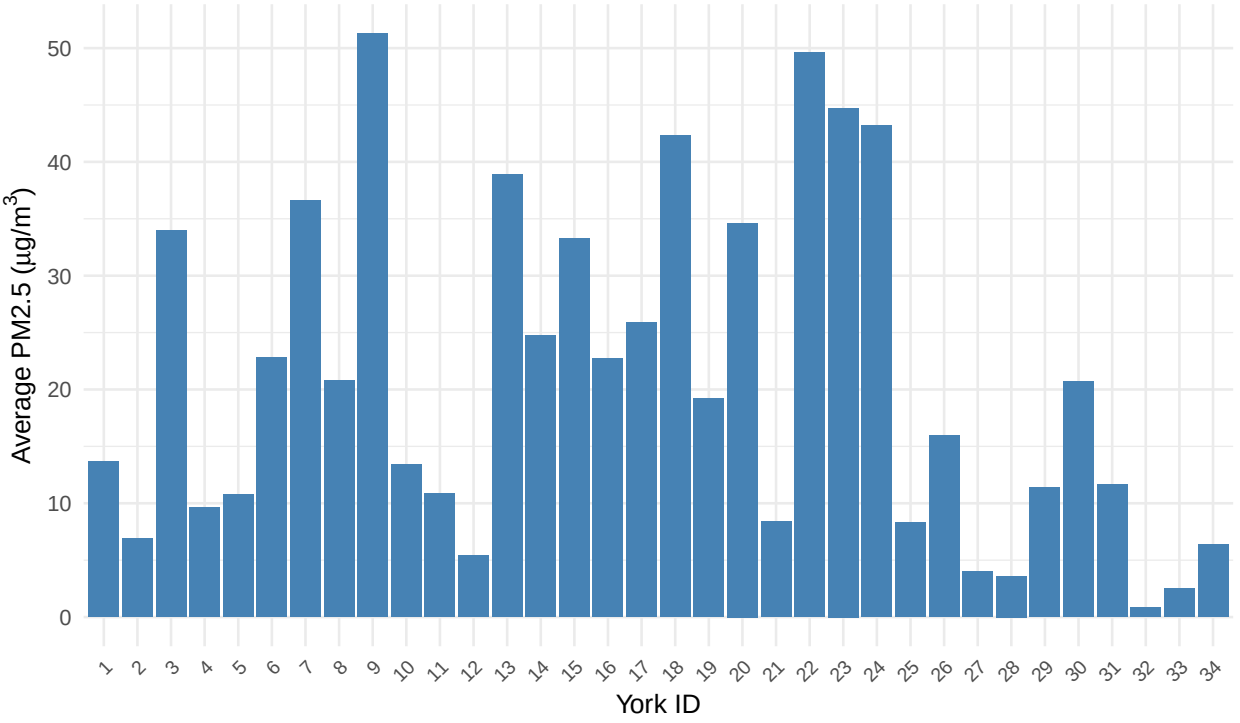



**Figure 47:** Bar plot showing average $PM_{2.5}$ concentrations across all households

Figure 47 comparing mean/average $PM_{2.5}$ concentrations across households shows that the distribution of values appears irregular and highly variable, with a range between 2.5 and 51 $\mu g/m^3$.

To investigate the trends in PM2.5 across the filter samples, the average PM2.5 data was compared to a number of different factors, including Index of Multiple Deprivation (IMD) group, housing tenure, smoking status, cooking fuels used in the home, and urban/rural designation.

The Index of Multiple Deprivation is used to measure relative deprivation in small geographical areas, and is produced by the Ministry of Housing, Communities and Local Government (MHCLG). The IMD integrates data across seven weighted domains of deprivation, reflecting a multidimensional understanding of social disadvantage:

- Income
- Employment
- Health Deprivation and Disability

- Education, skills, and training
- Crime
- Barriers to Housing and Services
- Living Environment

Each domain contributes to a cumulative deprivation score, which can then be ranked and segmented into deciles, in which Decile 1 represents the most deprived areas and Decile 10 the least deprived. This measure is frequently employed in public health and environmental justice research to explore how deprivation correlates with health outcomes, environmental exposures, and access to health services[132].

IMDs can thus be used to identify the areas which have the highest levels of deprivation. By stratifying areas based on IMDs, comparisons can be made to explore systemic differences in health amongst populations.

Figure 48 presents a boxplot comparing average $PM_{2.5}$ concentrations across households stratified by Index of Multiple Deprivation (IMD) group. The IMD is a widely used measure in the UK to assess relative levels of deprivation across small geographic areas, incorporating data across seven weighted domains: income, employment, health and disability, education and skills, crime, barriers to housing and services, and the living environment[132]. Each household's IMD group was identified based on its postcode.

The decision was made to combine IMD groups 1-3, 4-6, and 7-10 were combined to form three groups for analysis. The distribution of households across the stratified IMD groups is as follows:

- IMD Groups 1-3 (n=8)
- IMD Groups 4-6 (n=14)
- IMD Groups 7-10 (n=12)

Given the limited sample size of the study (n = 34) in contrast to the 10 IMD decile groups, the decision was made to consolidate the IMD groups into three broader categories to facilitate more meaningful analysis; these groups were IMD 1–3 (most deprived), IMD 4–6 (moderately deprived), and IMD 7–10 (least deprived). This stratification allowed for

a clearer comparison of $PM_{2.5}$ exposure across varying levels of deprivation while avoiding oversegmentation of the dataset. Further explanation of the distribution of households across IMD groups and a more in-depth explanation of IMD in general will be found in Section 4.8: Comparing LECR and Total PAH Across Demographic Groups.

Figure 48 suggests a potential trend in average $PM_{2.5}$ concentrations across different levels of deprivation.

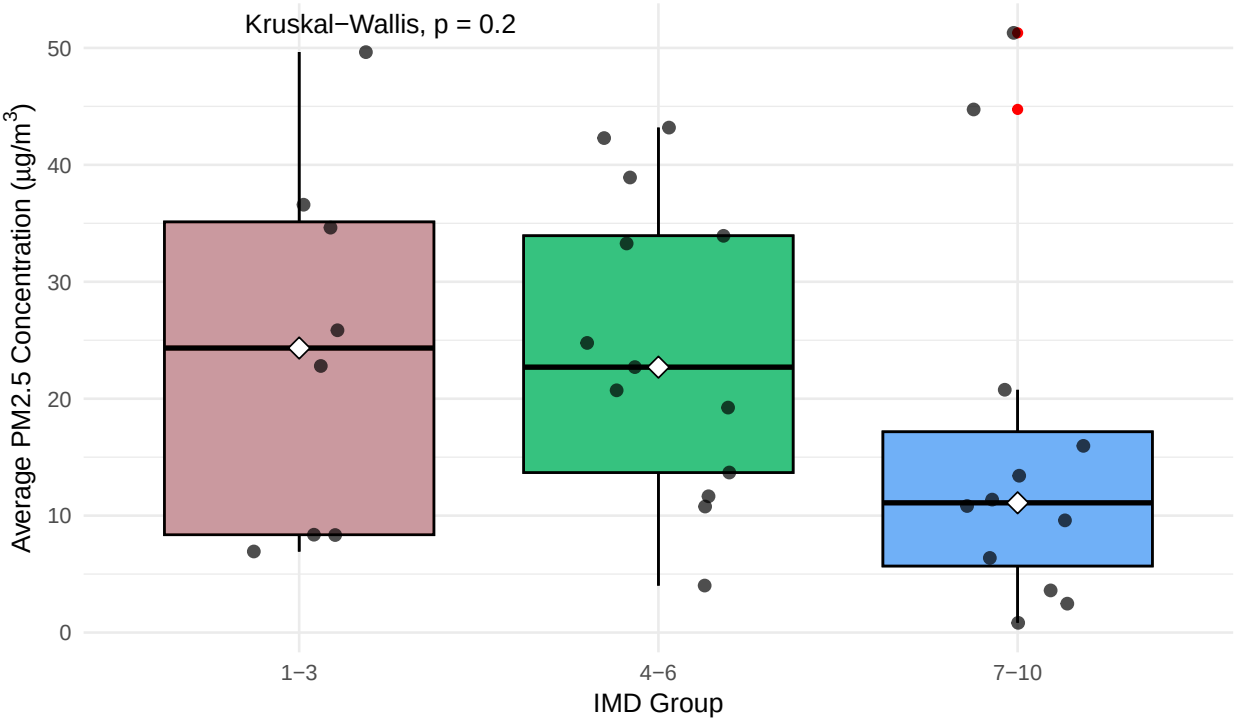



**Figure 48:** Boxplot showing average PM2.5 concentrations separated by IMD group

Specifically, there appears to be a gradual decrease in median $PM_{2.5}$ concentrations as levels of deprivation decrease. The median values for the first two IMD groups (1–3 and 4–6) are relatively close together and with similar interquartile ranges, indicating similar central tendencies and variability in PM exposure among the more deprived households. In contrast, the least deprived group (7–10) exhibits both a lower median and a narrower IQR, suggesting more consistent and reduced exposure to $PM_{2.5}$ within this group.

While these observations suggest a potential inverse association between socioeconomic status and PM2.5 exposure, the limited sample size within each IMD group limits the robustness of this analysis. No statistically significant difference in average PM2.5 was observed between IMD groups (Kruskal–Wallis test, $p = 0.2$), indicating no evidence of variation

across socioeconomic strata.Nevertheless, the pattern aligns with literature indicating that lower-deprivation households often benefit from environmental advantages, such as improved housing quality, more effective ventilation, and reduced proximity to pollution sources[101].

The households studied fell under three categories for housing tenure: Owned (includes mortgaged homes), Rented (from local council, housing association/trust or private landlord) and Other. The distribution of households across these groups was as follows:

- Owned (n=27)
- Rented (n=6)
- Other (n=1)

Due to the small sample size of the Other group, it was excluded from further analysis. Figure 49 shows a comparison of average PMPM$_{2.5}$ concentration values across the housing tenure groups.


**Figure 49:** Boxplot showing average PM$_{2.5}$concentrations by housing tenure

When comparing PM$_{2.5}$ concentrations across housing tenure groups, some differences can be observed. The median value for the Rented group is noticeably higher than that of the Owned group. However, the interquartile range differs notably between these two categories:

the Owned group exhibits a wider IQR, indicating greater variability in PM exposure among these households. Despite the differences in median and range, the upper whiskers of both groups appear to terminate at similar concentration levels, suggesting comparable maximum exposures. The lower whisker of the Owned group extends further downward, indicating the presence of a subset of households with particularly low $PM_{2.5}$ concentrations. However, no statistically significant difference in $PM_{2.5}$ concentrations was observed between housing tenure groups (Kruskal–Wallis test, $p = 0.14$).

Given the relatively small sample size of the Rented group ($n = 6$), these observations should be interpreted with caution. The limited and uneven group sizes reduce the statistical ability to detect meaningful differences, and larger, more balanced samples would be required to draw more robust conclusions regarding the relationship between housing tenure and indoor $PM_{2.5}$ levels.

Given the relatively small sample size of the Rented group ($n = 6$), observed trends should be interpreted cautiously; larger, more evenly distributed sample groups would be needed to draw firmer conclusions about the relationship between housing tenure and indoor $PM_{2.5}$ levels.

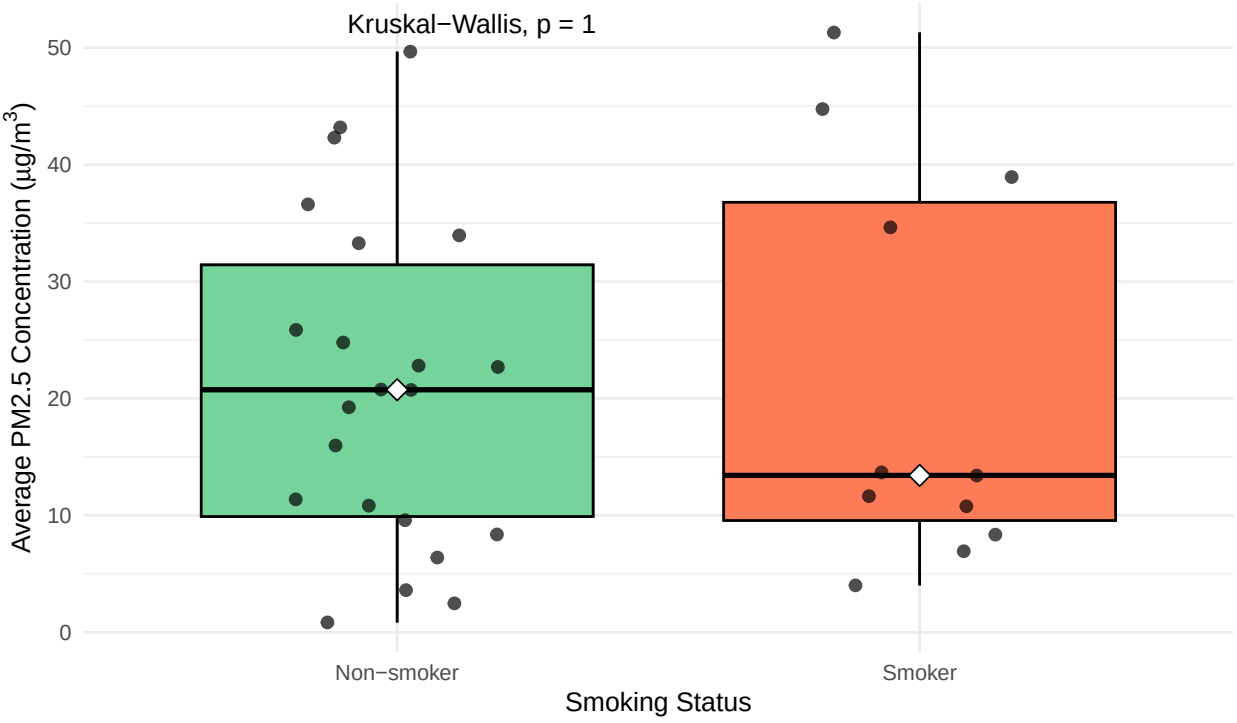



**Figure 50:** Boxplot showing average PM2.5 concentrations by smoking status

The distribution of smokers and non-smokers in the households studied is as follows:

- Smoker (n=11)
- Non-smoker (n=23)

The boxplot presented in Figure 50 compares the $PM_{2.5}$ concentrations between two household categories: those in which all residents identified as non-smokers, and those containing at least one smoking individual. Contrary to expectations, the median $PM_{2.5}$ concentration was lower in the smoker group than in the non-smoker group. Additionally, the IQR for the smoker group was slightly wider, suggesting a slightly greater variability in $PM_{2.5}$ levels within those households. However, no statistically significant difference in PM2.5 concentrations was observed between the two groups (Kruskal–Wallis test, p = 1.00), indicating no evidence of a difference in exposure between smoking and non-smoking households in this dataset. Notably, this is the only comparison that does not match the data from the analysis of all the Work Package 2 households, wherein the smoking households exhibited higher $PM_{2.5}$ concentrations[122].

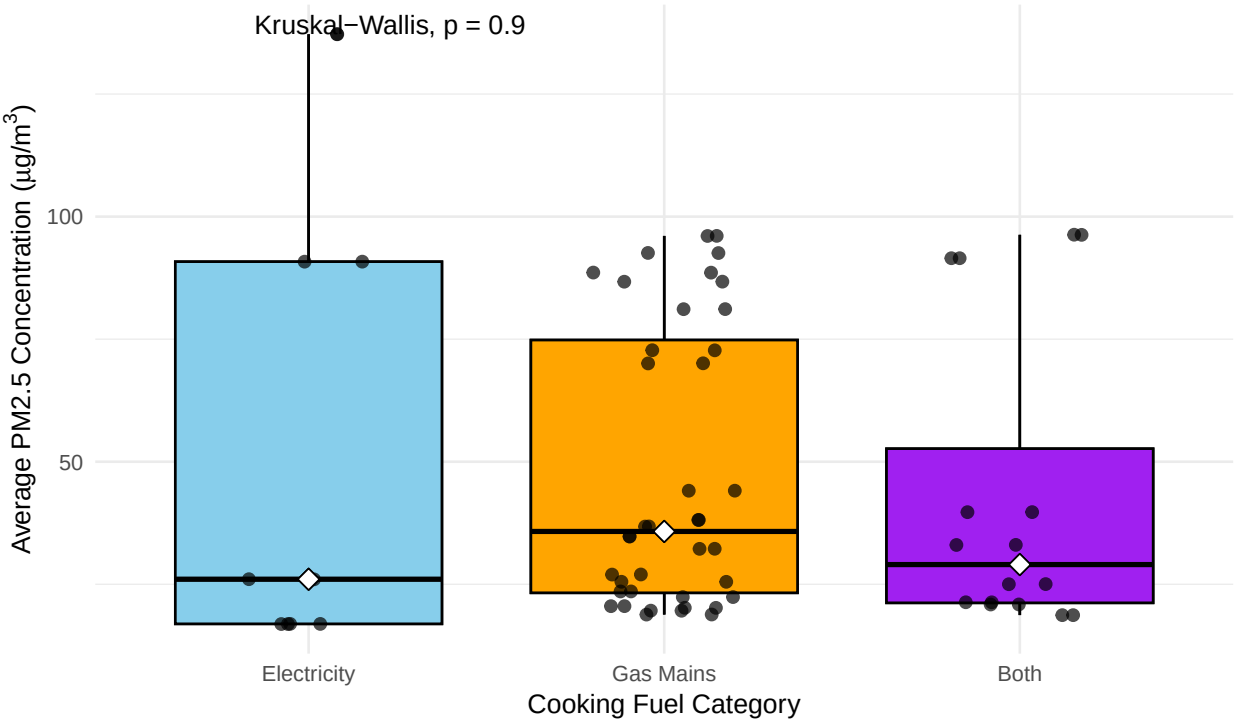



**Figure 51:** Boxplot showing average $PM_{2.5}$ concentrations by cooking fuel category

The impact of cooking fuel type on average $PM_{2.5}$ concentrations was also investigated. The distribution of cooking fuel type across households is as follows:

- Electricity (n=8)
- Gas Mains (n=21)
- Both (n=5)

These numbers were collected via the pre-sampling questionnaire, in which participants reported the types of cooking fuels used in their households. Figure 51 compares average $PM_{2.5}$ concentrations across cooking fuel categories.

Figure 51 presents a boxplot comparing the average $PM_{2.5}$ concentrations across these three cooking fuel categories. Households using electricity exclusively exhibited the lowest median $PM_{2.5}$ concentrations, as well as the narrowest IQR, suggesting lower and more consistent particulate matter levels. In contrast, households relying solely on gas mains demonstrated the highest median $PM_{2.5}$ concentration, with a moderately wider IQR than the electricity group. Interestingly, the Both group, representing households that used both gas and electricity for cooking, displayed the widest IQR and the highest third quartile (Q3) and upper whisker values.

It was expected that the Both group would exhibit values intermediate between the electricity-only and gas-only groups. However, the observed pattern indicated that the use of multiple cooking fuel types may introduce more variability in exposure. No statistically significant difference in $PM_{2.5}$ concentrations was observed between cooking fuel categories (Kruskal–Wallis test, $p = 0.90$), indicating no evidence of systematic differences in exposure between groups. Given the relatively small sample size of the Both group (n=5), these findings should be interpreted cautiously. It should also be noted that we do not know which cooking fuel types were used during the sampling period, and at what frequencies.

Household size can have a significant influence on indoor $PM_{2.5}$ concentrations because the number of occupants directly affects both emission and resuspension processes[34]. Cooking emissions are a major contributor to $PM_{2.5}$ indoors, and the frequency and scale of cooking events may increase with more people in a household[47].

In addition, the resuspension of particles from floors, carpets, and surfaces is strongly linked to human activity. More occupants in a household can result in a greater amount of movement, which can resuspend settled dust and particulate matter into the air[34]. Table

8 summarises the distribution of household sizes across the INGENIOUS households participating in this phase of Work Package 2.

**Table 8:** Household Sizes Across ING homes

| Household Size | Number of Households |
|---|---|
| 2 | 2 |
| 3 | 5 |
| 4 | 10 |
| 5 | 7 |
| 6 | 8 |
| 7 | 1 |
| 8 | 1 |

For the purposes of data analysis, the households were stratified into groups based on their household size.

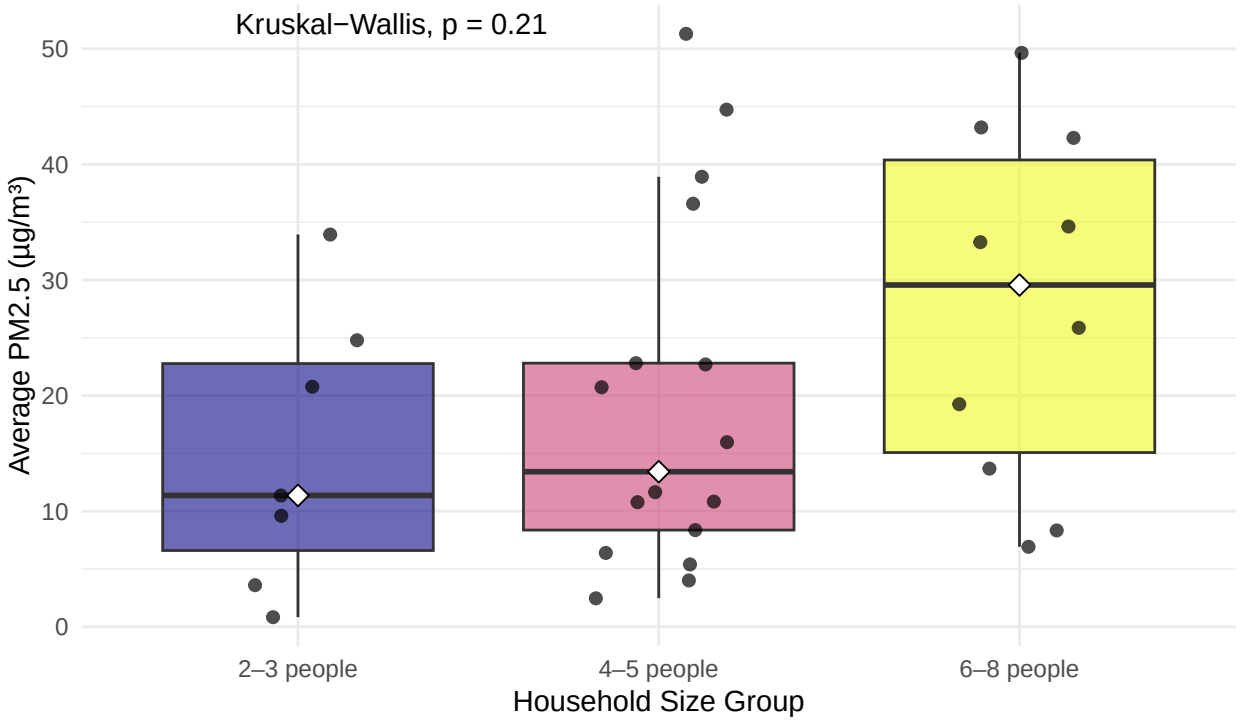



**Figure 52:** Boxplot showing average $PM_{2.5}$ concentrations by household size

Figure 52 compares the distribution of average PM2.5 concentrations across households of different sizes, grouped into 2–3 people, 4–5 people, and 6–8 people. The results show that

households with 2–3 occupants and those with 4–5 occupants display relatively similar central tendencies, with comparable medians and very similar interquartile ranges. In contrast, the 6–8 person households group exhibits both a higher median PM2.5 concentration and a wider interquartile range compared to the other groups, suggesting elevated exposure levels and greater variability in particle concentrations within this group.

These findings imply that increases in household size may be associated with greater particulate matter emissions and resuspension. However, no statistically significant difference in $PM_{2.5}$ concentrations was observed between household size groups (Kruskal–Wallis test, $p = 0.21$), indicating no evidence of systematic differences in exposure between groups.

Decorating activities such as painting, sanding, varnishing, or the use of adhesives and solvents can influence indoor $PM_{2.5}$ concentrations. Paint application introduces VOCs, which can then undergo reactions with indoor oxidants to form secondary organic aerosols[133]. Phthalates are also commonly found in paints and decorating materials; these compounds can off-gas into indoor air or adsorb onto airborne dust during and after decorating activities[61]. Phthalates may also partition between gas and particle phases, leading to the potential for continued indoor exposure even after the decorating event has concluded[61]. More specific investigation into the specific health effects of phthalate exposure will occur in the next chapter.

Participants were asked if various areas (e.g. bedrooms, living areas, kitchens) in their homes had been decorated in the 12 months preceding the sampling period. As the samplers for this particular study were placed in the kitchen, the responses for the kitchen area only were included in this analysis. The results are summarised below:

- No (n=25)
- Yes (n=9)

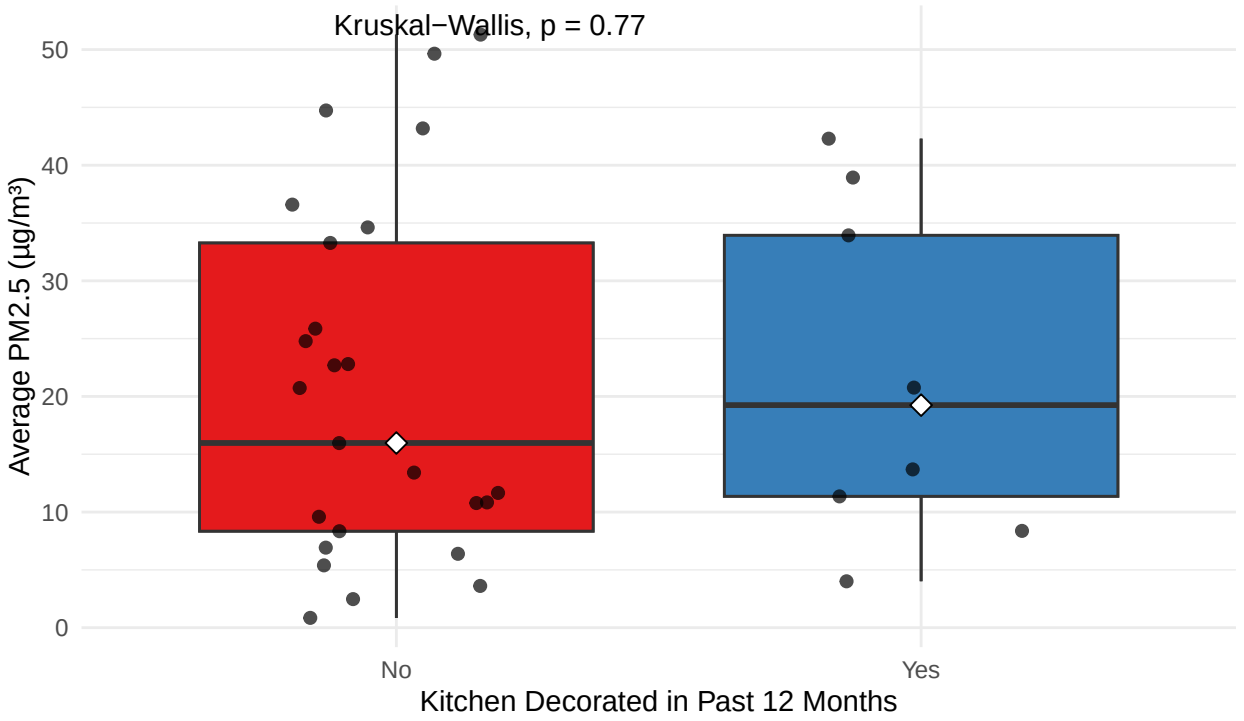



**Figure 53:** Boxplot showing average $PM_{2.5}$ concentrations by whether or not the kitchen was decorated in the past 12 months

Figure 53 compares average $PM_{2.5}$ concentrations between households that reported kitchen decorating within the past 12 months and those that did not. A surprising proportion of household (slightly more than a quarter) reported recent decorating activity. Despite this, the distributions between the two groups appear mostly similar. As shown in the figure, there are no pronounced differences in median concentrations or overall range of values between households with and without recent decorating work. No statistically significant difference in $PM_{2.5}$ concentrations was observed between households who had and had not decorated within the last year (Kruskal–Wallis test, $p = 0.77$), indicating no evidence of systematic differences in exposure between groups. As such, it can be concluded that in the context of this study, decorating has little effect on average $PM_{2.5}$ concentrations.

The relationship between indoor and outdoor $PM_{2.5}$ levels is complex and influenced by a number of factors. Building characteristics, such as ventilation and building materials, can affect the rate of infiltration of air from outdoors to indoors. Occupant behaviors such as the opening of windows, use of fans or air purifiers, and indoor activities such as cooking and smoking further affect indoor concentrations of $PM_{2.5}$. Ambient conditions such as wind, temperature differences, and humidity also play a key role. In addition, the proximity of outdoor

pollution sources, such as traffic and industrial or agricultural activity to homes can affect the types and concentrations of pollutants found indoors. Urban areas typically experience higher outdoor air pollution due to higher vehicle density and industrial activities[134].

In contrast, rural areas often experience lower background levels of outdoor $PM_{2.5}$, especially when distant from major roads or industrial centers. This can result in lower baseline indoor $PM_{2.5}$ concentrations.

To investigate potential effects from being located in urban or rural environments, the homes were separated into urban or rural designations based on postcodes and their subsequent Lower Super Output Area (LSOA) data, from the Office for National Statistics (ONS). Table 9 summarises these results, along with a few other variables.

In total, there were 7 households in the rural category and 27 in the urban category. This table is used to explore potential relationships between particulate matter concentrations and household characteristics. Table 9 shows that $PM_{2.5}$ concentrations vary across households and are not explained by a single factor. Higher concentrations are more frequently observed in urban homes, although substantial variability exists within this group. Larger households also tend to show higher $PM_{2.5}$ levels, suggesting a possible influence of increased indoor activity, but this relationship is not consistent.

Similarly, higher concentrations are often associated with more deprived households, although this pattern is not uniform. Overall, these results suggest that $PM_{2.5}$ levels are influenced by a combination of factors, including location, occupancy, and socio-economic conditions, rather than any single dominant variable.

This classification also enabled the investigation of potential hotspots within Bradford, identifying areas where homes sharing the same postcode may have exhibited higher or lower indoor $PM_{2.5}$ concentrations.

**Table 9:** Household-level comparison of mean $PM_{2.5}$ concentrations with postcode, urban/rural classification, occupancy (number of people), and Index of Multiple Deprivation (IMD) group.

| York ID | Postcode | Urban/Rural | Number of People | IMD Group | Mean $PM_{2.5}$ |
|---|---|---|---|---|---|
| 1 | BD9 | urban | 8 | 4 | 13.7 |
| 2 | BD8 | urban | 6 | 1 | 6.9 |
| 3 | BD9 | urban | 2 | 5 | 33.9 |
| 4 | BD18 | urban | 3 | 8 | 9.6 |
| 5 | BD18 | urban | 4 | 6 | 10.8 |
| 6 | BD8 | urban | 4 | 2 | 22.8 |
| 7 | BD4 | urban | 5 | 2 | 36.6 |
| 8 | BD15 | rural | 3 | 8 | 20.8 |
| 9 | BD12 | urban | 4 | 7 | 51.3 |
| 10 | BD12 | urban | 5 | 7 | 13.4 |
| 11 | BD20 | rural | 4 | 10 | 10.8 |
| 12 | BD7 | urban | 5 | 6 | 5.4 |
| 13 | BD7 | urban | 4 | 6 | 38.9 |
| 14 | BD18 | urban | 3 | 5 | 24.8 |
| 15 | BD2 | urban | 7 | 6 | 33.3 |
| 16 | BD7 | urban | 5 | 4 | 22.7 |
| 17 | BD5 | urban | 6 | 3 | 25.9 |
| 18 | BD2 | urban | 6 | 6 | 42.3 |
| 19 | BD9 | urban | 6 | 4 | 19.2 |
| 20 | BD2 | urban | 6 | 1 | 34.6 |
| 21 | BD7 | urban | 5 | 1 | 8.4 |
| 22 | BD5 | urban | 6 | 1 | 49.6 |
| 23 | BD6 | urban | 5 | 7 | 44.7 |
| 24 | BD2 | urban | 6 | 5 | 43.2 |
| 25 | BD7 | urban | 6 | 3 | 8.3 |
| 26 | BD15 | rural | 4 | 9 | 16.0 |
| 27 | BD12 | urban | 4 | 6 | 4.0 |
| 28 | BD14 | rural | 2 | 7 | 3.6 |
| 29 | BD18 | urban | 3 | 8 | 11.4 |
| 30 | BD17 | rural | 4 | 5 | 20.7 |
| 31 | BD9 | urban | 4 | 5 | 11.7 |
| 32 | BD16 | rural | 3 | 9 | 0.8 |
| 33 | BD18 | urban | 5 | 8 | 2.5 |
| 34 | BD15 | rural | 4 | 9 | 6.4 |

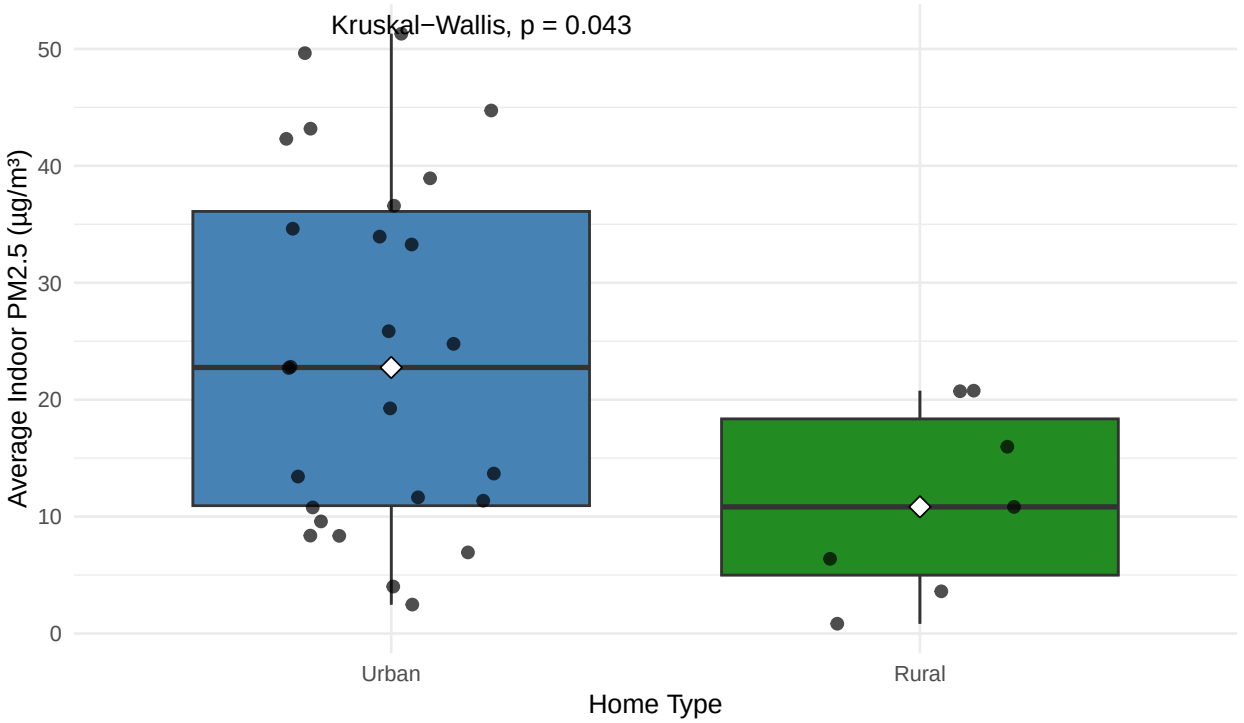



**Figure 54:** Comparing Urban and Rural Average $PM_{2.5}$ Concentrations

A notable difference was observed between the two groups in terms of both median values and variability. The Urban homes exhibited a higher mean indoor $PM_{2.5}$ concentration compared to the Rural homes, indicating greater overall exposure. In addition, the Urban group displayed a wider range of values, with both the interquartile range and the whiskers extending to higher concentrations, suggesting more pronounced variability amongst urban dwellings. A statistically significant difference in $PM_{2.5}$ concentrations was observed between Urban and Rural homes (Kruskal–Wallis test, $p = 0.043$), indicating evidence of variation in exposure between these settings.

However, it should be emphasized that the sample sizes were relatively small: ($n = 27$) for Urban homes and ($n = 7$) for Rural homes, which may limit the statistical robustness of the analysis and increase the uncertainty of these comparisons. As a result, while the observed trends are informative, they should be interpreted with caution; further studies with larger, more representative samples would be required to confirm the robustness of these patterns and fully understand the urban–rural differences in indoor $PM_{2.5}$ exposure.

Overall, the analysis of average $PM_{2.5}$ concentrations across households revealed several

patterns of interest, though many of these findings were nuanced by limitations in sample size and group distribution. However, stratification by demographic and environmental variables showed some tentative trends.

Households classified into the least deprived IMD group (deciles 7–10) exhibited lower median $PM_{2.5}$ concentrations and narrower interquartile ranges. Similarly, households that relied exclusively on electricity for cooking had lower $PM_{2.5}$ levels compared to those using gas or a combination of both, suggesting a potential relationship between fuel type and particulate matter emissions indoors. However, these differences were not statistically significant (Kruskal–Wallis tests, $p > 0.05$), indicating that the observed trends may reflect within-group variability rather than systematic effects.

Interestingly, the analysis found no substantial difference in $PM_{2.5}$ levels between smoking and non-smoking households, with the smoker group even showing a marginally lower median. This was supported by the statistical analysis, which showed no evidence of a difference between groups ($p = 1.00$). In contrast, a statistically significant difference was observed between urban and rural households (Kruskal–Wallis test, $p = 0.043$), indicating evidence of variation in PM2.5 exposure between these settings.

As such, while patterns in $PM_{2.5}$ concentrations were observed across several household variables, the findings should be interpreted cautiously due to sample size constraints. Nonetheless, these results provide valuable insight into how indoor air quality may be influenced by social and behavioural factors, and lay the groundwork for more detailed investigations in future research with expanded cohorts.

### 3.2.4 Relationship between $PM_{2.5}$ mass loading and measured organic tracers

To assess whether the targeted chemical analysis captures variability in particulate matter, the total concentration of measured organic tracers was compared with the estimated $PM_{2.5}$ mass loading for each sample. A Spearman's rank correlation analysis indicated a weak but statistically significant positive relationship between $PM_{2.5}$ mass loading and total measured organics ($\rho = 0.37$, $p = 0.032$), as seen in Figure 55. This suggests that increases in particulate mass are associated with increases in the concentration of the measured organic tracers, though the strength of this relationship is limited.

The relatively weak correlation suggests that the targeted organic tracers represent only a small fraction of the total $PM_{2.5}$ mass. This is expected as the targeted analysis only

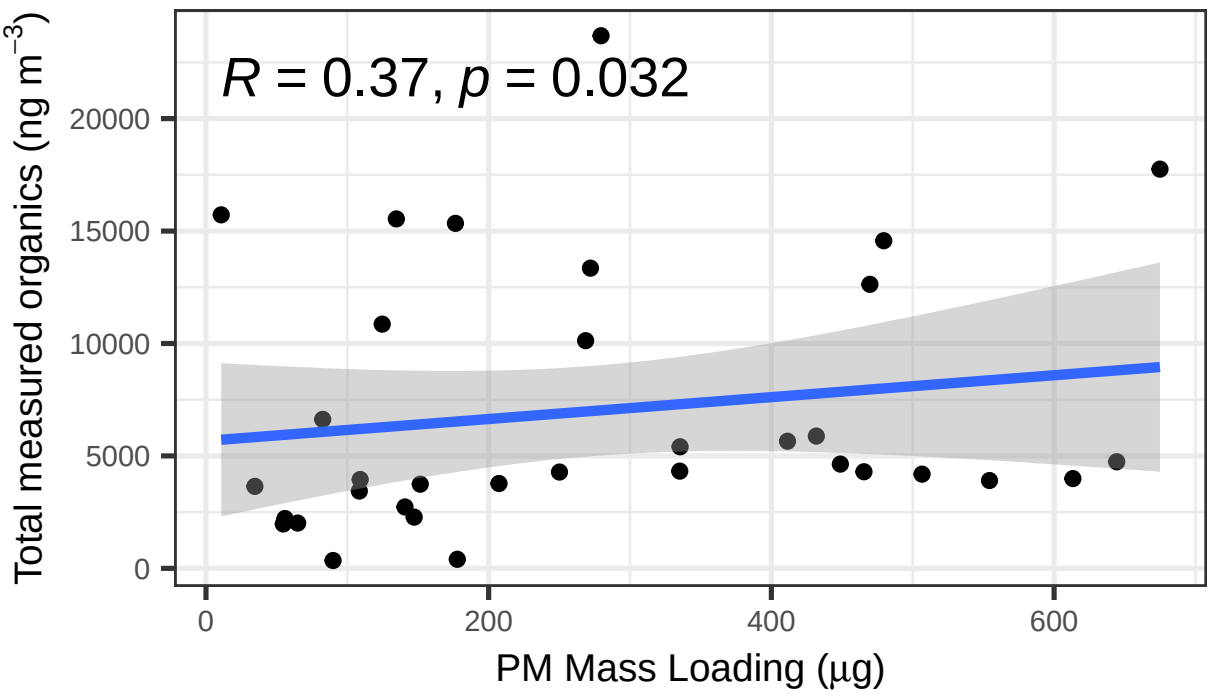



**Figure 55:** Scatter plot of $PM_{2.5}$ mass loading versus total measured organic tracers for each sample. The fitted line represents a linear regression.

comprised a small portion of the total species found in the samples. As a result, variability in $PM_{2.5}$ mass is likely driven in part by components not captured in the targeted analysis.

## 3.3 Conclusions

This chapter presented a targeted characterisation of indoor particulate matter ($PM_{2.5}$) in real-home environments. Building on the sampling framework described previously, the data from filter-based GC×GC–ToF-MS and low-cost sensors were used to quantify concentrations, explore chemical composition, and explore the effects of household behaviours and socioeconomic factors.

Tables 6 and the boxplots (Figures 42 and 43) revealed highly varied chemical compositions across homes. Several analytes were ubiquitous at low-to-moderate levels, while others appeared infrequently with large maxima, which are theorised to be consistent with episodic emissions. The breadth of the observed ranges, and the presence of long upper tails for multiple classes suggest that short, high-intensity events in some households may disproportionately drive overall exposure.

Analysis of average PM2.5 time series further emphasised temporal variability. Comparisons between two-week and three-day windows showed that the timing of short-term sampling strongly influences the results obtained. Stratified summaries pointed to plausible patterns, including lower medians and variability in the least deprived IMD group, lower concentration levels in electricity-only cooking homes compared to gas or mixed-fuel homes, higher central tendency and spread with larger household size, and higher values in urban compared to rural settings. Notably, smoking status did not correspond to higher average PM2.5 in this sample, a result that should be interpreted cautiously given group sizes and the possibility that episodic peaks were not captured in the aligned three-day windows.

Several limitations are present within these findings. The chemical analysis focused on a defined target list rather than full non-target discovery; as such, the results likely represent a limited view of indoor chemical diversity. This was unavoidable due to the massive and overwhelming number of peaks that are characteristic of GC×GC–ToF-MS outputs due to the sensitivity of the instrument. Group comparisons (e.g. housing tenure, cooking fuel type, urban/rural designation) were informative but constrained by sample sizes and uneven group distributions.

Despite this, several key findings were highlighted in this chapter. Indoor PM2.5 in real homes is chemically complex and highly variable. Filtering by diagnostic ions provided a method for interpreting complex 2D plots and linking composition to specific activities. Socio-economic, behavioural and dwelling characteristics may plausibly affect both total PM2.5 and the chemical composition of indoor air, which may lead to inform interventions such as cooking practices, ventilation, and lifestyle choices.

The next chapter builds on this foundation by narrowing focus to compounds of high toxicological relevance and clearer sources. This is intended to link analytical chemistry results with the public health sector to inform effective intervention strategies.

# Chapter 4: Investigating the concentrations of Polycyclic Aromatic Hydrocarbons and Phthalates in INGENIOUS households and their risks to human health

# 4 Investigating the concentrations of PAHs and Phthalates in INGENIOUS households and their risks to on human health

This chapter investigates the occurrence, sources, and health risks of two priority contaminant classes: polycyclic aromatic hydrocarbons (PAHs) and phthalates in real-home environments sampled as part of the INGENIOUS project. The data for this chapter was obtained via filter sampling, which was then extracted and run though a GC×GC–ToF-MS to obtain concentrations of targeted analytes. The aim of this chapter was to investigate concentrations and detection frequencies of target PAHs and phthalates across households, then use this to predict possible health risks. This was carried out using health-relevant metrics such as benzo[a]pyrene (BaP) equivalents and lifetime excess cancer risk (LECR) for PAHs, and age-stratified inhalation exposures and incremental cancer risk for bis(2-ethylhexyl) phthalate (DEHP).Variation across households was investigated by stratifying results according to sociodemographic and behavioural influences such as Index of Multiple Deprivation, housing tenure, smoking status, cooking fuel, and household size. Overall, the chapter aims to investigate how indoor air quality correlates with exposure and risk indicators to identify dominant contributors to and potential risks to human health in indoor environments.

## 4.1 Polyclyclic Aromatic Hydrocarbons

### 4.1.1 Introduction

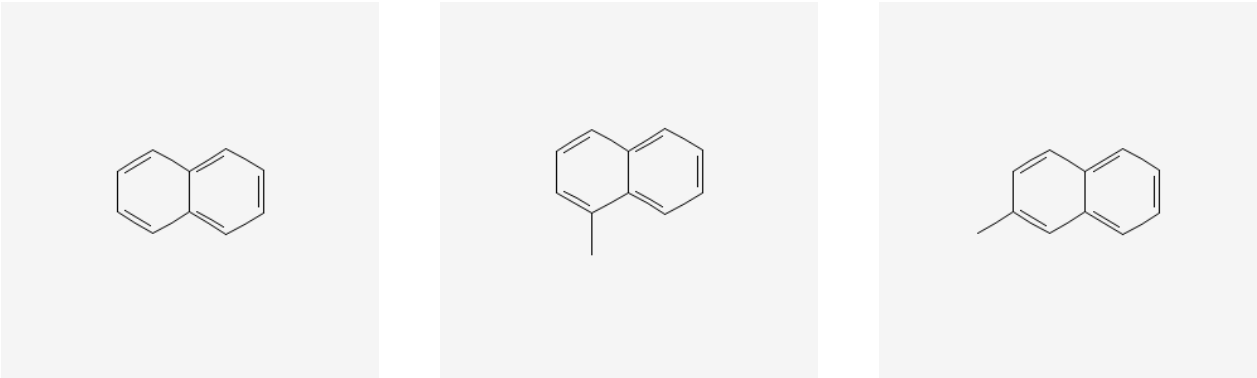
Polycyclic aromatic hydrocarbons (PAHs) are a class of organic compounds composed of multiple fused aromatic rings, primarily produced during incomplete combustion[135]. Inhalation is one of the main pathways of exposure to PAHs[135]. Indoor sources of PAHs include smoking, cooking activities, infiltration of pollutants from outdoors, and wood burning[136]. Inhalation of PAHs has been linked to several respiratory diseases (e.g. asthma, bronchitis), as well as cardiovascular disease[136].

Perhaps most importantly, several PAHs are classified as carcinogenic by the International Agency for Research on Cancer (IARC), with chronic exposure increasing the risk of lung and other cancers[15]. Vulnerable populations, such as children and individuals with pre-existing conditions, may be particularly at risk from sustained indoor PAH exposure, underscoring the importance of effective air quality management and public health interventions in indoor as well as outdoor environments [17].

The number of aromatic rings in a PAH determines its volatility and partitioning between gas and particle phases [137]. PAHs containing two or three rings are relatively volatile and as such mostly occur in the gas phase, allowing them to disperse widely [137]. In contrast, PAHs with five or six rings are much less volatile and are more likely to be adsorbed to airborne particulate matter[137]. Particle-bound PAHs are also more resistant to removal processes, leading to their accumulation in soils, dust, and indoor environments[60].

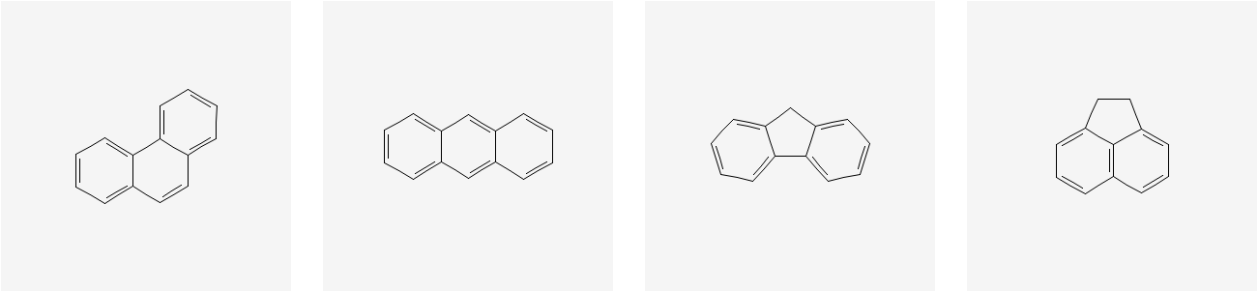
The toxicological properties of PAHs are likewise closely linked to their molecular structure. Low-numbered-ring PAHs can undergo atmospheric transformation to form secondary pollutants, including oxygenated PAHs and nitro-PAHs, many of which exhibit high reactivity and enhanced toxicity[138]. Higher-ring PAHs, such as benzo[a]pyrene and indeno[1,2,3-cd]pyrene, are well-established carcinogens and mutagens [15]. Their stability and ability to bioaccumulate, combined with their tendency to bind to DNA, make them particularly hazardous to human health[139]. Thus, the structural features of PAHs not only dictate their environmental fate but also their potential to cause long-term biological damage.

The following figures show the structures of all PAHs targeted and detected during the analysis of the INGENIOUS home samples. They are separated into different figures according to the number of aromatic rings that each PAH contains.

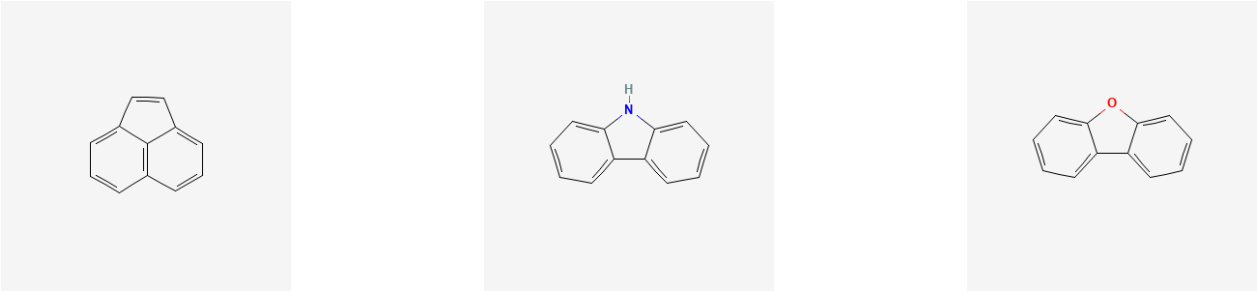


**(a)** Naphthalene **(b)** 1-Methylnaphthalene **(c)** 2-Methylnaphthalene

**Figure 56:** Targeted PAH compounds with two aromatic rings.

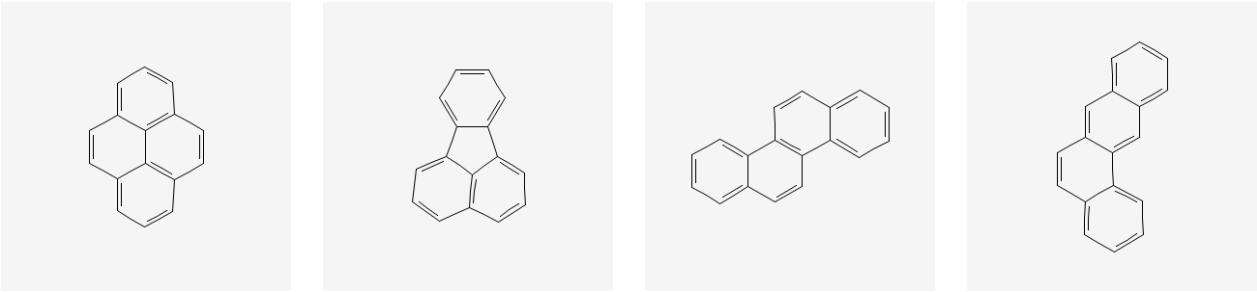


**(a)** Phenanthrene **(b)** Anthracene **(c)** Fluorene **(d)** Acenaphthene



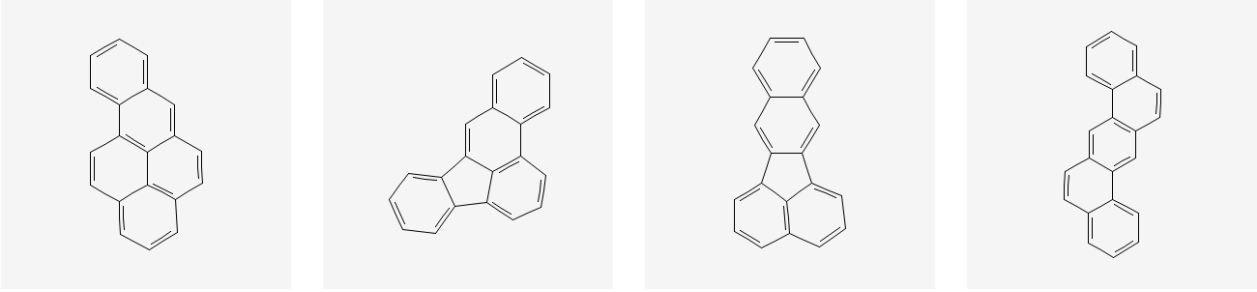

**(e)** Acenaphthylene **(f)** Carbazole **(g)** Dibenzofuran

**Figure 57:** Targeted PAH compounds with three aromatic rings (or fused aromatic heterocycles).



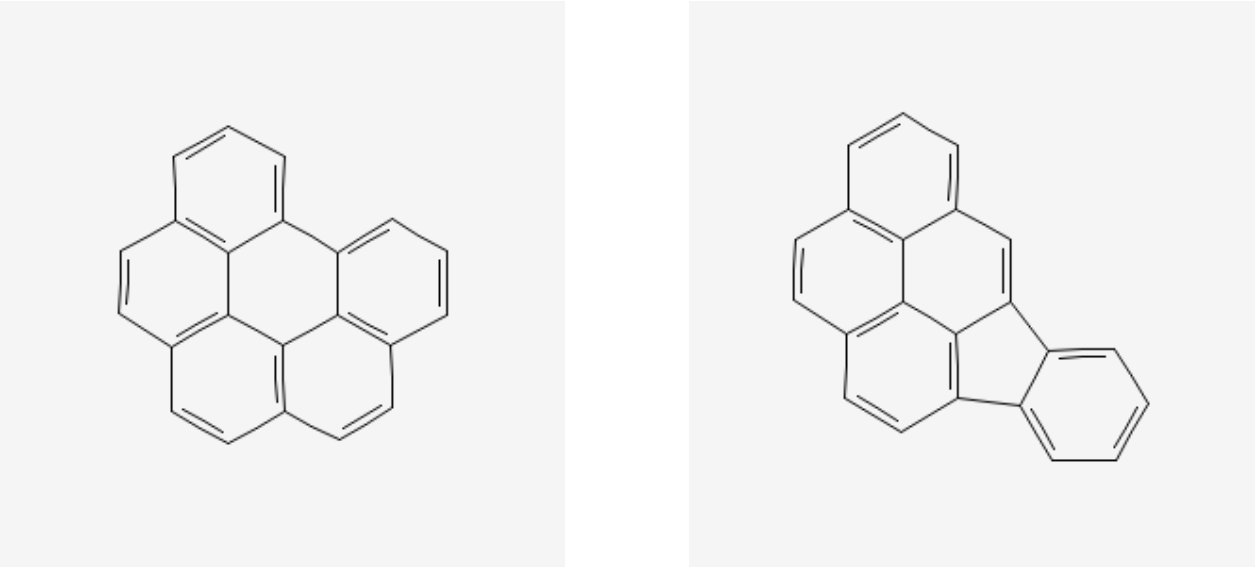
**(a)** Pyrene **(b)** Fluoranthene **(c)** Chrysene **(d)** Benz[a]anthracene

**Figure 58:** Targeted PAH compounds with four aromatic rings.



**(a)** Benzo[a]pyrene **(b)** Benzo[b]fluoranthene **(c)** Benzo[k]fluoranthene **(d)** Dibenz[a,h]anthracene

**Figure 59:** Targeted PAH compounds with five aromatic rings.



**(a)** Benzo[ghi]perylene **(b)** Indeno[1,2,3-cd]pyrene

**Figure 60:** Targeted PAH compounds with six aromatic rings.

### 4.1.2 PAHs and the INGENIOUS project

The following table (10) summarises the data for the PAHs targeted via the targeted analysis decribed in the previous chapter. All reported concentrations throughout this chapter and indeed, throughout this thesis, correspond to analytes associated with particulate matter collected on filters and therefore represent only the particle-phase fraction of each compound. Gas-phase concentrations were not measured in this study.

**Table 10:** Summary statistics of PAHs Detected

| Name | Min(ng/m$^3$) | Max (ng/m$^3$) | Median (ng/m$^3$) | No. of Samples Present In |
|---|---|---|---|---|
| Indeno[1,2,3-cd]pyrene | 7.1 | 51.9 | 41.8 | 14 |
| Dibenz[a,h]anthracene | 6.6 | 41.8 | 10.6 | 32 |
| Benzo[b]fluoranthene | 4.0 | 13.7 | 8.9 | 5 |
| Pyrene | 0.1 | 8.9 | 7.7 | 3 |
| Benzo[ghi]perylene | 6.0 | 13.9 | 7.5 | 20 |
| Benz[a]anthracene | 5.8 | 5.8 | 5.8 | 1 |
| Phenanthrene | 0.4 | 15.2 | 2.6 | 29 |
| Benzo[a]pyrene | 0.8 | 11.7 | 2.1 | 4 |
| Chrysene | 0.5 | 21.5 | 2.0 | 11 |
| Benzo[k]fluoranthene | 0.4 | 17.1 | 1.3 | 6 |
| Acenaphthene | 0.0 | 1.3 | 1.1 | 3 |
| Fluorene | 0.5 | 0.7 | 0.6 | 2 |
| Acenaphthylene | 0.1 | 1.6 | 0.3 | 21 |

Figure 61 shows a box and whisker plot displaying the concentration distributions of individual PAHs measured across all household samples. The analytes included range from lower molecular weight PAHs (e.g., acenaphthene, fluorene) to higher molecular weight, more carcinogenic compounds such as benzo [a] pyrene and dibenz [a,h] anthracene.

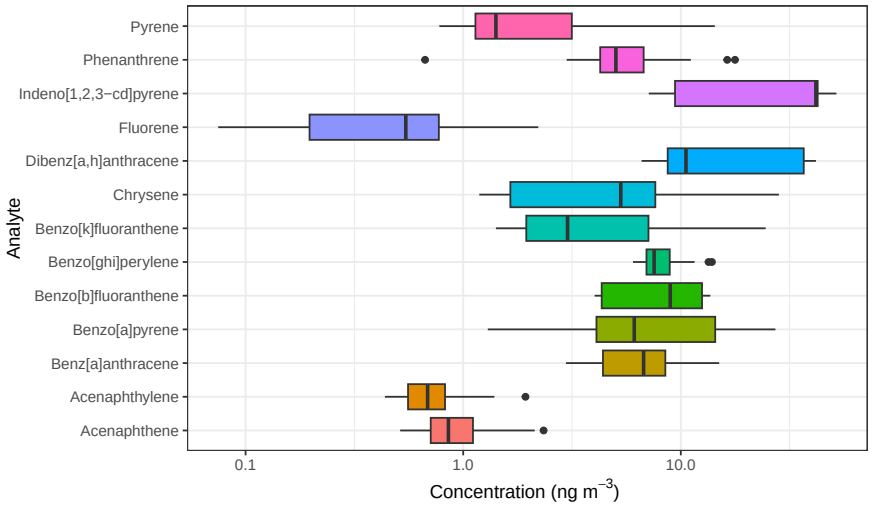



**Figure 61:** Boxplot of concentrations of of PAHs found in homes. Measured concentrations represent the particle-phase (PM-bound) fraction only

Benzo[a]pyrene, dibenz[a,h]anthracene, and indeno[1,2,3-cd]pyrene exhibit higher overall concentrations compared to the other PAHs, and are of significant concern due to their high toxic equivalency factors (TEFs) and thus their higher likelihood of causing cancer. Dibenz [a,h] anthracene was also found in 32 of 34 samples, and was by far the most ubiquitous compound found across samples.

The distribution of PAH concentrations reflects a complex mixture of sources and behaviours influencing indoor air quality. The presence of both high- and low-molecular-weight PAHs across the sample set underscores the need to consider both indoor and outdoor contributions when assessing exposure.

### 4.1.3 Total Concentrations of PAHs

The total PAH concentrations were calculated as the sum of all quantified PAH analytes in each sample. This approach reduced the dominating effect of individual highly carcinogenic PAHs and emphasised the cumulative presence of PAHs in the sample. As a result, differences between samples in terms of overall PAH load became more apparent, independently of individual compound toxicity.

The table below shows the total PAH concentrations obtained across all home samples. Measured concentrations represent the particle-phase (PM-bound) fraction only

**Table 11:** Total PAH Concentrations by York ID

| York ID | Total PAH Concentration (ng/m$^3$) |
|---|---|
| 1 | 70.1 |
| 2 | 72.7 |
| 3 | 81.1 |
| 4 | 88.6 |
| 5 | 91.5 |
| 6 | 92.6 |
| 7 | 137.2 |
| 8 | 90.8 |
| 9 | 96.3 |
| 10 | 96.1 |
| 11 | 25.0 |
| 12 | 26.2 |
| 13 | 25.5 |
| 14 | 38.1 |
| 15 | 39.7 |
| 16 | 20.2 |
| 17 | 32.2 |
| 18 | 44.1 |
| 19 | 20.6 |
| 20 | 27.0 |
| 21 | 19.6 |
| 22 | 36.8 |
| 23 | 34.8 |
| 24 | 21.3 |
| 25 | 23.6 |
| 26 | 20.9 |
| 27 | 26.0 |
| 28 | 16.9 |
| 29 | 22.4 |
| 30 | 33.0 |
| 31 | 18.8 |
| 32 | 86.7 |
| 33 | 16.9 |
| 34 | 18.7 |

Figure 62 shows a bar plot of total PAH across all 34 INGENIOUS households.

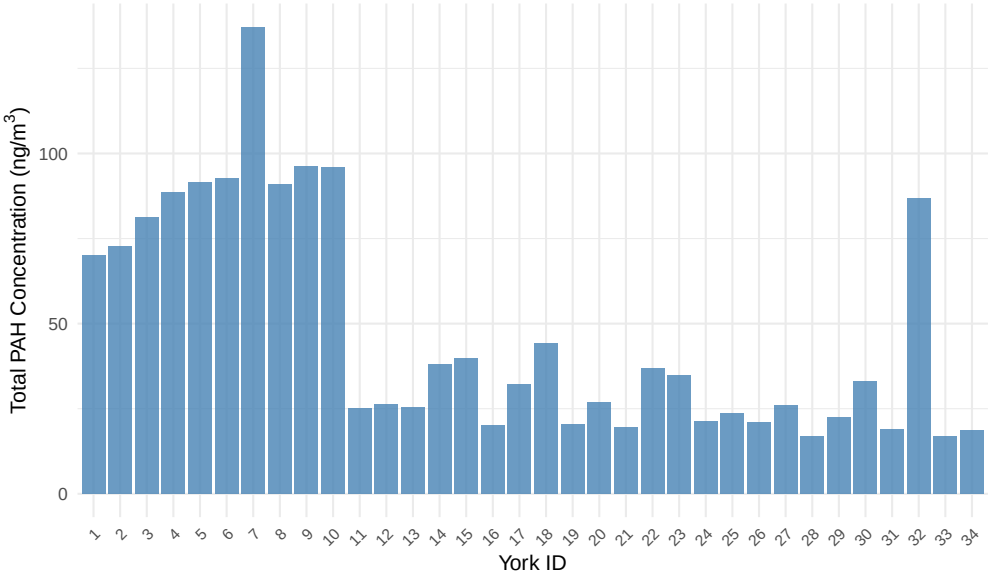



**Figure 62:** Bar plot showing summed PAH concentrations by York ID

Most samples show total PAH concentrations of between 15-100 ng/m$^3$. In particular, the first 10 samples (York ID= 1-10) have concentrations above 50 ng/m$^3$, while subsequent samples all have relatively lower concentrations, being in the range of 15-50 ng/m$^3$ with the exception of York ID=32, which shows a concentration similar to the first 10 samples taken. York ID=7 is also an exceptional sample, as its total PAH concentration (137.2 ng/m$^3$) is much higher than that of the other homes. These trends will be further investigated in a later section, which will assess each home's lifetime excess cancer risk, or LECR.

Dibenz [a,h] anthracene and Indeno [1,2,3-cd] pyrene were detected in nearly all samples and consistently exhibited the highest concentrations among the PAHs quantified. These PAHs are primarily produced during the incomplete combustion of organic materials, including fossil fuels, wood, and biomass[34]. Their widespread detection suggests the influence of regional combustion-related emissions, such as those from vehicular exhaust and industrial processes, infiltrating from outdoors.

However, this interpretation should be treated with caution. If outdoor infiltration were the dominant source, it would be expected that homes within similar geographic areas and sampling periods would exhibit comparable PAHs present and concentrations. This was not able to be investigated definitively within the dataset, due to only having the postcodes of the individual homes available to us; we do not know how close the homes were to one another,

as postcodes can span a wide geographical area.

Furthermore, while several detected PAHs are commonly associated with combustion sources such as vehicle emissions, the dataset does not include a source-specific tracer that would allow assured attribution to outdoor sources. As such, the observed patterns cannot be conclusively linked to a single dominant source.

Chrysene and Benzo [ghi] perylene were also commonly found across the samples, though generally at lower concentrations. Like Dibenz [a,h] anthracene and Indeno [1,2,3-cd] pyrene, these PAHs are also products of high-temperature combustion[140]. Their frequent detection further supports the likelihood of intrusion from outdoor sources.

The presence of these PAHs are consistent with studies that have documented their persistence in the environment due to their low volatility and resistance to degradation[35].

### 4.1.4 Lifetime Excess Cancer Risk Calculations

Benzo[a]pyrene (BaP) equivalents are a metric used to estimate the overall carcinogenic potential of a mixture of PAHs. Since PAHs vary greatly in their potential for toxicity, BaP equivalents provide a way to estimate the risk of exposure to PAHs relative to benzo[a]pyrene, which is a well-studied and highly carcinogenic PAH found in cigarettes. This is achieved by applying toxic equivalency factors (TEFs) to each PAH in the mixture, which reflect how potent each compound is in relation to benzo[a]pyrene. The concentration of each PAH is multiplied by its TEF, and the resulting values are summed up, resulting in a single concentration expressed in units of BaP equivalents, in this case ng/m$^3$. The equation showing this in Equation 2: BaP equivalents, and a table of the TEF values used in this analysis are in Table 12[141].

$$BAP_{eq} = \sum_{i=1}^{N} PAH_i \times TEF_i$$

*TEF*=Toxic equivalency factor
All TEF values obtained from EPA

(Equation 2: BaP equivalents)

**Table 12:** Toxic Equivalency Factors (TEFs) used in LECR calculations

| PAH Compound | TEF |
|---|---|
| Chrysene | 0.01 |
| Dibenz[a,h]anthracene | 1.00 |
| Benzo[b]fluoranthene | 0.10 |
| Benzo[a]pyrene | 1.00 |
| Pyrene | 0.001 |
| Acenaphthene | 0.001 |
| Anthracene | 0.001 |
| Benzo[k]fluoranthene | 0.10 |
| Indeno[1,2,3-cd]pyrene | 0.10 |
| Benzo[ghi]perylene | 0.01 |

$$LECR = BAP_{eq} \times UR$$

*UR*= WHO Unit Risk, $8.7 \times 10^{-5}$
meaning that 8.7 people per 100 000 people may contract lung cancer when exposed continuously to 1 ng/m³ of BaP over a lifetime of 70 years

(Equation 3: Lifetime Excess Cancer Risk)

Using the values obtained from Equation 2: BaP equivalents, the lifetime excess cancer risk (LECR) of the inhabitants of the homes was calculated. To calculate the LECR, the BAP equivalent concentrations were multiplied by a unit risk factor (UR), which is a value provided by the World Health Organisation that represents the estimated lifetime cancer risk from breathing 1 ng/m³ of benzo [a] pyrene over a lifetime of 70 years[15]. The WHO has set this value at $8.7 \times 10^{-5}$[15]. This value denotes that for every million people exposed to 1 ng/m³ of benzo [a] pyrene, 87 will develop lung cancer, which they would not have if they had not been exposed. The WHO has set the allowable LECR at $1 \times 10^{-6}$, or one excess case of lung cancer per every million individuals, meaning that they have deemed this an acceptable risk.

The LECR was calculated for every INGENIOUS household, using Equation 2: BaP equivalents and Equation 3: Lifetime Excess Cancer Risk. The results of this are plotted in Figure 63, with each individual plot point denoting the LECR calculated for that household. The red dotted line represents the WHO allowable LECR of $1 \times 10^{-6}$.

As seen in the plot, every single household in INGENIOUS WP2 had LECR values far exceeding the WHO allowable limit. In most cases, this was between two and three orders of magnitude higher than the allowable limit, meaning that for every million individuals exposed to these concentrations of the BaP equivalents, between 100 and 1000 individuals would be predicted to develop lung cancer as a result of their exposure. As a caveat, it must be noted that there was an assumption for methodological purposes that individuals were exposed to these concentrations of PAHs throughout their lifetimes, which is implausible. While this assumption is not reflective of real-world exposure, it allowed for standardised comparison across households and provides an assessment of the potential health implications of exposure toindoor PAH concentrations.

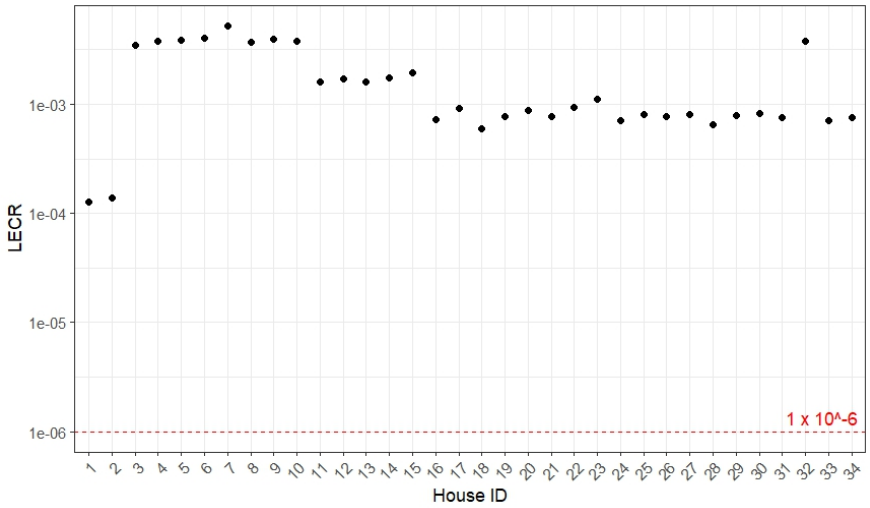



**Figure 63:** LECR across INGENIOUS households

Interestingly, the households included in this study did not follow the temporal patterns that were initially predicted. Samples were collected over a seven-month period, from October 2023 to April 2024, with household identifiers (York IDs) assigned in chronological order based on the date of collection. Prior to data collection, through discussion iwth representatives from the Bradford City Council it was expected that the winter months, especially during the peak of the cost of living crisis, would correspond with elevated levels of indoor PAH exposure and therefore higher associated Lifetime Excess Cancer Risk (LECR).

It was hypothesised that households might increasingly rely on alternative heating sources (e.g. wood-burning stoves, fireplaces, etc.) to offset high gas and electricity costs, to the

point where the local city council began preparing information to disseminate regarding safe materials to burn. Additionally, it was predicted that in colder months, reduced ventilation practices (e.g., keeping windows and doors closed to conserve heat) would contribute to increased indoor pollutant accumulation.

However, counter to these expectations, the observed LECR values did not follow a simple upward trend through the colder months. While the first two households sampled exhibited relatively low LECR values, subsequent households demonstrated higher LECRs, which generally declined over time, though not drastically. In addition, when comparing the data shown in Figure 63 to the total PAH concentration values per household (shown in Figure 62) there is much less variation shown between samples. In particular, the first two samples do not appear to be outliers when compared to the rest of the samples in the total PAH concentration calculations.

These unexpected trends prompted further investigation into assumptions related to household heating practices. Specifically, while wood-burning was predicted to be a potentially significant source of indoor PAHs, it became clear that this hypothesis did not align with the lived realities of most participating households. An oversight when predictions were made was the limited prevalence of wood-burning stoves or open fireplaces among the sampled homes. This absence is likely linked to the socioeconomic profile of the study population in Bradford, where features such as wood-burning stoves or fireplaces are more commonly found in higher income households. In addition, even where wood-burning appliances were present, the high cost of pre-treated wood may have been a significant obstacle to their use. These findings highlight how assumptions about behavioural responses to economic pressures must be grounded in an understanding of the lived reality of the studied communities.

Another important observation was the narrow range of LECR values across the majority of households, which suggested that intrusion from outdoor sources may have contributed significantly to levels of PAHs indoors. This is supported by the types of analytes detected across the samples. While there were distinct differences in the specific PAHs identified within different homes, the overall levels of LECR remained relatively consistent (within 2 orders of magnitude), indicating that external environmental contribution (e.g. vehicular or industrial emissions) may have been a dominating influence across households.

To explore this further, the contribution of each individual PAH to the calculated LECR for each sample was examined and is presented in Figure 64.

The first two samples (York ID= 1, 2) appeared at first glance to be outliers, and the question of exclusion of these two samples was considered due to the possibility of errors occurring during the sample collection period or extraction procedure. Upon further investigation, these two samples have lower calculated LECR due to dibenz [a, h] anthracene not being present. The concentrations of other compounds (all other PAHs, phthalates, etc.) found in these samples were found to be comparable to the other 32 samples. Thus, the decision was made not to exclude these two samples.

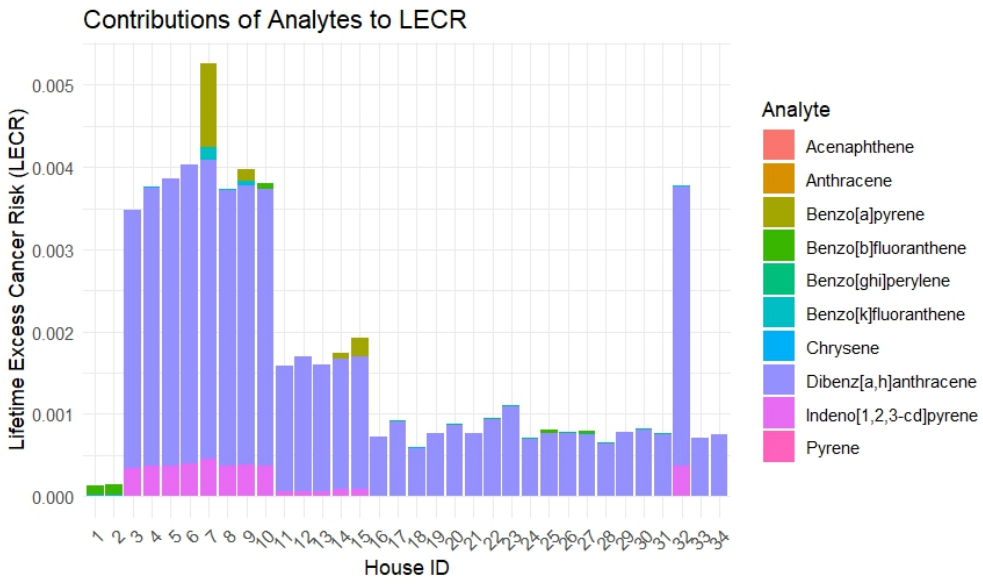



**Figure 64:** Stacked bar chart of contributions of PAHs to LECR

The analysis presented in Figure 64 and Figure 65 clearly demonstrates that the primary contributor to LECR in the studied households was Dibenz [a,h] anthracene. This compound is well known for its high carcinogenic potency, as reflected in its Toxic Equivalency Factor (TEF). With a TEF value of 1.0, Dibenz [a,h] anthracene is considered equivalent in toxicity to the reference compound Benzo [a] pyrene (BaP), which serves as the benchmark for calculating cancer risk from PAH exposure[104]. As such, even when present at comparatively lower concentrations than other PAHs, Dibenz [a,h] anthracene exerted a disproportionately large influence on overall LECR values.

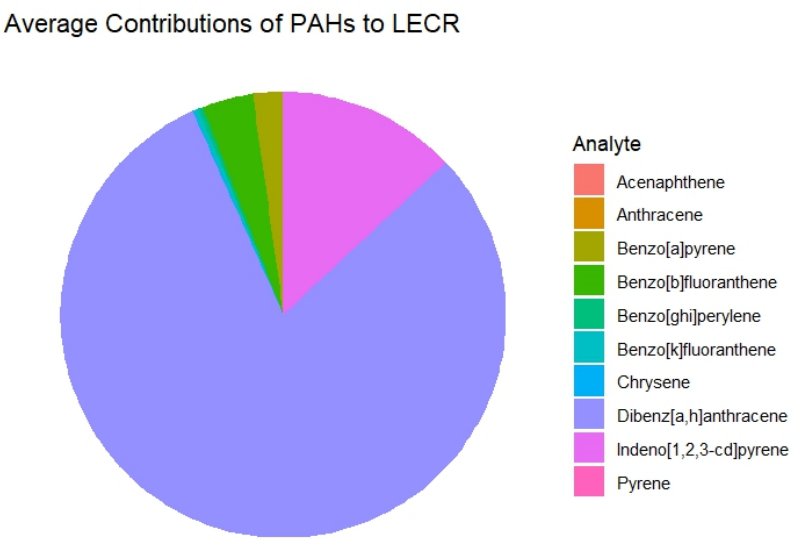



**Figure 65:** Pie chart showing the contribution of individual PAHs to LECR

Figure 65 further supports this showing the average contribution of each individual PAH to LECR across all sampled households. Again, Dibenz [a,h] anthracene stands out as the dominant contributor to cancer risk, followed by Benzo [b] fluoranthene and pyrene as other bigger contributors. While these latter compounds are also classified as probable human carcinogens, their TEF values are considerably lower (0.1 for Benzo [b] fluoranthene and 0.001 for pyrene), making their impact on LECR much lower comparatively.

The ubiquity of Dibenz [a,h] anthracene across samples called for investigation of its source. As this compound was consistently detected in most households despite natural variations in behavioural practices such as cooking, heating, and ventilation habits, it is possible that ambient, outdoor sources played a major role in these indoor PAH profiles. One of the primary urban sources of Dibenz [a,h] anthracene is vehicular emissions, particularly from diesel-powered engines[142].

These findings suggest that external environmental exposure may be a major contributor to elevated cancer risk caused by PAH exposure. The ubiquity of Dibenz [a,h] anthracene highlights the importance of source attribution in public health assessments and underscores the need for urban air quality interventions aimed at reducing emissions from vehicular emissions, as outdoor intrusion into indoor environments is highly suggested by the data.

### 4.1.5 Comparing LECR and Total PAH levels across Demographic Groups

In order to investigate the effects of potential socioeconomic inequalities and/or lifestyle choices on LECR, a number of factors were chosen for consideration. These included the Index of Multiple Deprivation (IMD), housing tenure, cooking fuel type, heating type, and smoking status. The data for these factors was acquired by Born in Bradford via their Day 1 Questionnaires, which were given to participating households on Day 1 of the 2-week sampling period in which WP2 took place. The boxplots throughout this chapter show median and interquartile range (IQR), with whiskers representing 1.5× IQR. Individual data points are overlaid. Statistical differences between groups were assessed using the Kruskal–Wallis test, as the data were non-normal.

#### Index of Multiple Deprivation Groups

Figure 66 shows the distribution of the 34 households across IMD groups.

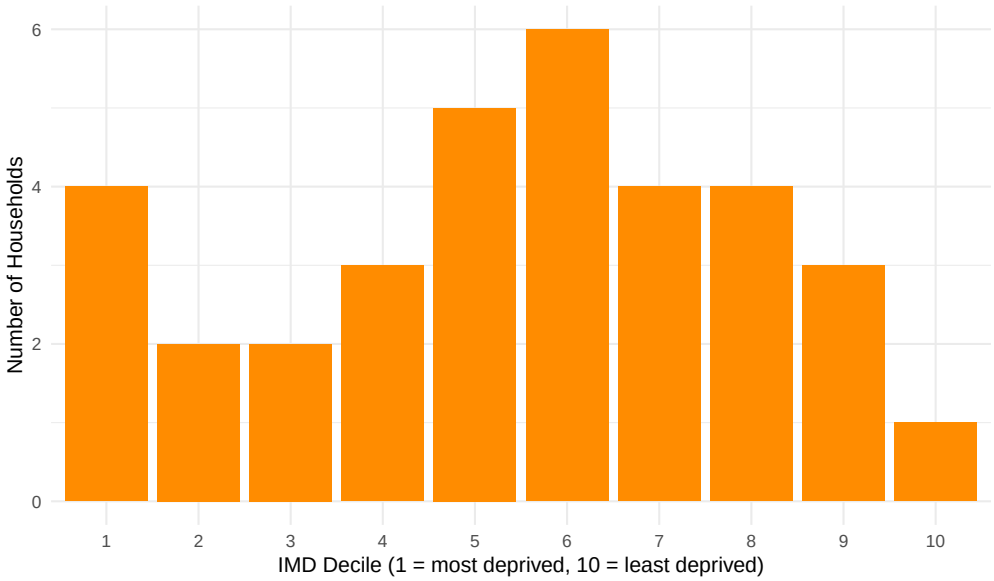



**Figure 66:** Bar chart showing the distribution of IMDs across households sampled

As aforementioned, due to the relatively low number of households (n=34) when compared to the number of IMD groups (n=10), the decision was made to combine IMD groups to further explore possible correlations with higher lifetime cancer risk.

- IMD Groups 1-3 (n=8)
- IMD Groups 4-6 (n=14)
- IMD Groups 7-10 (n=12)

Figure 67 shows LECR across the stratified IMD groups.

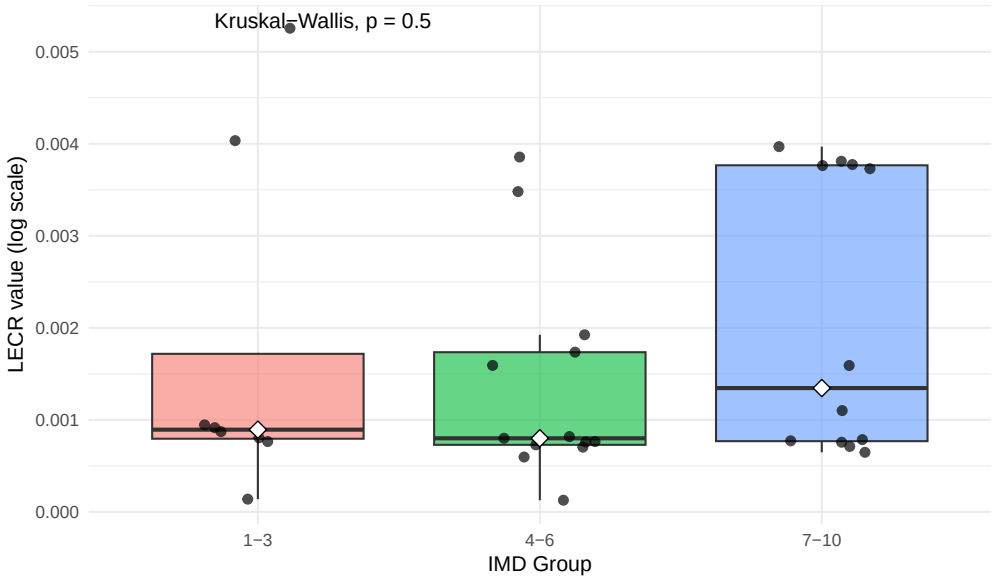



**Figure 67:** Boxplot showing LECR across IMD groups

The median LECR values appear relatively consistent across the three deprivation groups. Interestingly, the highest median LECR is observed in the least deprived group (deciles 7–10), although the difference is not pronounced. The most deprived (deciles 1–3) and least deprived (deciles 7-10) groups display the widest ranges of LECR values, suggesting greater variability in exposure within these groups.

While minor differences in LECR distributions are observed across IMD categories, the overall pattern does not suggest a clear or statistically significant correlation between deprivation and estimated cancer risk from PAH exposure. This is supported by the statistical analysis, which showed no evidence of a difference between groups ($p = 0.5$).

Analysis by total PAH concentration produces slightly different results. While the median total PAH levels remain relatively consistent across all three deprivation groups, notable differences arise in the spread and distribution of the data, shown in Figure 68.

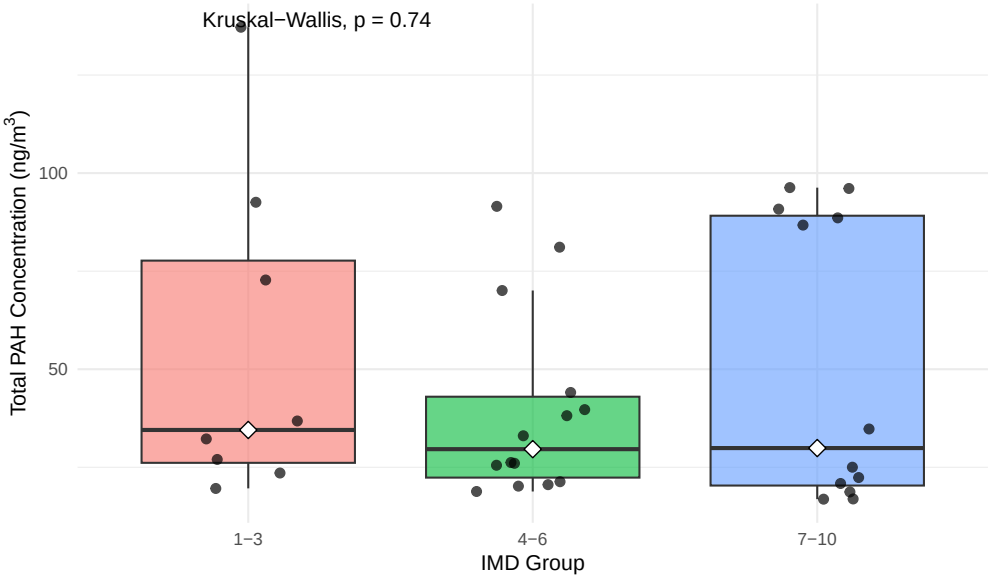



**Figure 68:** Boxplot showing Total PAH Concentrations across IMD Groups. Measured concentrations represent the particle-phase fraction only

The most deprived group (1–3) exhibits the widest overall range in total PAH concentrations, suggesting considerable variation in exposure levels within this group. This may reflect varying proximity to pollution sources such as busy roads and industrial areas, or greater variation in cooking and other lifestyle habits. In contrast, the moderately deprived group (4–6) displays the narrowest range, indicating more consistent exposure levels across households in this category.

The least deprived group (7–10) demonstrates the widest interquartile range (IQR), meaning the middle 50% of values are more dispersed. This pattern could suggest that even among less deprived households, variability in PAH exposure remains, potentially due to more lifestyle/behavioural-related choices which are independent of socioeconomic status .

Despite these differences in spread, the median total PAH concentrations remain similar across all three groups, reinforcing the earlier observation that deprivation level, as measured by IMD, does not appear to have a strong or consistent influence on median exposure to PAHs within these samples. This is supported by the statistical analysis, which showed no evidence of a difference between groups ($p = 0.74$).

Nevertheless, the variation in data across deprivation categories warrants further study, as it may reflect underlying environmental inequalities within IMD bands that are not fully

captured due to the relatively small sample size.

### Housing Tenure Groups

The households studied fell under three categories for housing tenure groups groups: Owned (includes mortgaged homes), Rented (from local council, housing association/trust or private landlord) and Other. The distribution of households across these groups was as follows:

- Owned (n=27)
- Rented (n=6)
- Other (n=1)

Due to the Other group containing only a single household, it was excluded from subsequent analysis. Figure 69 shows LECR values across the three housing tenure groups.

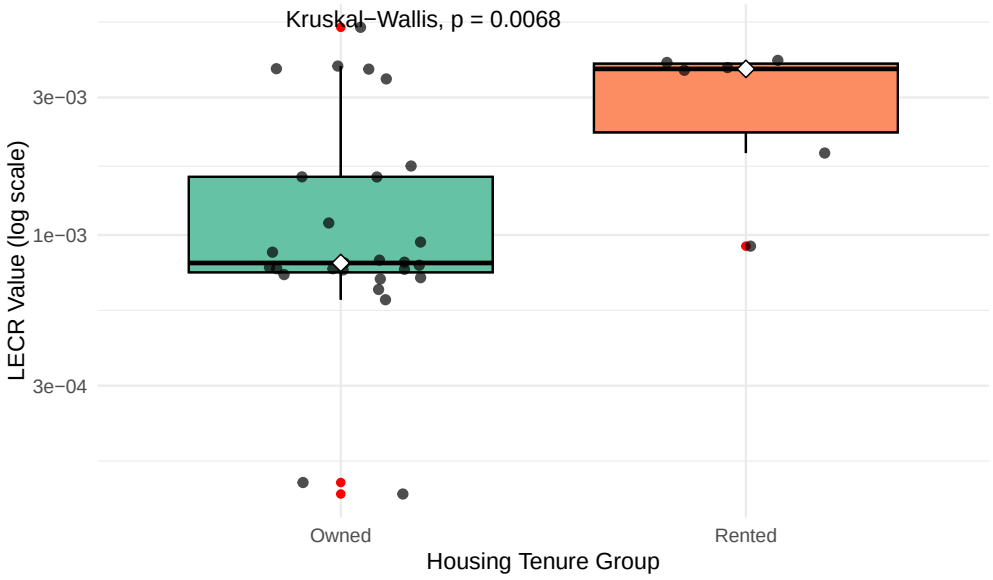



**Figure 69:** Boxplot showing LECR across Housing Tenure Groups

Households within the Rented group exhibited higher LECR values compared to those in the Owned category. However, these differences may be significantly influenced by the differences in group size. The dominance of Owned households (n = 27) may limit the statistical robustness of these inter-group comparisons.

However, the median value for the Rented group still falls outside the IQR for the Owned group, which does suggest that there may be some relationship between housing tenure group

and LECR, perhaps linked to socioeconomic factors. Nevertheless, this difference was not statistically significant (Kruskal–Wallis test, p = 0.068), indicating that there is insufficient evidence to confirm a systematic relationship between housing tenure and LECR in this dataset. Given the small sample size, further investigation with a larger and more balanced cohort would be necessary to determine whether housing tenure is a significant predictor of PAH exposure and its association with an increased LECR.

When the same stratification is applied using total PAH concentration values instead of estimated LECR, a more distinct pattern emerges between the housing tenure groups. As seen in Figure 70, households in the Rented category exhibit a noticeably wider range of total PAH concentrations, as well as a substantially higher median concentration (92.6 ng/m$^3$), compared to the Owned (23.4 ng/m$^3$) group.

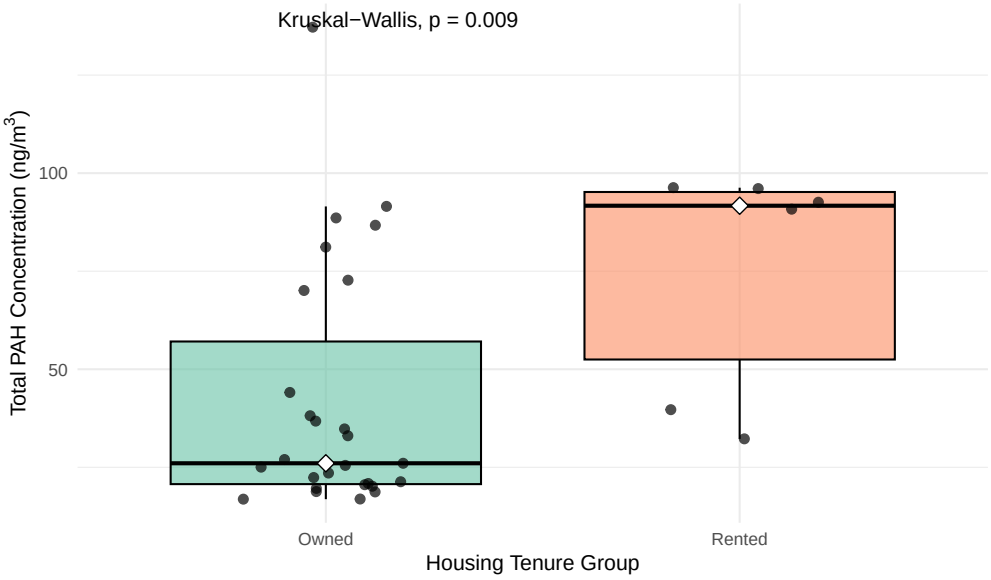



**Figure 70:** Boxplot showing Total PAH Concentrations across Housing Tenure Groups. Measured concentrations represent the particle-phase fraction only

This broader range in the Rented group may suggest greater diversity in environmental conditions within rented properties. This is potentially caused by factors such as proximity to pollution sources or behavioral factors such as cooking and ventilation practices. The elevated median further supports the possibility that households who rent may be experiencing higher levels of PAH exposure than those in owned properties. Notably, the 25th percentile of the total PAH concentrations for the Rented group is above the 75th percentile for the Owned group, suggesting that even the lower end of exposures in rented homes exceeds the higher end of those measured in owned properties, indicating a systematic difference in indoor pollutant burden between the two housing types. A statistically significant difference in total PAH

concentrations was observed between housing tenure groups (Kruskal–Wallis test, $p = 0.009$), providing evidence of variation in exposure between rented and owned households.

Nonetheless, the disparity in total PAH concentrations, particularly the elevated values within the Rented group, raises important questions about potential environmental inequalities associated with socioeconomic status. These findings align with existing literature suggesting that lower-income rental housing may be disproportionately affected by indoor and outdoor environmental exposures due to factors such as poorer insulation, older infrastructure, and closer proximity to pollution sources.[143]. However, it is unclear whether the households in the Rented group fall within the same conditions (whether socioeconomic or environmental) as those of the urban public housing developments which formed the basis of these studies; as such, these conclusions are very tentative.

Therefore, the combined analysis of total PAH concentrations suggests an association between housing tenure and environmental exposure risk, particularly among rented properties. Future research involving a larger cohort would be necessary to further assess these trends and explore possible implications for public health and housing policy.

**Smoking Status**

Smoking is a well-established and significant source of PAHs in indoor environments, and particularly benzo [a] pyrene [57]. As such, evaluating the relationship between household smoking status and both LECR and total PAH concentrations was a necessary part of this analysis.

As part of the pre-sampling questionnaires, participants were asked to provide information about smoking behaviour in their household, including both frequency and location. Smoking status was classified into two distinct categories: indoor smoking and outdoor smoking. Within each of these categories, most participants clearly fell into one of two groups: those who reported never smoking and those who reported smoking on a daily basis. Participants who smoked only occasionally or infrequently represented a very small proportion of the sample and were not numerous enough to justify separate analysis.

To enable a more robust comparison of smoking-related effects on indoor PAH levels and associated health risks, the indoor and outdoor smoking data were consolidated into a single binary variable. In addition; previous analysis indicated no significant differences between indoor and outdoor smokers. This allowed households to be grouped into two broader cate-

gories: "smokers" (any frequency of smoking, whether indoors or outdoors) and "non-smokers" (participants who reported never smoking in either situation). This binary stratification facilitated clearer analysis of the relationship between smoking behaviour and total PAH concentrations and LECR across households.

The distribution of smokers and non-smokers in the households studied is as follows:

- Smoker (n=11)
- Non-smoker (n=23)

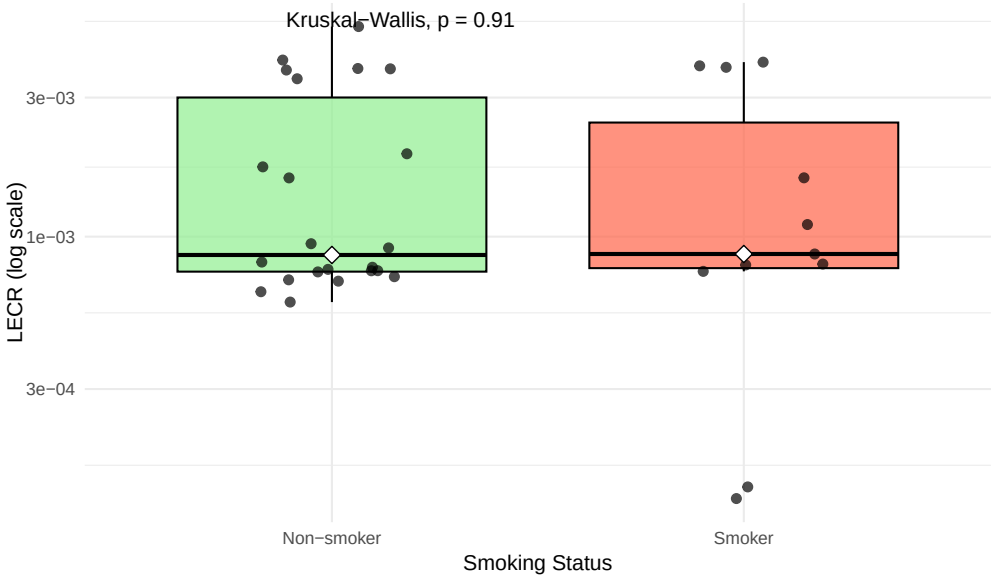



**Figure 71:** Boxplot showing LECR by Smoking Status

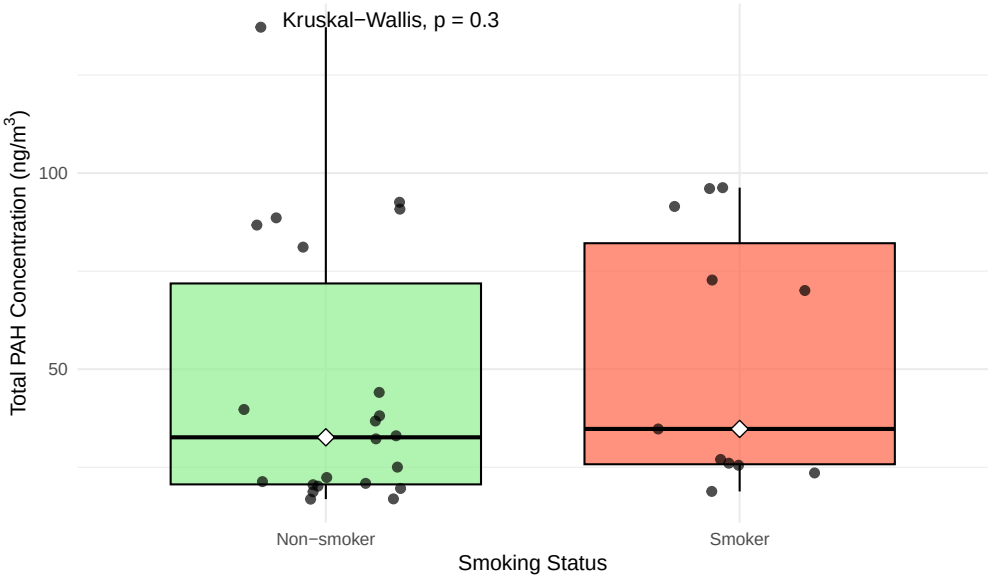



**Figure 72:** Boxplot showing Total PAH Concentrations by Smoking Status. Measured concentrations represent the particle-phase fraction only

Figures 72 and 71 present comparative analyses of total PAH concentrations and LECR values between smoker and non-smoker households. Contrary to expectations, the results reveal no clear or consistent differences between the two groups. Both total PAH levels and LECR values appear to be similarly distributed across smoker and non-smoker households, with comparable medians and interquartile ranges. It should be noted that minor differences were observed in the range of values between the two groups. The non-smoker group exhibited a slightly higher maximum value for both total PAH concentration and LECR, while the smoker group had a lower minimum LECR value. However, these variations were minimal and are unlikely to represent meaningful distinctions between the groups. No statistically significant differences were observed between smoking and non-smoking households for either total PAH concentrations ($p = 0.30$) or LECR ($p = 0.91$) (Kruskal–Wallis tests), indicating no evidence of variation in exposure risk between the groups.

This suggests that, within the context of this dataset, smoking behaviour may not be a primary determinant of overall PAH exposure or estimated cancer risk in the home environment. This finding could be caused by several factors. Firstly, the relatively small number of smoking households (n=11) may limit the ability to detect subtle differences. Secondly, it is possible that other environmental factors, such as infiltration from outdoor sources, were more influential sources of PAHs in these households than smoking activity. Lastly, the questionnaire did not ask how many participants in each household engaged in smoking

activity; the questionnaire was filled out by one person representing the household. As such, the responses may not accurately reflect the true extent or frequency of smoking activity within each household.

### Cooking Fuel Categories

Cooking fuel type plays a role in determining indoor concentrations of PAHs, as they are formed as byproducts of incomplete combustion[135]. Cooking with gas burners has been associated with elevated levels of particle emissions[144].

In contrast, cooking with electrical sources does not typically result in the direct generation of PAHs because it does not involve combustion. However, PAHs may be produced produced during high-heat cooking processes like deep frying, roasting, or grilling, regardless of cooking fuel type. Analysis of the data by cooking fuel type was deemed necessary to determine potential effects of cooking fuel type on LECR and total PAH concentrations across households.

The distribution of cooking fuel type across households is as follows:

- Electricity (n=8)
- Gas Mains (n=21)
- Both (n=5)

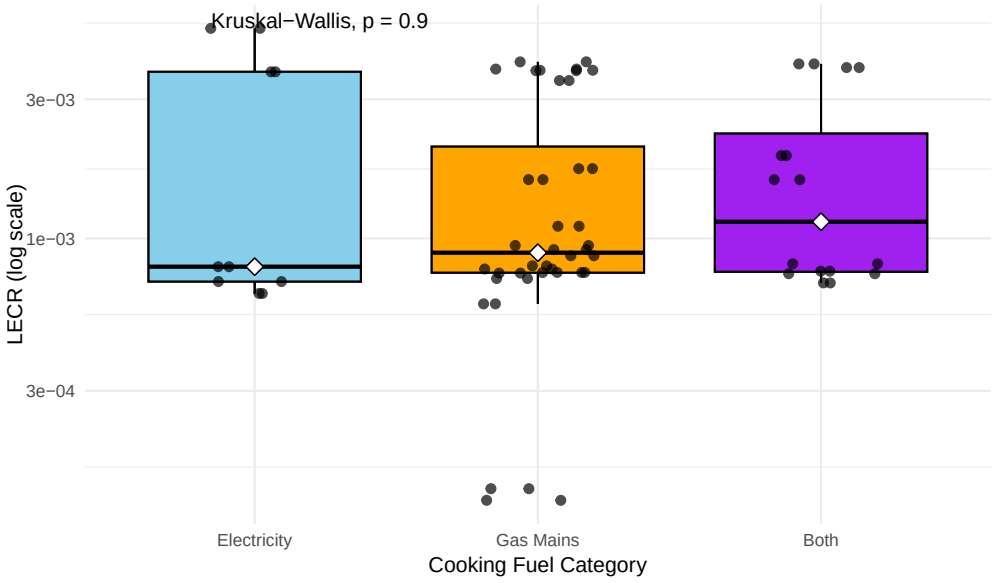



**Figure 73:** Boxplot showing LECR across Cooking Fuel Categories

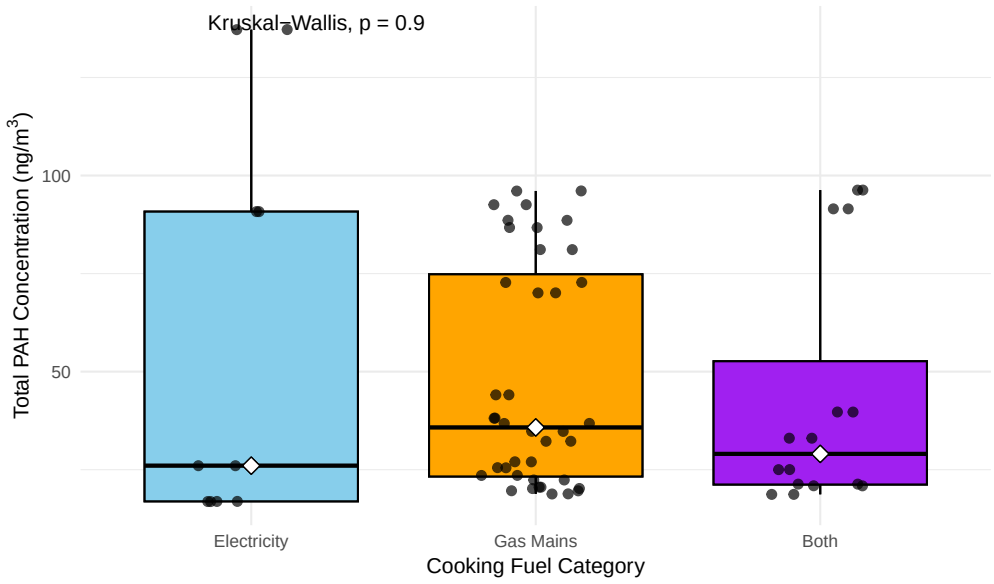



**Figure 74:** Boxplot showing Total PAH Concentrations across Cooking Fuel Category Groups. Measured concentrations represent the particle-phase fraction only

In both Figures 73 and 74, the variation in estimated lifetime excess cancer risk (LECR) and total PAH concentrations across cooking fuel types is minimal. In Figure 73, which shows LECR values, the Gas Mains group exhibits a slightly broader range compared to the Electricity and Other categories. However, the interquartile ranges and median values across all three groups remain quite consistent, suggesting no substantial differences in LECR attributable to cooking fuel type. Similarly, in Figure 74, the Electricity group displays a somewhat wider range of total PAH concentrations, though the median and interquartile range also remain comparable to those of the Gas Mains and Other groups.

These findings suggest that cooking fuel type may not be a major driver of variation in PAH exposure or elevated cancer risk in the households studied. No statistically significant differences were observed between cooking fuel categories for either total PAH concentrations or LECR (Kruskal–Wallis tests, $p = 0.90$ for both), supporting this suggestion.

### Heating Types

Although the type of heating used in homes was initially considered a critical variable in analysing total PAH concentrations and LECR, it became apparent during the analysis phase that stratifying households by heating type would not produce reliable or meaningful results. One key limitation was the relatively small sample size, which significantly restricted the statistical power when dividing households into multiple heating categories. In addition, many households reported using more than one type of heating method, further complicating classification and reducing the number of cases within each distinct category.

In the pre-sampling questionnaire, participants were asked to indicate all heating methods used within their household. While this provided insight into the variety of heating sources present, there was no information reported regarding which methods were used for the duration of the sampling period. Initially, this information was meant to be obtained through the Daily Diaries that participants were meant to fill out; however, this measure was ultimately unsuccessful. Without temporal specificity, it was not possible to determine the relative contribution of each heating method to the observed indoor PAH concentrations during sampling. Additional analysis was carried out on the homes where the presence of wood-burning stoves was reported; these homes were not unique from homes without wood-burning stoves and as such, further analysis was not carried out as it was hypothesised that the stoves had not been in use during the sampling period.

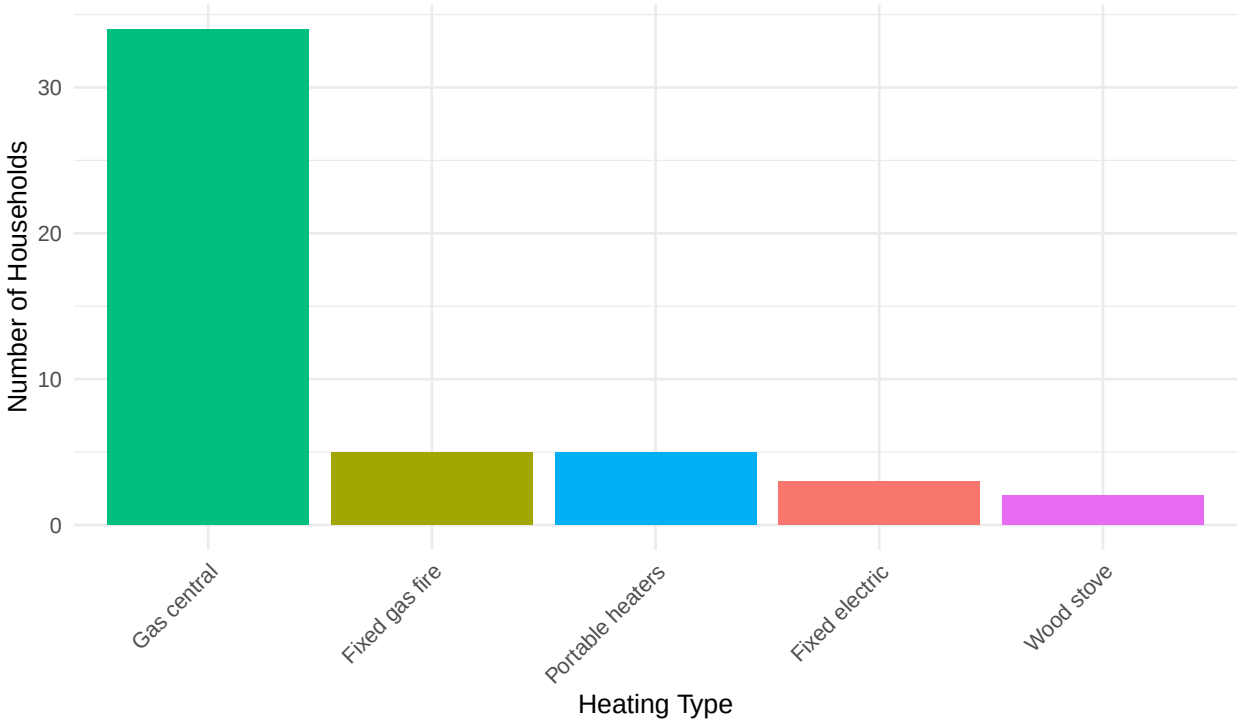



**Figure 75:** Bar plot showing the distribution of heating types across households

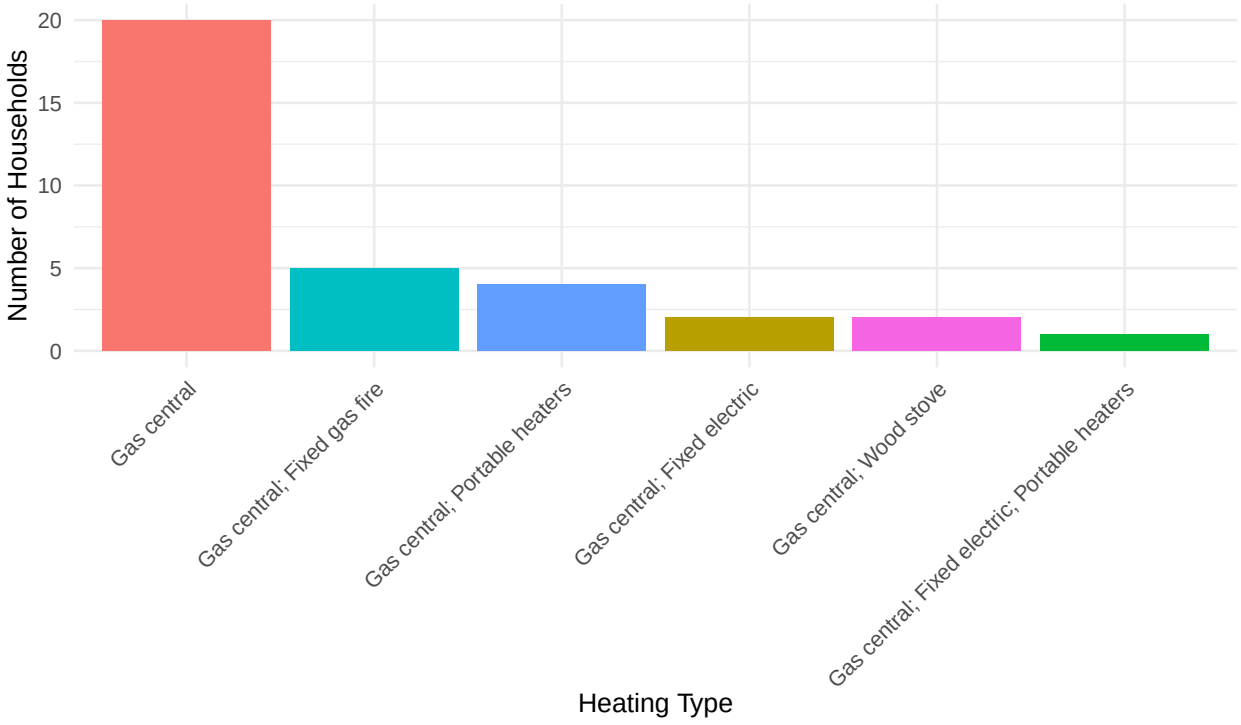



**Figure 76:** Bar plot showing the distribution of heating types across households

These limitations meant that a meaningful stratified analysis by heating type could not be conducted. Additionally, attempts to isolate the individual heating methods for analysis were also rendered unfeasible due to these constraints. The results of the initial attempts to stratify households by heating type are presented in Figures 75 and 76. It was clear even at the preliminary stage that further analysis was unlikely to be fruitful. As a result, heating type was ultimately excluded from the final comparative analysis.

## 4.2 Phthalates

Phthalates are compounds which are commonly used as plasticizers, which are added to plastics and polyvinyl chloride (PVC) to increase their flexibility, durability, and longevity[145]. They are widely found in consumer products such as vinyl flooring, personal care products, household furnishings, medical devices, and food packaging[146]. Due to their widespread use and tendency to leach out of products over time, phthalates are ubiquitous in indoor environments[147].

Human exposure to phthalates can occur through ingestion, dermal contact, or inhalation[147]. Phthalates can attach to airborne particles or exist in the gas phase, especially in poorly ventilated environments. Indoor sources include building materials, personal care products, cleaning products, and off-gassing from furnishings, upholstery and electronics[147].

Exposure to phthalates is of concern due to their potential to cause health effects. Several phthalates are known endocrine-disrupting chemicals (EDCs)[147]. Health effects associated with phthalate exposure include respiratory, reproductive and developmental toxicity[145, 146]. Children, pregnant individuals, and people with pre-existing health conditions may be especially vulnerable due to their higher relative exposure levels and/or greater sensitivity[148].

Several phthalates were found in the INGENIOUS $PM_{2.5}$ samples and a summary of the no of times detected, mean, min, max and standard deviation in ng/m$^3$ are shown in Table 13.

**Table 13:** Summary statistics of phthalates found in home samples

| Name | No. of Samples Detected | Mean (ng/m³) | Min (ng/m³) | Max (ng/m³) | SD |
|---|---|---|---|---|---|
| Benzyl butyl phthalate | 15 | 2.5 | 0.0 | 14.4 | 3.6 |
| Bis(2-ethylhexyl) phthalate | 25 | 129.0 | 0.0 | 358.3 | 111.2 |
| Di-n-octyl phthalate | 6 | 2.8 | 0.0 | 12.3 | 3.9 |
| Dibutyl phthalate | 34 | 233.3 | 18.1 | 483.8 | 126.1 |
| Diethyl phthalate | 29 | 29.2 | 0.0 | 334.6 | 68.8 |
| Dimethyl phthalate | 23 | 1.9 | 0.0 | 8.0 | 2.1 |

A summary of the distribution of phthalates in the INGENIOUS samples is shown in Figure 77

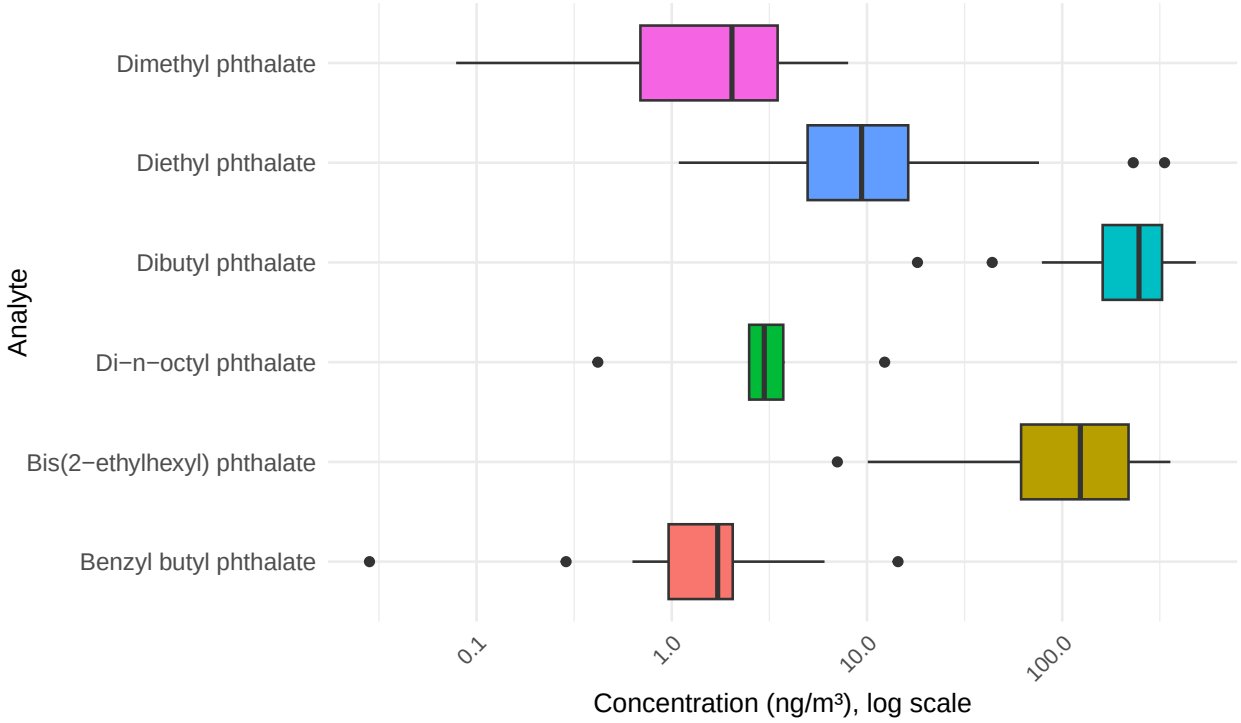



**Figure 77:** Boxplot showing the concentrations of phthalates found in samples

Phthalate concentrations across the sampled houses shows substantial variability in both detection frequency and concentration levels. Dibutyl phthalate exhibited the highest mean concentration (245.301 ng/m³) of all measured phthalates and was detected in all of the samples (n=34). This high prevalence and concentration suggest widespread sources and/or persistence in the sampled environments. Bis(2-ethylhexyl) phthalate (DEHP), although present in 25 samples, showed the second-highest mean concentration (128.995 ng/m³) but also a wide range (0–358.279 ng/m³) and a high standard deviation (111.187 ng/m³), which

indicates a lot of variability in its prevalence. DEHP is one of the most widely used plasticizers globally, being primarily added to PVC and plastics to give flexibility and durability[105]. Due to its extensive use in products such as building materials, food packaging, and wire insulation, DEHP is also one of the most commonly detected phthalates in environmental monitoring studies[105].

Diethyl phthalate was detected in 29 samples, with a a very broad range (mean=29.215 ng/m$^3$) and a large standard deviation (68.779 ng/m$^3$). This pattern suggests occasional high-level events or specific emission sources. Benzyl butyl phthalate, di-n-octyl phthalate, and dimethyl phthalate were generally present at lower mean concentrations (2–3 ng/m$^3$) and detected in fewer samples, though maximum values for some, e.g. di-n-octyl phthalate (12.317 ng/m$^3$), indicate that concentrations were high in a small number of households.

Overall, the results highlight that while some phthalates are both prevalent and occur at elevated concentrations, others are less widespread and typically present at much lower levels.

Figure 78 shows the sum of the concentrations of the targeted phthalates in each home.

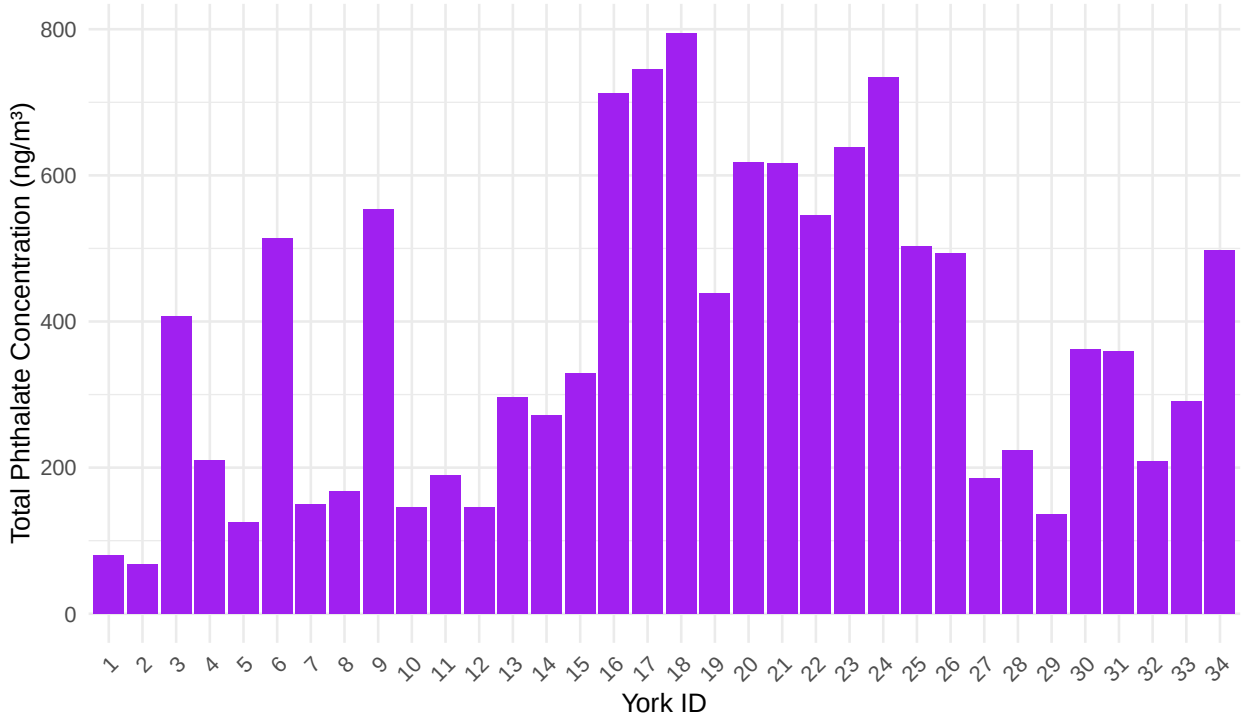
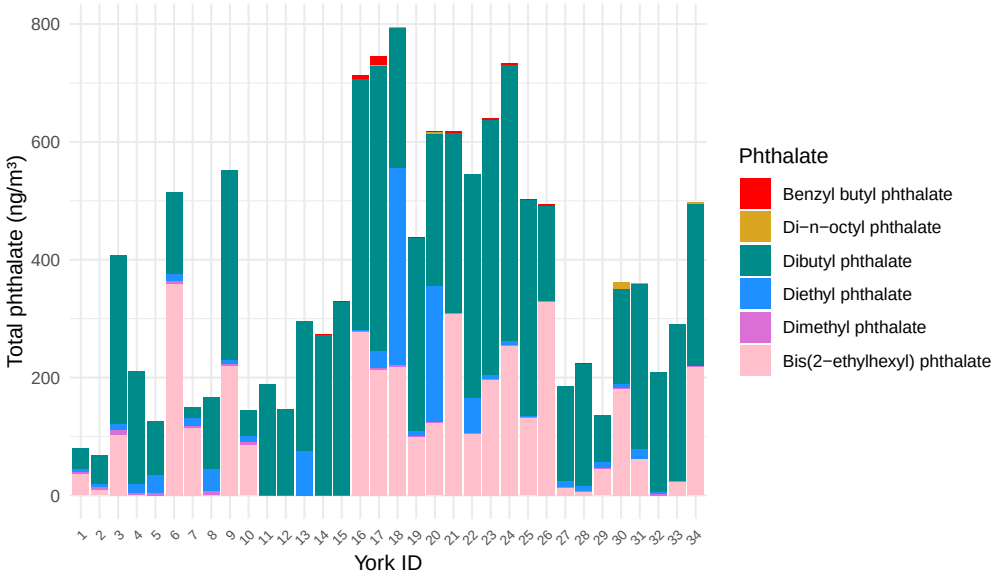


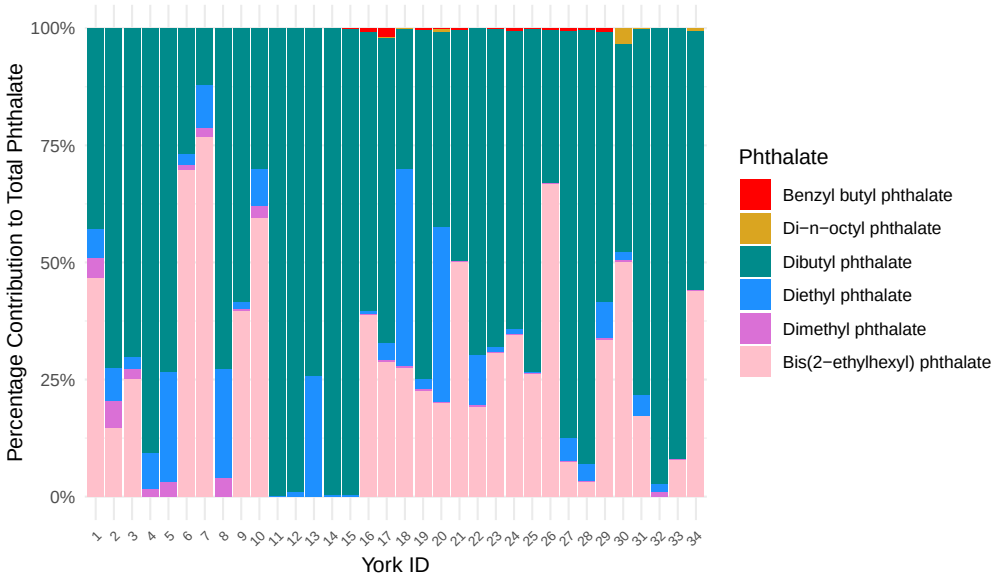

**Figure 78:** Bar plot showing the total concentration of phthalates found in samples

There is substantial variability among sites; while some homes report relatively low concentrations, others exhibit markedly elevated levels (min= 68 ng/m$^3$, max= 795 ng/m$^3$, mean= 375 ng/m$^3$), suggesting significant heterogeneity in indoor environments and/or emission sources.

Figures 79a and 79b illustrate the relative contributions of individual phthalates to the total phthalate concentration across all samples, expressed both in concentrations and as percentages.




**(a)** Stacked bar plot showing the absolute contribution of each phthalate across samples




**(b)** Stacked bar plot showing the percentage contribution of each phthalate across samples

**Figure 79:** Relative Contributions of Phthalates to Total Pthalate Concentration Across Samples

The results demonstrate that bis(2-ethylhexyl) phthalate (DEHP) and dibutyl phthalate (DBP) are the dominant contributors in all households. Diethyl phthalate (DEP) also appears with some consistency, though at distinctly lower concentrations compared to DEHP and DBP.

When calculated as percentage contributions, dibutyl phthalate clearly emerges as the single largest contributor, accounting for the majority share of total phthalate concentration in most (but not all) households. Bis(2-ethylhexyl) phthalate consistently represents the second-largest proportion (when not the largest contributor), while the contributions of other phthalates are negligible by comparison. This pattern is especially evident in Figure 79b, which highlights the importance of these highly-represented phthalates in shaping overall exposure levels.

These findings indicate that indoor phthalate exposure within the sampled households is largely driven by one or two dominant compounds, rather than an even mixture across multiple phthalates. This finding is consistent with previous studies that have identified DBP and DEHP as the most widespread phthalates in indoor environments, reflecting their extensive previous and ongoing use in plastics, consumer products, and building materials[105, 149]).

**Phthalate Exposure Risk Assessment**

In the field of toxicology, DEHP has been extensively studied and is often used as a reference compound for evaluating phthalate toxicity; its environmental persistence and ability to leach from products into air, dust, water, and food make it a frequent target compound for exposure assessment[61]. This is due to its endocrine-disrupting properties, and in particular its ability to interfere with reproductive hormone regulation[146]. DEHP has been shown to disrupt androgen synthesis, impair sperm production, and alter reproductive development in both humans and in animal models[150]. Because of this, DEHP frequently serves as a benchmark compound in comparative toxicity studies, with other phthalates assessed relative to its toxicity[61].

In addition, regulatory agencies such as the U.S. EPA have classified DEHP as a reproductive toxicant and probable carcinogen, and its toxicological profile has been used to develop risk assessment frameworks for the phthalate group as a whole[61].

Here, DEHP will be employed as the representative compound for calculating inhalation exposure and estimating the incremental lifetime cancer risk (ILCR) due to phthalate exposure for the households included in this study. The inhalation exposure equation provides a means of estimating the daily dose of a contaminant that an individual receives through breathing indoor air[103].

$$IE = \frac{C \times IR \times CF \times ED}{BW}$$

(Equation 5)

Where:
IE= inhalation exposure dose ($\mu$g/(kg $\cdot$ d))
C= total concentration of phthalates in indoor air ($\mu$g/m$^3$)
IR= is inhalation rate (m$^3$/h)
CF= is contact factor, 0.9
ED= is exposure duration (h/d)
BW=is body weight (kg)

Once the inhalation exposure dose has been calculated, the incremental cancer risk can be estimated using a linear dose–response function. This is shown in Equation 4

$$R = q \times E$$

(Equation 4)

Where:
q= 95% upper confidence limit of the linearized cancer slope factor of the dose–response function $(\mathrm{mg\,(kg\,}d)^{-1})^{-1}$, which is 0.014 for DEHP
E= exposure dose (mg/(kg $\cdot$ d))

These equations are applied across multiple age groups in order to account for potential variability in susceptibility to inhalation exposure and its associated cancer risk. Physiological differences between age groups play a critical role in determining relative exposure levels. Younger children inhale a greater volume of air relative to their body weight when compared to adults; this results in proportionally higher intake of airborne contaminants under equal concentrations[103]. This increased inhalation rate combined with their lower body mass

generally makes children more vulnerable to adverse health outcomes from exposure to indoor pollutants, in this case phthalates.

The U.S. Environmental Protection Agency (EPA) has established a threshold for acceptable incremental cancer risk at $1 \times 10^{-6}$, meaning that one individual out of every million will develop cancer based on their exposure to their environment.

Table 14 shows the calculated inhalation exposure and incremental cancer risk values from exposure to measured DEHP concentrations across age groups in one home, York ID=18. Here, the increased exposure in children can be seen, as the first five age groups have cancer risk values exceeding the EPA limit of $1 \times 10^{-6}$, with these values at their highest in the 0-<1 age group, and gradually descending until they fall below the limit in the 11-<16 group.

**Table 14:** Inhalation Exposure and Incremental Cancer Risk for DEHP in York ID=18 by Age Group

| Age Group (years) | Inhalation Exposure (mg/kg/day) | Cancer Risk |
|---|---|---|
| 0-<1 | 1.15e-04 | 1.61e-06 |
| 1-<2 | 1.38e-04 | 1.93e-06 |
| 2-<3 | 1.27e-04 | 1.77e-06 |
| 3-<6 | 1.07e-04 | 1.49e-06 |
| 6-<11 | 7.42e-05 | 1.04e-06 |
| 11-<16 | 5.26e-05 | 7.36e-07 |
| 16-<21 | 4.47e-05 | 6.26e-07 |
| 21-<31 | 3.86e-05 | 5.40e-07 |
| 31-<41 | 3.93e-05 | 5.50e-07 |
| 41-<51 | 3.93e-05 | 5.50e-07 |
| 51-<61 | 3.86e-05 | 5.40e-07 |
| 61-<71 | 3.49e-05 | 4.88e-07 |
| 71-<81 | 3.17e-05 | 4.44e-07 |
| 81+ | 3.00e-05 | 4.20e-07 |

It must be noted that for the purposes of calculating inhalation exposure and incremental

cancer risk, it was assumed that participants spent 100 percent of their time in the kitchen as that was the only location in which the particulate matter samplers were placed. The lack of concentration data for the other rooms in the homes made it impossible to apply the more nuanced time-use profiles recommended in the U.S. EPA Exposure Factors Handbook, specifically those detailing the proportion of time individuals spend in various indoor microenvironments (e.g., living areas, bedrooms, kitchens)

In addition, the handbook's time-use tables primarily cover children under 21 years of age, and do not include established recommendations for adults over 21, further limiting the applicability of this method for an adult population. As a result, the inhalation exposure calculations in this study were methodologically necessary, but have the caveat of potential over- or under-estimation of exposure that may result from the assumed 100 percent kitchen occupancy during the sampling period.

Table 15 shows the estimated inhalation exposure to DEHP and the corresponding incremental cancer risk for the 34 households studied. Mean exposure and carcinogenic risk represent the average daily exposure and carcinogenic riskacross all age groups, whereas maximum exposure and carcinogenic risk represent the highest exposure and carcinogenic risk observed in the most vulnerable age group, in this case infants aged 0-1 years.

**Table 15:** DEHP Inhalation Exposure and Incremental Cancer Risk by York ID

| York ID | Mean Exposure (mg/kg/day) | Max Exposure (mg/kg/day) | Mean Carcinogenic Risk | Max Carcinogenic Risk |
|---|---|---|---|---|
| 1 | 5.12e-06 | 5.53e-05 | 7.16e-08 | 7.75e-07 |
| 2 | 5.19e-06 | 6.04e-05 | 7.27e-08 | 8.45e-07 |
| 3 | 6.81e-05 | 7.96e-04 | 9.53e-07 | 1.11e-05 |
| 4 | 6.05e-05 | 8.37e-04 | 8.47e-07 | 1.17e-05 |
| 5 | 8.92e-05 | 1.08e-03 | 1.25e-06 | 1.51e-05 |
| 6 | 1.22e-04 | 1.51e-03 | 1.71e-06 | 2.12e-05 |
| 7 | 7.72e-05 | 8.60e-04 | 1.08e-06 | 1.20e-05 |
| 8 | 7.07e-05 | 8.29e-04 | 9.90e-07 | 1.16e-05 |
| 9 | 9.81e-05 | 1.21e-03 | 1.37e-06 | 1.70e-05 |
| 10 | 8.45e-05 | 1.15e-03 | 1.18e-06 | 1.61e-05 |
| 11 | 2.35e-05 | 2.53e-04 | 3.29e-07 | 3.54e-06 |
| 12 | 1.31e-05 | 1.28e-04 | 1.83e-07 | 1.79e-06 |
| 13 | 2.94e-05 | 4.52e-04 | 4.12e-07 | 6.33e-06 |
| 14 | 4.24e-05 | 4.04e-04 | 5.93e-07 | 5.65e-06 |
| 15 | 3.97e-05 | 8.73e-04 | 5.56e-07 | 1.22e-05 |
| 16 | 2.76e-05 | 2.84e-04 | 3.87e-07 | 3.97e-06 |
| 17 | 1.89e-05 | 3.88e-04 | 2.64e-07 | 5.43e-06 |
| 18 | 1.87e-05 | 2.12e-04 | 2.61e-07 | 2.96e-06 |
| 19 | 2.15e-05 | 2.13e-04 | 3.01e-07 | 2.98e-06 |
| 20 | 2.21e-05 | 4.38e-04 | 3.09e-07 | 6.13e-06 |
| 21 | 2.17e-05 | 1.95e-04 | 3.04e-07 | 2.74e-06 |
| 22 | 2.15e-05 | 5.72e-04 | 3.01e-07 | 8.00e-06 |
| 23 | 2.21e-05 | 3.52e-04 | 3.09e-07 | 4.92e-06 |
| 24 | 2.01e-05 | 4.93e-04 | 2.82e-07 | 6.90e-06 |
| 25 | 1.91e-05 | 2.32e-04 | 2.68e-07 | 3.25e-06 |
| 26 | 2.21e-05 | 2.08e-04 | 3.09e-07 | 2.91e-06 |
| 27 | 1.06e-05 | 1.02e-04 | 1.49e-07 | 1.43e-06 |
| 28 | 1.26e-05 | 1.31e-04 | 1.76e-07 | 1.84e-06 |
| 29 | 1.22e-05 | 1.38e-04 | 1.71e-07 | 1.93e-06 |
| 30 | 5.07e-05 | 3.57e-04 | 7.10e-07 | 4.99e-06 |
| 31 | 2.13e-05 | 1.78e-04 | 2.99e-07 | 2.49e-06 |
| 32 | 9.04e-05 | 1.71e-03 | 1.27e-06 | 2.39e-05 |
| 33 | 2.13e-05 | 2.37e-04 | 2.98e-07 | 3.32e-06 |
| 34 | 3.59e-05 | 3.11e-04 | 5.02e-07 | 4.35e-06 |

Incremental cancer risk estimates are a direct result of these exposure levels. Mean carcinogenic risk ranges from $1.49 \times 10^{-7}$ to $1.71 \times 10^{-6}$, and maximum risk ranges from $1.43 \times 10^{-6}$ to $2.39 \times 10^{-5}$. Notably, several households, including York IDs 3–6, 9–10, and

32, exhibit maximum cancer risk values above the EPA benchmark of $1 \times 10^{-6}$, primarily due to the high exposures estimated in infants. These households also had some of the highest LECR values calculated from exposure to PAHs in their homes.

However, this observation requires further consideration. While PAHs were hypothesised to be influenced in part by outdoor sources, phthalates are generally associated with indoor emissions from consumer products and building materials. As such, it is not expected that both compound classes would exhibit elevated concentrations in the same households. The co-occurrence of high PAH- and phthalate-derived risk in these homes therefore suggests that multiple factors may be contributing simultaneously.

It is possible that the observed similarity in cancer risk across these households arises from different calculations of exposures. For example, some homes may exhibit relatively low concentrations of the more toxic PAHs, while others may contain higher concentrations of less toxic compounds or phthalates, resulting in comparable overall risk estimates. This would suggest that the apparent overlap in high-risk households is not necessarily indicative of a shared source.

Table 4.2 presents the mean DEHP inhalation exposure and the corresponding estimated incremental cancer risk across different age groups; these age groups were outlined in the US EPA Exposure Factors Handbook. Mean exposure represents the average daily exposure for each age group across all York IDs. The "Exceeds EPA Limit?" column is based on the mean carcinogenic risk, which was calculated based on age group across all households.

**Table 16:** Average DEHP Inhalation Exposure and Cancer Risk by Age Group

| Age Group (years) | Mean Exposure (mg/kg/day) | Carcinogenic Risk | Exceeds EPA Limit? |
|---|---|---|---|
| 0-<1 | 7.23e-05 | 1.01e-06 | Yes |
| 1-<2 | 8.65e-05 | 1.21e-06 | Yes |
| 2-<3 | 7.95e-05 | 1.11e-06 | Yes |
| 3-<6 | 6.69e-05 | 9.37e-07 | No |
| 6-<11 | 4.65e-05 | 6.51e-07 | No |
| 11-<16 | 3.30e-05 | 4.62e-07 | No |
| 16-<21 | 2.81e-05 | 3.93e-07 | No |
| 21-<31 | 2.42e-05 | 3.39e-07 | No |
| 31-<41 | 2.46e-05 | 3.45e-07 | No |
| 41-<51 | 2.46e-05 | 3.45e-07 | No |
| 51-<61 | 2.42e-05 | 3.39e-07 | No |
| 61-<71 | 2.19e-05 | 3.06e-07 | No |
| 71-<81 | 1.99e-05 | 2.78e-07 | No |
| 81+ | 1.88e-05 | 2.63e-07 | No |

The results show that DEHP exposure is highest among the youngest age groups, with infants (0–<1 years) experiencing a mean exposure of $7.23 \times 10^{-5}$ mg/kg/day and an associated cancer risk of $1.01 \times 10^{-6}$. Exposure remains elevated in toddlers and young children (1–<6 years), gradually decreasing with age. Adults (21–<81 years) have lower mean exposures, ranging from $2.19 \times 10^{-5}$ to $2.46 \times 10^{-5}$ mg/kg/day, with corresponding cancer risks between $3.06 \times 10^{-7}$ and $3.45 \times 10^{-7}$.

Notably, the estimated incremental cancer risk exceeds the EPA threshold of $1 \times 10^{-6}$ only for infants and very young children (0–<3 years), while all other age groups remain below the threshold but still demonstrate measurable risk. This trend highlights that younger populations, particularly infants, are the most vulnerable to DEHP and inhalation exposures in general, emphasising the importance of targeted risk mitigation for these groups. This pattern is also seen in studies investigating the impacts of other harmful pollutants (e.g. nitrosamines) on infants and children[151].

### Correlation between DEHP and other phthalates

The potential correlations between DEHP and the other five phthalates targeted in this study were investigated to interpret exposure data in relation to a well-characterized reference compound. As aforementioned, DEHP serves as a benchmark due to its widespread use, established toxicology, and frequent inclusion in environmental assessments. By comparing other phthalates to DEHP, it can be determined whether they tend to co-occur in the same environments, which in turn aids in identifying potential shared sources.

This approach also allows for relative risk evaluation. If another phthalate shows a strong positive correlation with DEHP, it suggests that populations with high DEHP exposure are likely also experiencing elevated levels of that phthalate. This information is crucial for understanding cumulative or combined chemical risks, particularly since many phthalates have similar mechanisms of action. Conversely, weak correlations can indicate different sources or behaviors, informing more targeted mitigation strategies.

Lastly, correlating against DEHP will help in bridging findings from previous studies with new data. Because DEHP is the most widely reported phthalate in epidemiological and toxicological studies, comparing other phthalates to DEHP makes it easier to compare results across studies, integrate datasets, and identify emerging trends in human or environmental exposure. Overall, this method will strengthen the interpretability and relevance of multi-phthalate exposure assessments.

Figure 80 depicts the outcomes of the correlation analyses.

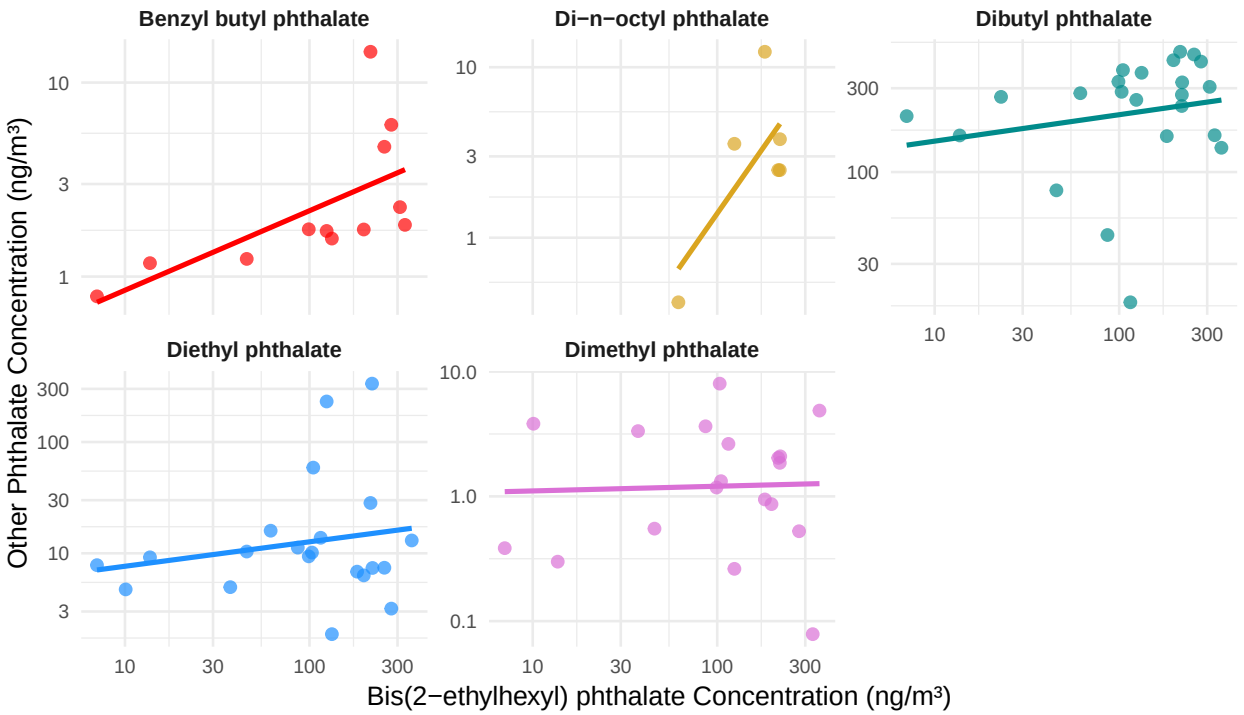



**Figure 80:** Scatter plots showing the correlation between DEHP and all other phthalates analysed, faceted

Table 17 shows the $R^2$ values obtained for each phthalate, when correlated against DEHP.

**Table 17:** $R^2$ Values for Each Phthalate when compared to DEHP

| Phthalate | $R^2$ |
|---|---|
| Benzyl butyl phthalate | 0.404 |
| Di-n-octyl phthalate | 0.506 |
| Dibutyl phthalate | 0.134 |
| Diethyl phthalate | 0.037 |
| Dimethyl phthalate | 0.001 |

The correlation analysis between DEHP and other phthalates showed varying degrees of association, with $R^2$ values ranging from 0.001 to 0.506. The highest correlation was observed for di-n-octyl phthalate ($R^2$ = 0.506), despite its relatively low detection frequency (n=6). This strong relationship, despite the low detections of di-n-octyl phthalate, may indicate that both compounds are emitted from the same sources. While benzyl butyl phthalate gave a moderate $R^2$ value ($R^2$ = 0.404), the L-shaped relationship observed in Figure 80 indicates that benzyl butyl phthalate concentrations increase alongside DEHP concentrations up to

approximately 200 ng/m$^3$, after which the association weakens.

In contrast, dibutyl phthalate, which was the most prevalent phthalate in the dataset (n=32), had a very low $R^2$ value (0.134) with DEHP, though it should be noted that it appears that a few outliers appear to be driving this. This finding suggests that although both compounds are common, they likely are produced from different sources, reducing their statistical association. Diethyl phthalate (n=15) showed an even lower correlation value ($R^2$ = 0.037), indicating even less likelihood of shared sources.

The weakest relationship was observed for dimethyl phthalate ($R^2$ = 0.001). This lack of correlation suggests that dimethyl phthalate and DEHP are emitted from entirely different sources or activities.

## 4.3 Bringing it all together

To further investigate the drivers of exposure, a subset of households was examined in more detail based on average $PM_{2.5}$ (obtained friom the sensor data), total PAH concentration, and estimated lifetime excess cancer risk (LECR), alongside phthalate-derived risk from DEHP exposure. Households with the highest mean $PM_{2.5}$ concentrations (e.g. York IDs 9, 22, 23, 24, and 18) were compared with those exhibiting the highest PAH concentrations and LECR (e.g. York IDs 7, 6, 9, 5, and 10), as well as those with the highest phthalate-related cancer risk (e.g. York IDs 6, 9, 5, 32, and 10).

Households with the highest $PM_{2.5}$ concentrations generally showed elevated phthalate levels, suggesting the presence of consistent indoor emission sources. However, the households with the highest PAH concentrations and LECR did not always correspond to those with the highest $PM_{2.5}$ levels. For example, York IDs 5 and 10 exhibited moderate $PM_{2.5}$ concentrations but elevated PAH concentrations and phthalate-derived cancer risk when compared to the rest of the hosueholds, indicating that risk in these households are influenced by chemical composition rather than total particulate mass.

This is further highlighted by households such as York ID=32, which exhibited one of the lowest mean $PM_{2.5}$ concentrations but among the highest phthalate-derived cancer risks and elevated PAH concentrations. This demonstrates that low particulate mass does not necessarily correspond to low health risk, particularly for outcomes such as cancer that are influenced by specific toxic compounds.

Overall, these findings indicate that different households may experience elevated risk through distinct pathways, including sustained high $PM_{2.5}$ concentrations, composition-driven toxicity, or intermittent emission events. This reinforces the importance of incorporating chemical composition, in addition to particulate mass, when assessing indoor air quality and its associated health risks.

## 4.4 Physicochemical Properties of PAHs and Phthalates

To better interpret the measured concentrations of analytes in particulate matter, it is necessary to consider the physicochemical properties that govern gas-particle partitioning. Many of the compounds targeted in this study are semi-volatile organic compounds (SVOCs), which exist in equilibrium between the gas and particle phases under ambient conditions[152]. As a result, the concentrations reported here represent only the particle-associated fraction and do not necessarily reflect total airborne concentrations or overall exposure.

Physicochemical properties for all detected polycyclic aromatic hydrocarbons (PAHs) and phthalates were compiled from the PubChem database and are presented in Tables 18 and 19. These data were used to assess how volatility and partitioning influence the observed concentrations.

The physicochemical properties of the detected compounds provide important context for interpreting the concentration data presented in Tables 13 and 10. Higher molecular weight PAHs such as benzo[ghi]perylene, indeno[1,2,3-cd]pyrene and dibenz[a,h]anthracene exhibit very low vapour pressures (typically $<10^{-6}$ Pa) and relatively high log $K_{ow}$ values (>6), which indicate a strong tendency to partition into the particulate phase. This is consistent with their frequent detection across samples, suggesting that the measured PM concentrations are broadly representative of their presence in indoor air.

In contrast, lower molecular weight PAHs such as the naphthalene derivatives, fluorene and acenaphthylene exhibit substantially higher vapour pressures and lower log $K_{ow}$ values, indicating a tendency towards the gas phase. The lower detection frequency and more variable concentrations observed for these compounds in particulate matter are therefore potentially a result of phase partitioning. As such, their measured concentrations in PM are not expected to be representative of total airborne exposure.

A similar trend is observed for phthalates. Compounds such as bis(2-ethylhexyl) and dibutyl phthalate, which exhibit relatively low vapour pressures and high log $K_{ow}$ values, were detected in a greater number of samples and at higher concentrations, indicating a

**Table 18:** Physicochemical properties of polycyclic aromatic hydrocarbons (PAHs) detected in INGENIOUS samples

| Compound | log $K_{ow}$ | Vapour Pressure (Pa) | Source |
|---|---|---|---|
| Fluoranthene | 5.2 | 1.23e-03 | PubChem |
| Chrysene | 5.7 | 8.31e-07 | PubChem |
| Benzo[a]pyrene | 6.0 | 7.32e-07 | PubChem |
| Phenanthrene | 4.5 | 1.61e-02 | PubChem |
| Benz[a]anthracene | 5.8 | 2.80e-05 | PubChem |
| Pyrene | 4.9 | 6.00e-04 | PubChem |
| Fluorene | 4.2 | 8.00e-02 | PubChem |
| Anthracene | 4.4 | 3.56e-04 | PubChem |
| Benzo[ghi]perylene | 6.6 | 1.33e-08 | PubChem |
| Dibenz[a,h]anthracene | 6.5 | 1.27e-07 | PubChem |
| Benzo[b]fluoranthene | 6.4 | 6.67e-05 | PubChem |
| Benzo[k]fluoranthene | 6.4 | 1.28e-08 | PubChem |
| Indeno[1,2,3-cd]pyrene | 7.0 | 1.73e-08 | PubChem |
| Acenaphthene | 3.9 | 2.87e-01 | PubChem |
| Acenaphthylene | 3.7 | 8.91e-01 | PubChem |
| Naphthalene | 3.3 | 1.07e+01 | PubChem |
| Naphthalene, 1-methyl- | 3.9 | 8.93e+00 | PubChem |
| Naphthalene, 2-methyl- | 3.9 | 7.33e+00 | PubChem |
| Carbazole | 3.7 | 1.83e-04 | PubChem |
| Dibenzofuran | 4.1 | 3.31e-01 | PubChem |

**Table 19:** Physicochemical properties of phthalates detected in INGENIOUS samples

| Compound | log $K_{ow}$ | Vapour Pressure (Pa) | Source |
|---|---|---|---|
| Dimethyl phthalate | 1.6 | 1.33e+00 | PubChem |
| Bis(2-ethylhexyl) phthalate | 7.4 | 1.30e-03 | PubChem |
| Diethyl phthalate | 2.5 | 2.80e-01 | PubChem |
| Dibutyl phthalate | 4.7 | 2.68e-03 | PubChem |
| Benzyl butyl phthalate | 4.9 | 1.10e-03 | PubChem |
| Di-n-octyl phthalate | 9.1 | 3.47e-04 | PubChem |

stronger tendency to stay in the particle phase. In contrast, more volatile phthalates such as dimethyl phthalate and diethyl phthalate , which have higher vapour pressures, showed lower concentrations and greater variability, consistent with partial partitioning into the gas phase.

These observations highlight that differences in measured concentrations in particulate matter may not necessarily correspond to differences in total exposure. Rather, they may reflect gas–particle partitioning, which is governed by compound-specific physicochemical properties.

## 4.5 Conclusions

The analysis of polycyclic aromatic hydrocarbons investigated chemical toxicity as well as concentrations. Although overall concentrations varied considerably across households, carcinogenic PAHs such as dibenz [a,h] anthracene contributed disproportionately to lifetime excess cancer risk. Surprisingly, households with smokers did not consistently show higher total PAH concentrations, with some non-smoker households exhibiting comparable or even higher burdens; however, no statistically significant differences were observed between smoking groups for either total PAHs or LECR (Kruskal–Wallis tests, $p = 0.30$ and $p = 0.91$, respectively). Similarly, attempts to differentiate PAH exposure by heating or cooking fuel type produced inconclusive results, with no statistically significant differences observed. In contrast, housing tenure showed a statistically significant difference in total PAH concentrations ($p = 0.009$), and a statistically significant difference in $PM_{2.5}$ concentrations was also observed between urban and rural households ($p = 0.043$), indicating potential influences of socioeconomic and environmental context on exposure.

The examination of phthalates further demonstrated the uneven distribution of chemical burdens in household air. Bis(2-ethylhexyl) phthalate (DEHP) and dibutyl phthalate (DBP) dominated both absolute and relative contributions across samples, with diethyl phthalate (DEP) also showing notable prevalence. The disproportionate contribution of these compounds to total concentrations underscores how a narrow set of pollutants often drives the majority of exposure risks. Notably, DEHP exposure in infants was associated with cancer risk estimates exceeding the EPA threshold, indicating that this is not solely an issue in high-emitting households but may represent a broader public health concern. As phthalates are continuously emitted from indoor materials and persist in dust, reducing exposure is challenging, although strategies such as improved ventilation and the use of phthalate-free products may mitigate exposure. Regulatory restrictions on certain phthalates (e.g. benzyl

butyl phthalate) has led to the development of alternative plasticisers, though the safety of these substitutes is still being investigated.The scatter and boxplot analyses further showed differences across households, but also highlighted the limitations of small sample sizes in fully identifying the effects of housing or behavioural factors.

Notably, the risks estimated from the two different pollutant classes (PAHs and phthalates) were high in mostly the same households. Therefore, these households are at increased risk from exposure to indoor $PM_{2.5}$ pollution. In contrast, these are not always the most polluted homes in terms of total $PM_{2.5}$ concentration. For example, House York ID=32 has a high cancer risk in both cases but one of the lowest mean $PM_{2.5}$ concentrations.

This suggests that PM2.5 mass alone is a poor proxy for assessing health risk, particularly for outcomes such as cancer that are strongly influenced by chemical composition rather than total particulate load. Across the dataset, variability in risk appears to be driven more by the presence and relative abundance of specific highly toxic compounds than by overall PM2.5 concentration. As such, care needs to be taken when extrapolating health effects based on concentration or mass alone.

Despite the relative complexity of the GC×GC-ToFMS method, the data collected here represent a valuable and unique archive of $PM_{2.5}$ composition in real-world residential environments. This dataset could serve as an important foundation for future investigations into other key pollutants, such as flame retardants and legacy persistent organic compounds, thereby enhancing understanding of the composition of indoor air.

In conclusion, these findings highlight the complex relationship between building characteristics, behavioural factors, and environmental sources in shaping indoor air quality and health risks. While phthalates and PAHs each displayed unique patterns, a consistent theme which emerged was the disproportionate influence of a few dominant pollutants in determining overall exposure and risk. The evidence also points towards environmental inequality, with deprivation of various types emerging as a potential risk factor. The chapter also highlights several methodological caveats, including assumptions about time–activity patterns, reliance on kitchen-based sampling, and limited subgroup sizes, which constrain the robustness of conclusions. Therefore, future work should seek to expand household sample sizes, collect samples across multiple rooms (considerations would have to be made for soundproofing these samplers or building quiet and reliable samplers; the sampler used in this study, with its soundproofing box, would have been far too loud for a bedroom/sleeping environ-

ment), and refine demographic stratifications to better capture the true variability of indoor exposures and their associated health risks.

# Chapter 5: The DOMESTIC Project: Cooking Experiments Performed in a Controlled Kitchen Environment

# 5 The DOMESTIC Project: Cooking Experiments Performed in a Controlled Kitchen Environment

While previous chapters have discussed the sources and health implications of indoor air pollution in general, cooking remains one of the most significant and variable contributors to indoor particulate matter concentrations[33]. Cooking processes release a complex mixture of particles and gases originating from combustion, heating of oils and fats, and the thermal degradation of food ingredients[153]. These emissions can vary widely in both concentration and chemical composition depending on the ingredients used, the cooking method, and the cooking fuel type[36].

The characterization of cooking-related particle emissions is particularly important because of their potential to contribute substantially to human exposure within home environments. Fine and ultrafine particles generated during cooking can remain airborne for extended periods and travel deep into the respiratory system, thereby causing potential risks to respiratory and cardiovascular health[154]. The understanding of variability in these emissions is essential for informing interventions to improve indoor air quality.

Controlled experiments provide an effective means of investigating these emissions under standardized and repeatable conditions. Conducting cooking experiments in a controlled environment allows for the manipulation of variables (e.g. overall ingredients, meat-containing vs. vegetarian dishes) while minimizing potential confounding factors. This type of setting enables meaningful comparisons between different cooking processes. These experiments were conducted in collaboration with researchers working on INGENIOUS Work Package 1, which carried out emissions experiments in laboratory settings.

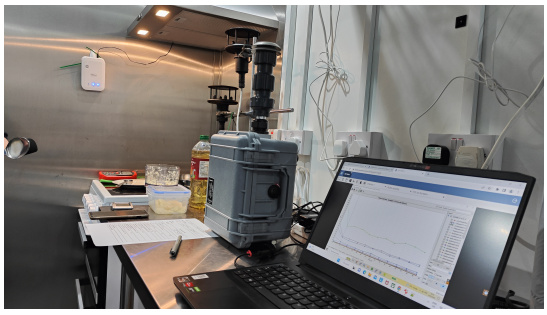
This chapter aims to identify differences in particle generation from different cooking recipes and ingredients (specifically, non-vegetarian and vegetarian versions of three recipes). The findings will contribute to a broader understanding of the role of cooking in indoor air quality and provide a foundation for future assessments of exposure and potential health risks.

## 5.1 Methodology

### 5.1.1 The DOMESTIC facility

The DOMESTIC (DOMestic Energy Systems and Technologies InCubator) facility was designed to replicate real-world residential cooking environments, but under controlled conditions. The facility consists of two converted shipping containers. The first container was converted into a fully functional kitchen space, containing a counter top, stove and oven unit, and a small preparation table. Specifically, it consists of a kitchen (4.3 m × 2.2 m × 2.3 m, volume = 22.0 $m^3$) and bathroom (1.5 m × 2.2 m × 2.3 m, volume = 7.1 $m^3$). A window was installed to provide adjustable ventilation. An extractor hood unit was also installed, and additional appliances such as a microwave and air fryer were purchased and placed within the kitchen. The kitchen area also contained several models of low-cost sensors; however, these were not utilized within the scope of INGENIOUS Work Package 2 and will therefore not be discussed further in this chapter. The kitchen area of the DOMESTIC facility is shown in Figure 81(a), which depicts the setup used to conduct cooking experiments. Figure 81(b) depicts the Minivol Tactical Air Sampler (Minivol TAS), the primary sampling instrument deployed in this chapter, positioned on the kitchen counter adjacent to the stove. This configuration created the greatest likelihood of capturing particulate matter emitted through the cooking activities due to the proximity to the emission source and replicates the positioning of the sampler on counter tops during the real home deployments. Figure 82 depicts the interior layout of the kitchen area.

**(a)** The kitchen area where cooking activities were carried out



**(b)** The Minivol TAS, deployed next to the cooking area

**Figure 81:** DOMESTIC Container 1

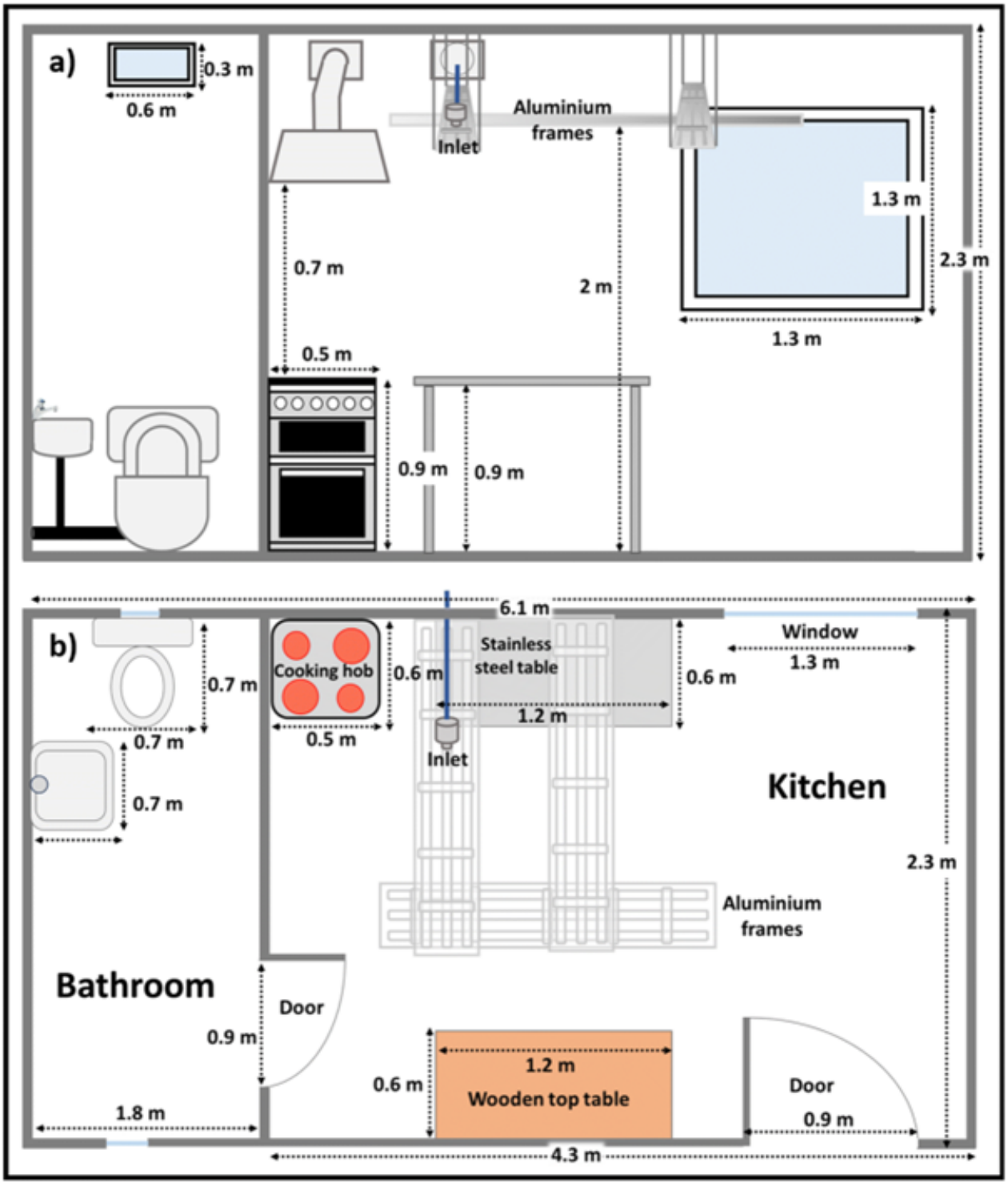



**Figure 82:** Diagram showing the interior layout of the kitchen area, courtesy of Kumar et. al. and appearing in a separate paper[38]

Sampling lines were installed within the kitchen container to continuously draw air samples during cooking experiments. These sampling lines were routed to the adjoining container,

which served dual purposes as both a data acquisition area and as a space for food preparation. This second container housed the monitoring instruments used to collect concentration and composition data in real time. The exterior of the facility, along with the interior of the container that functioned as the office and preparation area, is shown in Figure 83.

**(a)** The DOMESTIC facility, exterior

**(b)** DOMESTIC container 2, the office and food preparation area

**Figure 83:** The DOMESTIC facility and DOMESTIC Container 2

The design of the DOMESTIC facility enabled precise control over experimental conditions while creating a layout representative of a realistic residential kitchen space.

### 5.1.2 Recipe Selection

To capture a representative range of cooking-related particle emissions, two dishes were selected based on their demographic relevance in Bradford, as well as their overall popularity within the United Kingdom. Bradford has a significant South Asian population, as outlined in previous chapters, and thus a curry dish (prepared with either chicken or paneer) was included to in order to reflect cooking practices within the local community. Another popular meal cooked within the UK is chilli; as such, a beef or vegetarian chilli was also included.

The specific recipes were selected and modified via a combination of practical and cultural considerations. Feasibility was an important factor; all ingredients needed to be readily available year-round and easily sourced from UK supermarkets. Cultural authenticity was maintained wherever possible, particularly in the preparation of the curry dishes, to ensure that cooking methods and ingredients were representative of real-world domestic practices in households. For example, a popular Nigella Lawson curry recipe was modified by Dr. Ashish Kumar of the University of York in order to more accurately reflect the flavours that a household of South Asian descent might aspire to, though it should be noted that individual preferences will always vary widely. This approach allowed the experiments to incorporate both cultural diversity and typical UK cooking behaviours, providing a comprehensive assessment

of household cooking emissions to supplement the laboratory and real-home INGENIOUS studies.

### 5.1.3 Controlling and Standardising Experiments

Prior to the first experiment and between each subsequent cooking and background sampling period, the kitchen container was thoroughly ventilated to remove any residual aerosols. Ventilation was achieved by opening the window and door to the kitchen container, and operating the extractor hood for a period of approximately eight hours. During each sampling period, all doors and windows remained closed, and the extractor hood was kept switched off to maintain consistent conditions. Each recipe was cooked only once, but two samples were collected simultaneously for each cooking experiment. Continuous VOC measurements were taken throughout the experiment, which allowed capture of the periods corresponding to before cooking (room background), during cooking, and after cooking. For the PM component, background samples were taken for a period of 72 hours after the ventilation process had been carried out, and in the same conditions as for the sampling periods. All cooking tools were made of stainless steel only (pan, spatula)

To standardize the cooking process and provide reproducibility across all experiments, detailed recipe sheets were developed and followed for each dish. These standardized protocols are attached in the Appendix of this thesis and include step-by-step instructions with durations, ingredient quantities, and specific brands (where applicable). The differences between the meat-containing and vegetarian recipes were kept as minimal as possible, usually substituting just one ingredient, in order to better investigate the differences in emissions arising from including meat in cooking activities. Prior to each cooking experiment, all ingredients were pre-prepared, measured, and weighed outside the kitchen container to minimise activity in and out of the experimental area. Similarly, all required cooking utensils, cookware, and appliances were brought into the kitchen simultaneously to reduce the frequency of door openings and maintain a controlled indoor environment.

Cooking oil type was standardised across all recipes. For recipes where the use of a food processor was required, the food processor was brought into the kitchen area and operated during the sampling process. A stainless steel pan and spatula were used for all cooking experiments to avoid the potential release of particles associated with non-stick or coated cookware. A timer was used to ensure that all steps would take place during their allotted duration. To ensure consistency, a digital surface thermometer was used to verify that the pan reached the target temperature specified in each recipe before cooking commenced. Upon

completion of cooking ( specifically, once the burner was switched off), all utensils, cookware, and leftover ingredients were collected and removed from the kitchen container. The container remained sealed for the remainder of the 72-hour sampling period to minimise disturbance to the sampling area.

### 5.1.4 Sampling Methodology: Particulate Matter and Volatile Organic Compounds

AirGradient ONE (Model I-9PSL) sensors were used to obtain $PM_{1.0}$, $PM_{2.5}$, and $PM_{10.0}$ emissions data throughout the cooking process. More information on these sensors can be found in the experimental section of Chapter 3. In the DOMESTIC facility, this was fixed to the ceiling above and to the side of the hob.

Data for volatile organic compound emissions were obtained by use of a Selected Ion Flow Tube Mass Spectrometer (SIFT-MS, Syft Technologies, Christchurch, New Zealand). The SIFT-MS was operated with a flow tube temperature of 120 °C, pressure of 460 mTorr, a flowtube voltage of 25 V, a sample flowrate of 100 sccm and a nitrogen ($N_2$, research grade, BOC) carrier gas flow of 1 Torr/L/s which was maintained throughout the measurement period. The results for volatile organic compound emissions were obtained courtesy of Dr. Ashish Kumar, and details about both the sampling strategy and data processing method can be found in his paper entitled *Chemical fingerprints of cooking emissions and their impact on indoor air quality*[38]. The SIFT-MS was calibrated for methanol, acetonitrile, ethanol, acetaldehyde, acetone, isoprene, toluene and limonene in a calibration experiment performed prior to the measurement campaigns via dynamic dilution of a custom VOC gas mixture, and the total measurement uncertainty for these calibrated VOCs was <26%; for the uncalibrated compounds that are reported in this work, a total measurement uncertainty of 35% can be assumed [38].

### 5.1.5 Sampling Methodology: Minivol TAS for $PM_{2.5}$

Background samples were collected for a duration of approximately 72 hours prior to each cooking experiment, providing a baseline measure of indoor $PM_{2.5}$ levels. Entry to and exit from the kitchen container were minimized as far as practicable during both background and cooking periods to avoid disturbance of airflow and particle levels. The Minivol TAS was turned on immediately prior to each cooking event and continued to operate for approximately 72 hours, ensuring comparable sampling durations between background and cooking measurements. The operation and settings of the Minivol TAS (3 L/min flow rate) were

identical to that described for the sampling strategy of the INGENIOUS project; the filter extraction and analysis process was also maintained (ASE-EXTREVA, Pegasus 4D). More information can be found in Chapter 2.

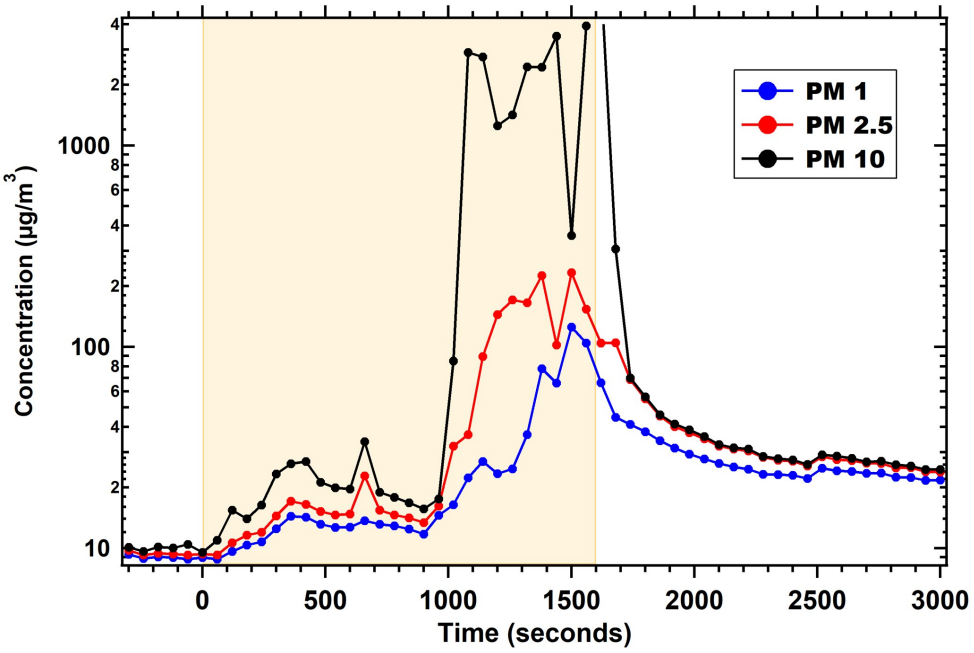
## 5.2 Results

### 5.2.1 Results from low-cost sensors and SIFT-MS: Particulate Matter and Volatile Organic Compounds

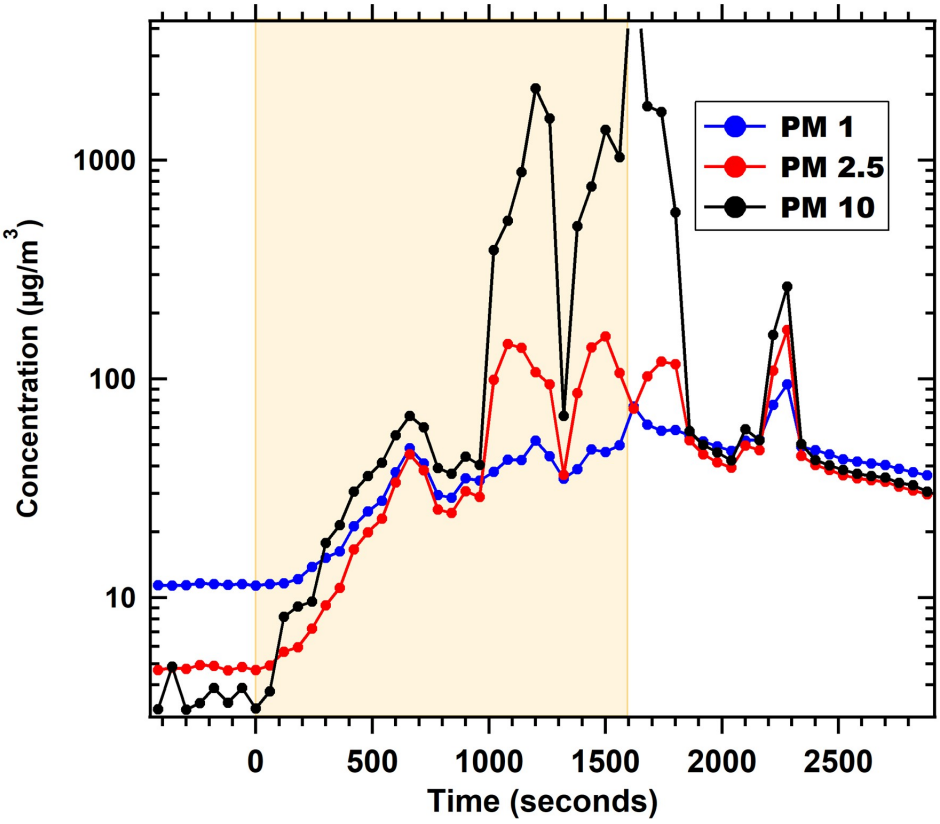
#### Particulate Matter

The figures below show time series plots of particulate matter concentrations recorded by the AirGradient sensor during cooking experiments for chilli and curry recipes. The area highlighted in yellow corresponds to the period when cooking activities were occurring.

Figure 84 shows the particulate matter time series for the beef and vegetarian chilli cooking experiments. As shown, the highest recorded peak exceeds the upper bound of the plotted axis, corresponding to a concentration greater than 8000 µg/m$^3$. Such a value is implausible under real-world indoor conditions and is likely an artefact. It is hypothesised that elevated humidity within the facility during these two experiments (both conducted one week apart) caused sensor interference or overloading. Relative humidity readings at the time approached 100%, which may have led to condensation effects within the optical chamber of the AirGradient sensor. Optical particle counters are known to overestimate mass concentrations under high humidity, because hygroscopic growth or condensation on particles increases light scattering[155].




**(a)** Time series plot showing PM emissions from the beef chilli cooking experiment

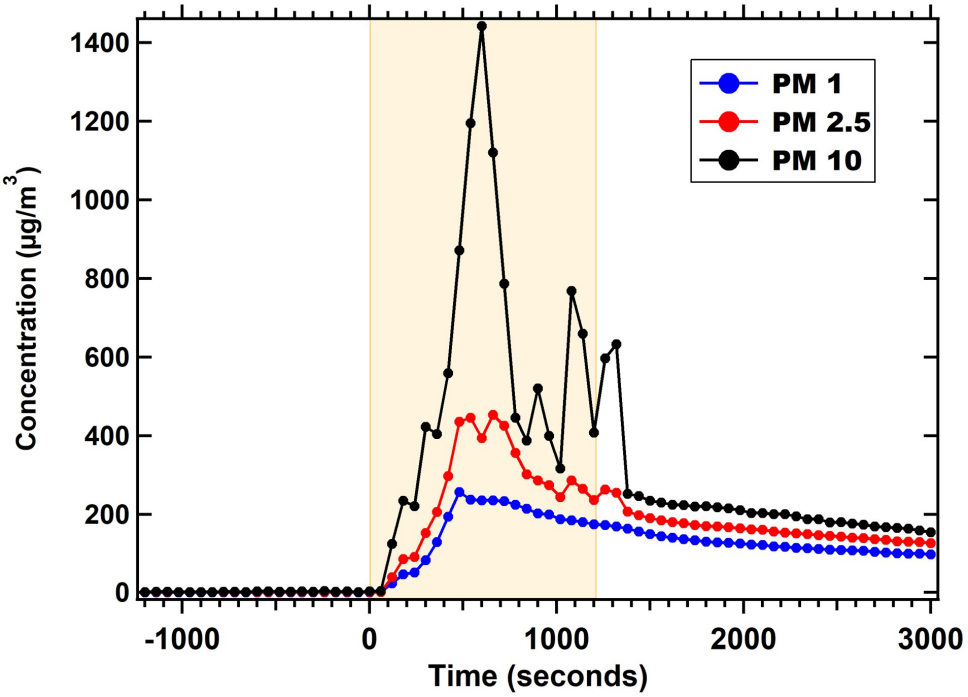



**(b)** Time series plot showing PM emissions from the vegetarian chilli cooking experiment

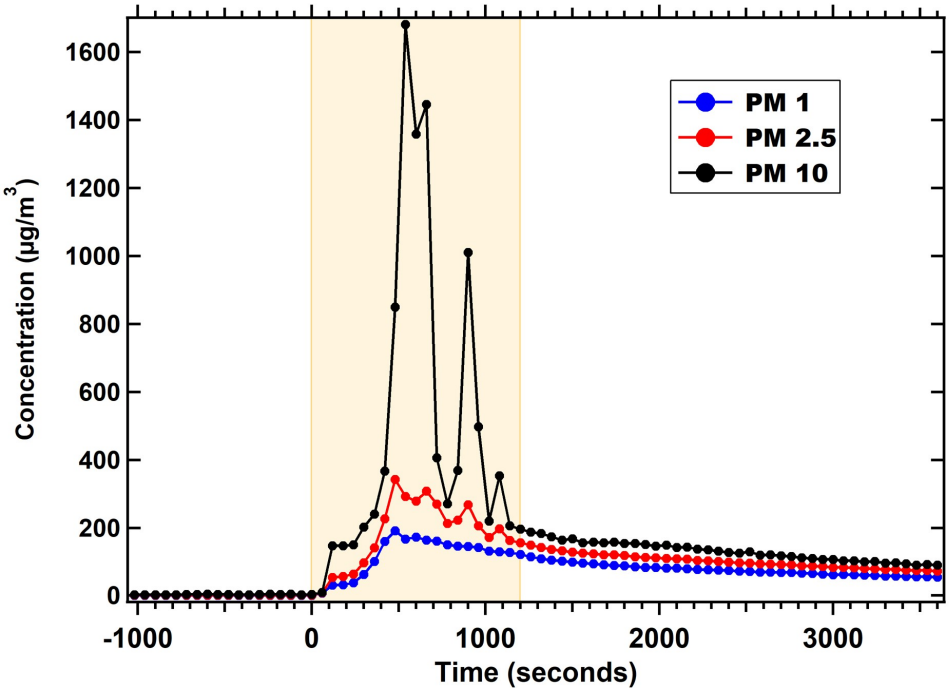
**Figure 84:** Comparison of $PM_{1.0}$, $PM_{2.5}$, and $PM_{10.0}$ emissions from the beef and vegetarian chilli cooking experiments, log scale. The yellow region indicates active cooking being carried out.

However, Figure 84 still allows for visualisation of the changes in particulate matter concentration that occur as cooking begins and as ingredients are gradually added. The post-cooking period illustrates how concentrations decline over time before stabilising at a level higher than the initial background, likely caused by the lack of ventilation in the closed environment.

The PM time series for curry cooking in Figure 85 appears to have been less affected by humidity than the chilli experiments, allowing the results to be interpreted with greater confidence. The chicken curry produced higher peak concentrations of $PM_{2.5}$ compared to the vegetarian curry, indicating that the inclusion of meat may influence the magnitude of particulate emissions—potentially due to differences in fat content, cooking temperature, or the types of aerosols generated during frying. Despite these peak differences, both recipes exhibit similar post-cooking behaviour, with particulate concentrations gradually declining and stabilising at levels above the pre-cooking background.




**(a)** Time series plot showing PM emissions from the chicken curry cooking experiment




**(b)** Time series plot showing PM emissions from the paneer curry cooking experiment

**Figure 85:** Comparison of $PM_{1.0}$, $PM_{2.5}$, and $PM_{10.0}$ emissions from the chicken and paneer curry cooking experiments

These figures are included primarily to illustrate the temporal variability in particulate

matter emissions during cooking activities. Unlike the compositional analyses presented later in the two-dimensional gas chromatography section, which provide compositional information but lack time resolution, these time series plots capture dynamic changes in concentration. They demonstrate how particulate emissions evolve throughout the cooking process, from the initiation when ingredients are added to the eventual decline as airborne particles disperse or deposit within the environment.

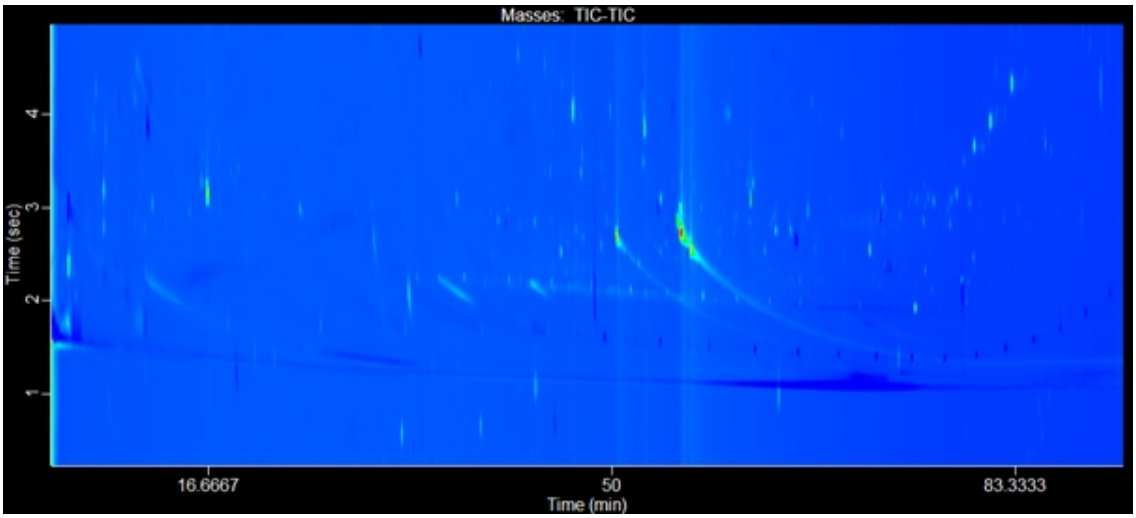
### 5.2.2 GC × GC-ToFMS: $PM_{2.5}$

Figure 86 presents two-dimensional (2D) contour plots of solvent extracts of samples from the PM filters collected during the cooking of a chicken curry and a paneer curry. Both plots have been background-subtracted to isolate emissions caused by the cooking processes. Visually, the contour plots appear extremely similar due to the plotting software scaling each plot according to its highest peak (although differences may still be observed for some); as such, direct visual comparison between the two is not particularly productive. As such, the analyses presented in this section focus on contour plots which are generated by subtracting one sample from another to better highlight differences between the emissions profiles of the different samples. In addition, this section will contain chromatograms comparing the spectra of the different samples to compare peak heights of specific compounds and to visualise differences between samples.

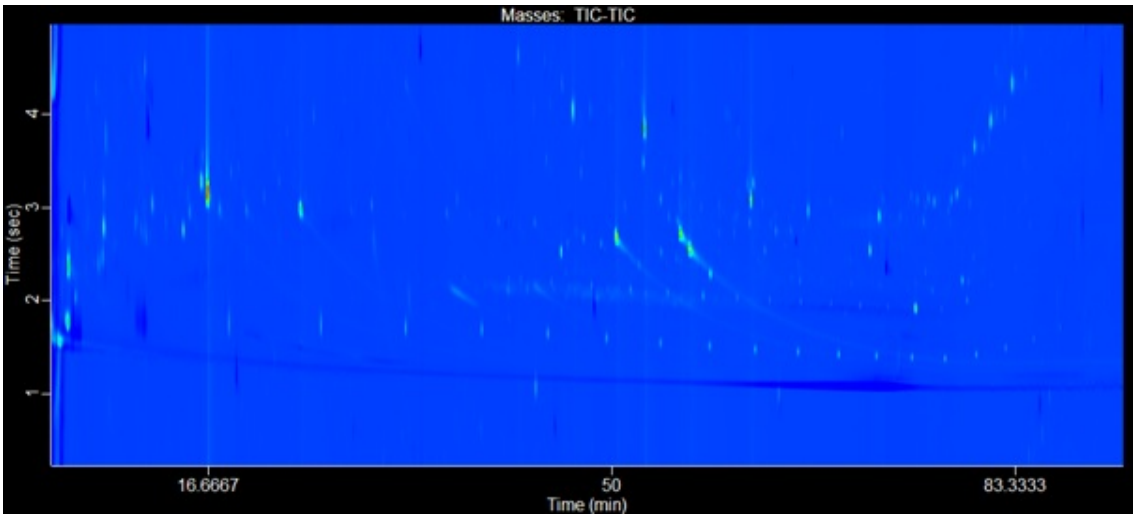
Figure 87 presents both the two-dimensional (2D) contour plot and the corresponding chromatogram for the sterol region, comparing emissions from the chicken curry and paneer curry cooking experiments. In this analysis, the paneer curry chromatogram has been subtracted from the chicken curry spectrum. This subtraction approach aimed to isolate the contribution of the meat-based ingredients to the recipe.

The results show that the chicken curry produced higher levels of several sterols: stellasterol, campesterol, and $\beta$-sitosterol. Elevated peak signals for hexadecanoic acid (palmitic acid) were also detected in the chicken curry sample. These compounds are commonly associated with the breakdown of animal fats during high-temperature cooking[156]. In contrast, the paneer curry, which is comprised of dairy and plant-based oils, exhibited comparatively lower sterol-related emissions.

Conversely, Figure 88 shows that the paneer curry exhibited elevated levels of several fatty aldehydes, including heptanal, octanal, and nonanal, as well as higher signal intensity of nonanoic acid, when compared to the chicken curry. This result was somewhat unexpected,

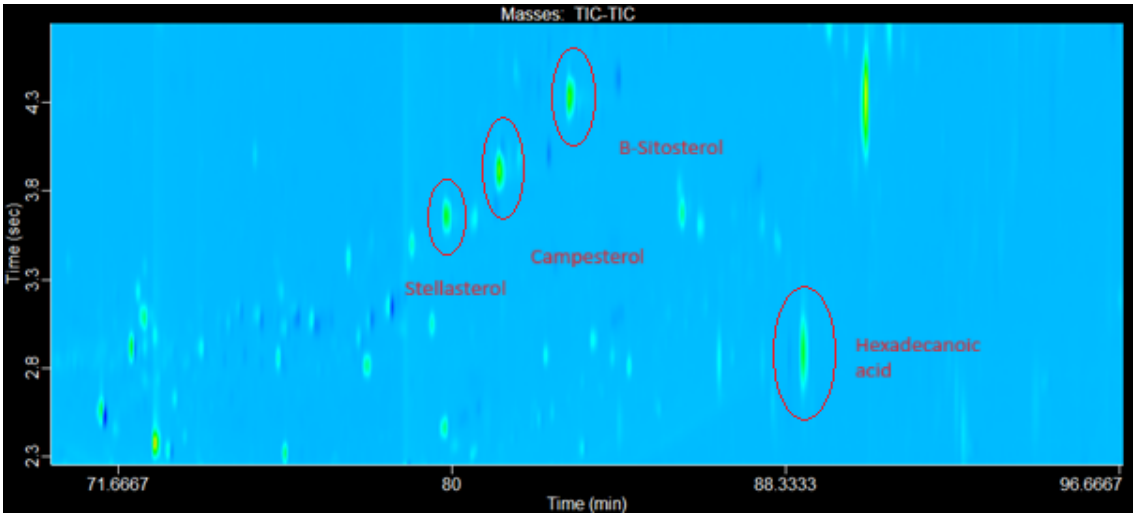



**(a)** Chicken curry cooking experiment, background subtracted

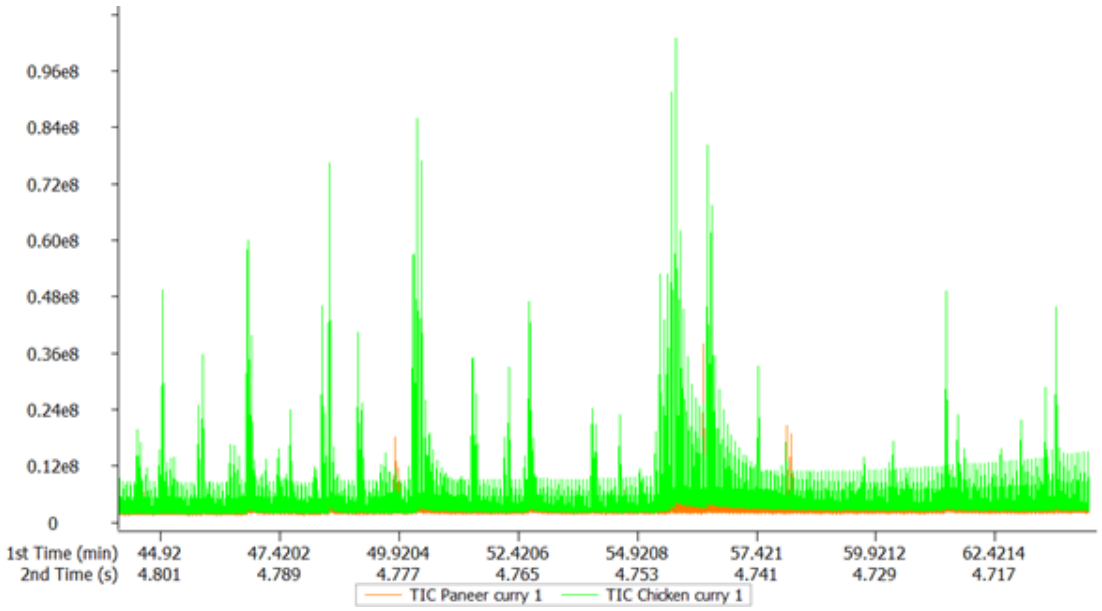



**(b)** Paneer curry cooking experiment, background subtracted

**Figure 86:** Comparison of GC×GC contour plots for (a) chicken curry and (b) paneer curry cooking experiments following background subtraction. Colours represent signal intensity; warmer colours indicate higher signal intensity.



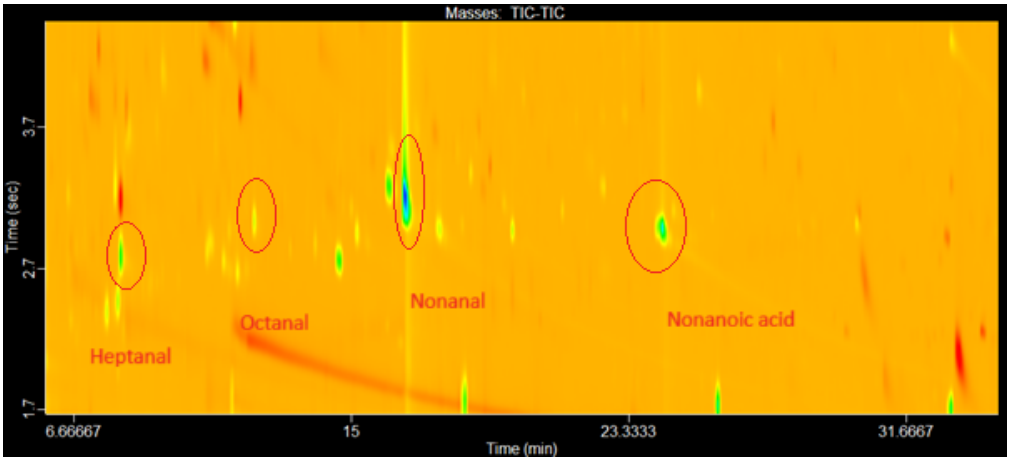

**(a)** Contour plot showing differences in sterol profiles between the chicken and paneer curry samples (chicken − paneer). Colours represent differences in signal intensity, with warmer colours indicating higher abundance in the chicken sample and cooler colours indicating higher abundance in the paneer sample.



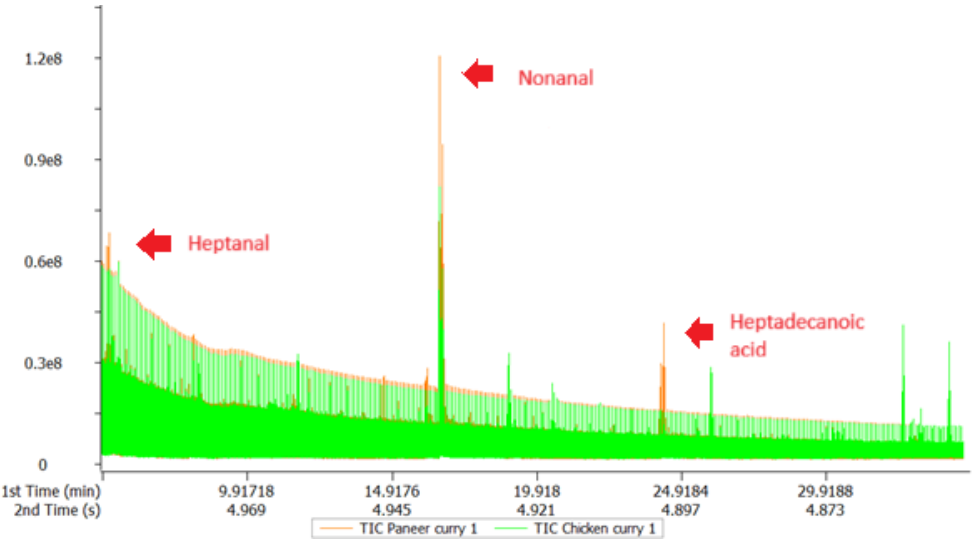

**(b)** Chromatogram illustrating variations in sterol peak intensities between the two cooking experiments.

**Figure 87:** Comparison of sterol emissions from chicken and paneer curry cooking experiments.

as it was initially hypothesised that the chicken curry, containing a greater proportion of animal fats, would produce higher overall emissions of lipid-related compounds.




**(a)** Contour plot showing the difference in fatty aldehyde profiles between the chicken and paneer curry samples (chicken − paneer)



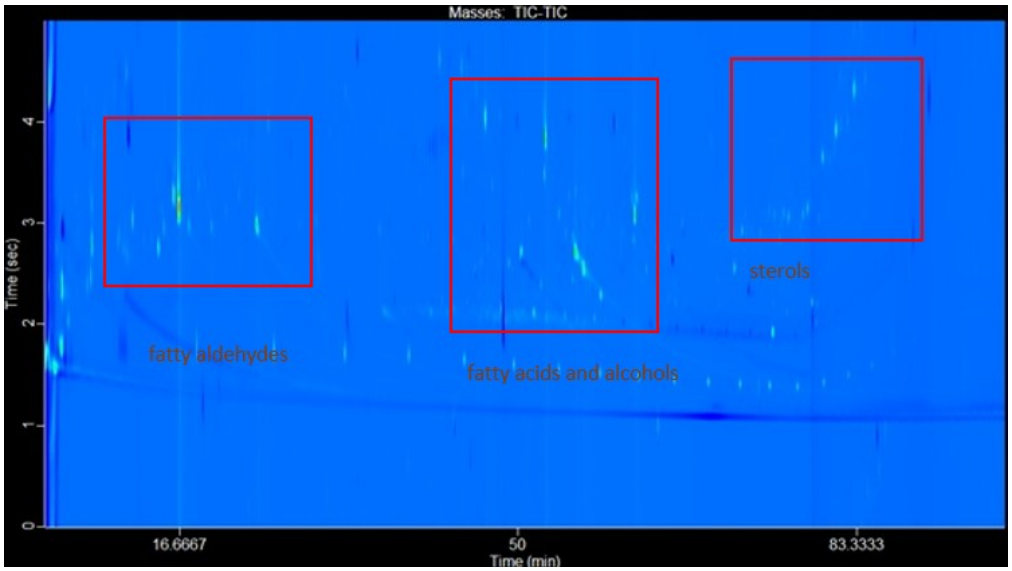

**(b)** Chromatogram illustrating variations in sterol peak intensities

**Figure 88:** Comparison of fatty aldehyde emissions from chicken and paneer curry cooking experiments

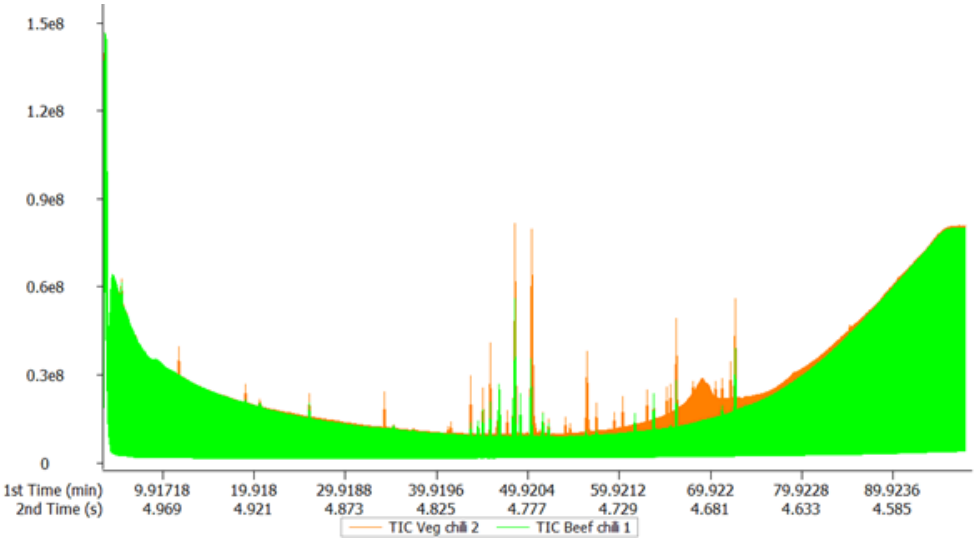
The higher signal intensities of fatty aldehydes observed in the paneer curry may possibly be explained by differences in the lipid composition and oxidation behavior of the fats used in the two dishes. Paneer and the oils used in its preparation contain more unsaturated fatty acids, which are more prone to oxidation during heating[157]. In a comparison of unsaturated fats per 100g, a quick online search yielded results of 1 g for chicken and 7-8 g for paneer. When exposed to high temperatures, these lipids break down to form volatile aldehydes such as those observed here[157].

In contrast, the fats in chicken are more saturated and less susceptible to oxidation. Natural antioxidants present in meat may also help limit aldehyde formation[158]. These results suggest that degree of unstauration plays a role in fatty aldehyde emissions.

Figure 89 shows the differences in the PM emitted from the beef and vegetarian chilli samples, with the vegetarian sample subtracted from the beef sample.




**(a)** Contour plot showing the difference between profiles of the beef and vegetarian chilli samples (beef − vegetarian)



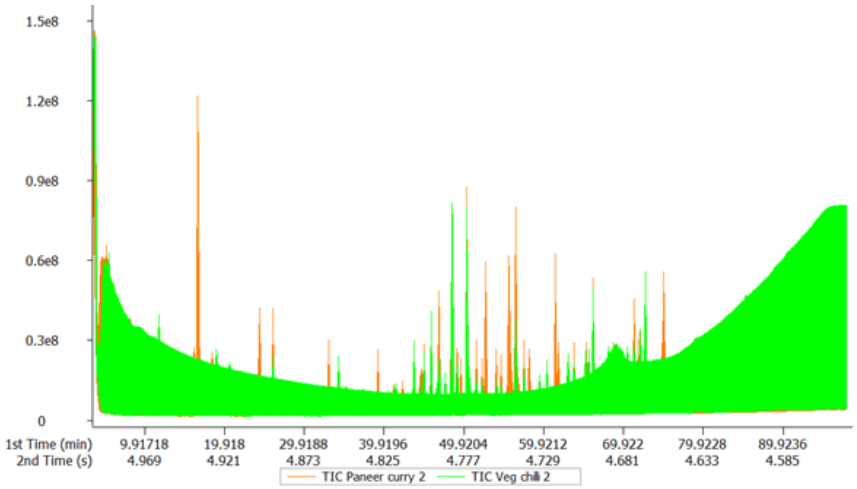

**(b)** Chromatogram illustrating variations in sterol peak intensities between the beef and vegetarian chilli samples (beef − vegetarian)

**Figure 89:** Comparison of fatty aldehyde emissions from chicken and paneer curry cooking experiments

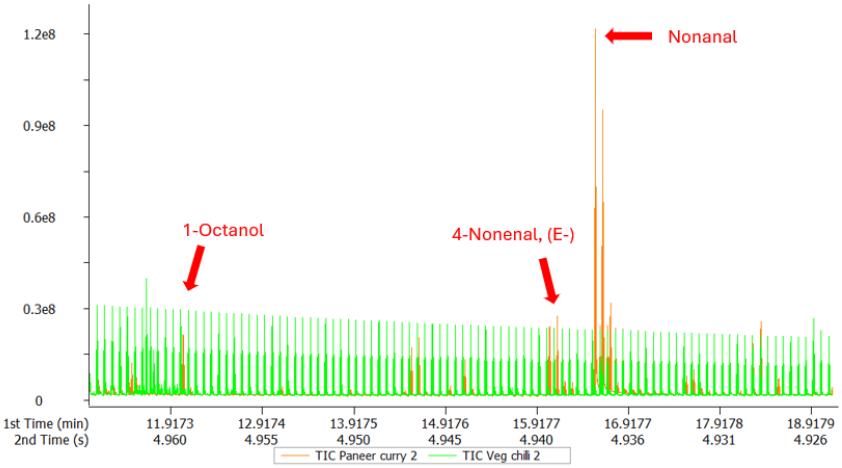
The results indicate that several compound classes (fatty aldehydes, fatty acids and alcohols, and sterols) were present at higher levels in the beef chilli. Beef typically contains cholesterol and animal-derived lipids, which upon heating can oxidize into sterols and similar compounds. Animal fats also include unsaturated fatty acids, which are more prone to thermal oxidation, generating volatile compounds like aldehydes and secondary alcohols[157].

It was observed that the differences between the meat and vegetarian versions of the same recipe were smaller than the differences observed between different recipe types. This was expected, as each recipe employed different combinations of spices and ingredients, which influenced the overall chemical profile of the experiments.

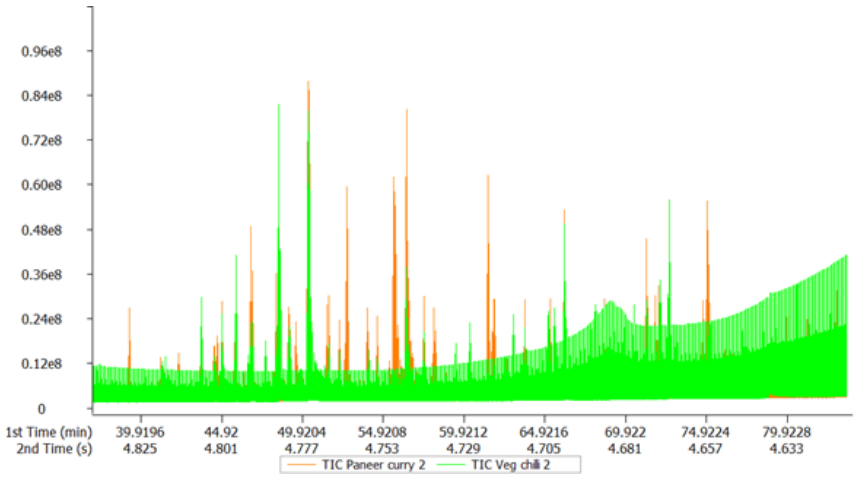
Figure 90 presents three chromatograms illustrating the compositional differences between the curry and chilli samples, with the vegetarian chilli sample being subtracted from the paneer curry. These chromatograms highlight that while substituting meat with vegetarian alternatives alters the emission profile, the overall chemical composition of emissions is more strongly influenced by the recipe type. Subfigure 90a presents the overall chromatogram comparing the paneer curry and vegetarian chilli samples, while Subfigures 90b and 90c focus on specific regions of interest within the chromatogram, showing the fatty aldehyde and fatty acid regions respectively.




**(a)** Chromatogram highlighting differences in aldehyde signal intensity between the paneer curry and vegetarian chilli



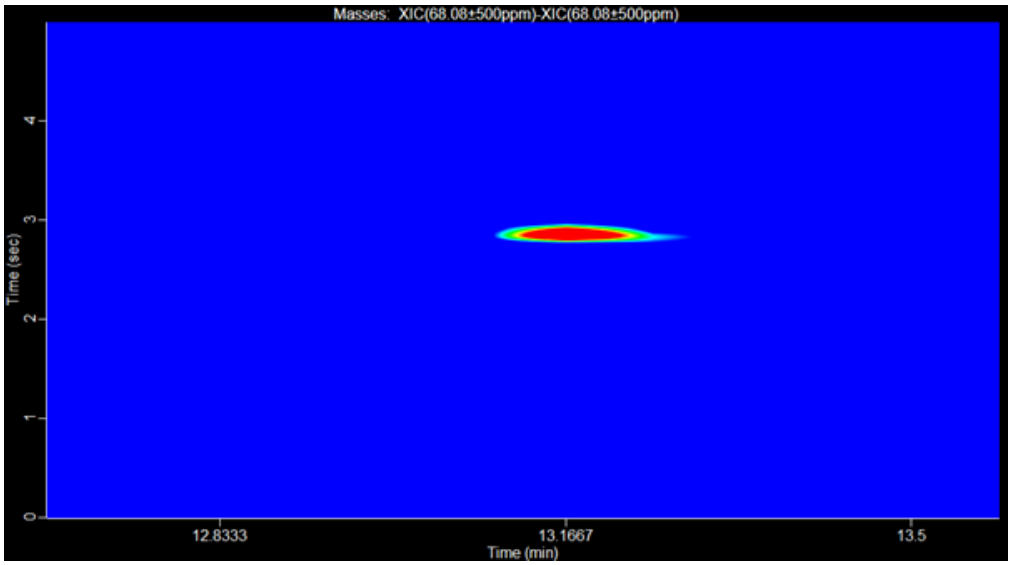

**(b)** Chromatogram comparing differences in the fatty aldehyde region between the paneer curry and vegetarian chilli recipes



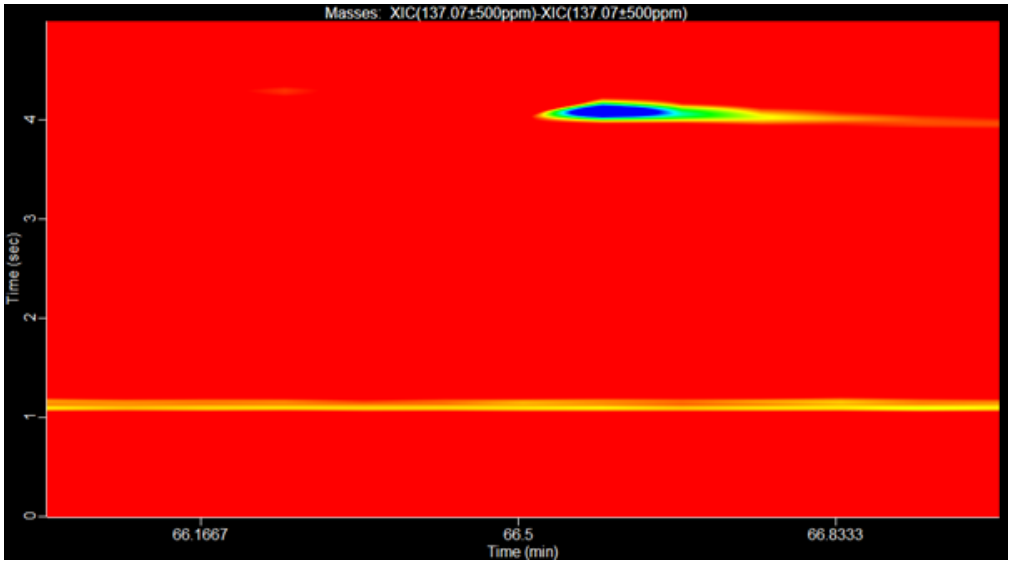

**(c)** Chromatogram comparing the fatty acid and alcohol regions between the paneer curry and vegetarian chilli recipes

**Figure 90:** Chromatograms comparing cooking emissions between paneer curry and vegetarian chilli

Across all three chromatograms, the paneer curry exhibited higher emissions for most shared compounds. The paneer sample also displayed a greater number of detectable peaks overall, suggesting a more chemically complex emission profile. In addition, the curry recipes used a greater quantity of spices than the chilli recipes. Dishes containing a greater number of spices and aromatic ingredients tend to produce higher emissions, as many spices release volatile organic compounds and semi-volatile oxidation products when heated[37]. Evidence of this can be seen in Figure 91, where two spice markers, limonene and capsaicin, were detected across recipes. Figure 91a shows that the curry recipe contained more limonene, which was expected due to the greater amount and variety of spices used in this recipe. In contrast, Figure 91b shows the opposite, with the chilli recipe containing higher levels of capsaicin.




**(a)** Contour plot highlighting differences in limonene between curry and chilli recipes




**(b)** Contour plot highlighting differences in capsaicin between curry and chilli recipes

**Figure 91:** Comparison of limonene and capsaicin between vegetarian curry and chilli recipes (curry − chilli)

These differences support the idea that cooking emissions are closely linked to the specific ingredient (and their respective quantities) composition of each recipe, rather than simply the variety of ingredients used.

## 5.3 Conclusions

This chapter presented a series of controlled cooking experiments conducted within the DOMESTIC facility to investigate emissions from household cooking activities. By simulating

realistic domestic cooking scenarios under controlled laboratory conditions, the study enabled direct comparison of emission profiles arising from different recipes and ingredient types.

The inclusion of time series plots offered a clear visualisation of the temporal variability in emissions that occurs as cooking progresses. These plots demonstrate how PM and VOC concentrations fluctuate dynamically in response to specific cooking actions.

The use of these real-time datasets complements the compositional data obtained from collected samples, enabling the interpretation of emission events in their full context. Together, they provide a clearer picture of how emissions develop, peak, and gradually decline following cooking, reflecting conditions that are both realistic and representative of everyday domestic activity.

The chromatographic results demonstrated that emissions varied substantially depending on both the recipe and the inclusion of meat or vegetarian ingredients. In general, the differences between distinct recipes (e.g. curry versus chilli) were greater than those observed between the meat and vegetarian variants of a single recipe. This finding highlights the strong influence of ingredient composition on overall emissions.

Comparisons between meat- and vegetarian-based dishes revealed meaningful compositional differences in the emitted compounds. Meat-containing recipes, such as the chicken curry and beef chilli, produced higher signal intensities of sterols, fatty acids, and other lipid-derived species, consistent with the oxidation of animal fats during high-temperature cooking. These compounds are generally less volatile and more likely to partition into the particulate phase, suggesting they may persist longer in indoor air and contribute to sustained $PM_{2.5}$ exposure following cooking events [153]. Conversely, a vegetarian dish containing a dairy-derived product showed higher emissions of unsaturated fatty aldehydes, which are more volatile and chemically reactive[159]. These species are associated with respiratory irritation and may contribute more strongly to short-term exposure and acute health effects[159]. As a caveat to the conclusions drawn from the data, it is important to note that there was only 1 experiment carried out per recipe type (though duplicate samples were collected from each cook). Thus, it is difficult to generalise from the results, and further research is required to draw more definitive conclusions.

Had time allowed, more replications of each recipe would have been carried out. In terms of analysis, target analytes would have been selected, reference standards purchased, and quantitation carried out. This would have provided a more detailed understanding of

emissions from cooking activities and enabled an assessment of potential health implications.

These findings reinforce the complex and varied nature of cooking-related emissions. The use of a controlled facility such as DOMESTIC enabled detailed chemical characterisation under standardized conditions, providing valuable insights into how everyday cooking behaviors may contribute to indoor air quality and exposure. The results from this chapter provide a foundation for further work quantifying exposure and mitigating potential health effects.

# Chapter 6: Conclusions

# 6 Conclusions

## 6.1 Reiteration of Project Aims

This project sought to improve understanding of indoor particulate matter by investigating its concentrations and composition under real-life conditions. The primary aims were to:

- Quantify concentrations of particulate matter in residential indoor environments under real-home conditions.
- Characterise the chemical composition of collected particles to aid source identification.
- Evaluate the toxicological significance of observed concentrations of compounds with known health effects.
- Carry out an exploratory non-targeted analysis of cooking emissions in a controlled environment. This was initially outside the scope of this study, but due to a global helium shortage causing delays in the original study, experiments were added to compare emissions across different popular recipes and between vegetarian and non-vegetarian ingredients.

## 6.2 INGENIOUS

The results from sampling across 34 homes in residential environments, particulate matter displays great temporal and spatial variability, influenced by factors including occupant behaviour, ventilation, and building characteristics.

In Chapter 2, the development of a sampling methodology tailored for indoor use was detailed. A key achievement was the development of a low-intrusion sampling method that maintained occupant safety and sampling reliability while being practical for use in occupied environments. This was achieved through the creation of a custom-built soundproofing enclosure, within which was placed an outdoor sampler. This approach enabled their deployment under real-world household conditions, ensuring occupant comfort and reducing likelihood of behavioural changes due to the presence of the samplers. One limitation of this approach was the need to reduce the sampler's flow rate to minimise noise. While this compromise improved acceptability for use in occupied homes, it also resulted in a higher particle size cut-off, slightly above 2.5 $\mu$m. In future studies, improved soundproofing or the use of quieter pumps could allow higher flow rates to be maintained without compromising participant comfort. In addition, it should be noted that this work required a team of people for the

deployment and collection of these samplers. Despite these constraints, the methodology provides a framework for future studies seeking to collect indoor particulate samples in real residential environments.

Chapter 3 presented the results of chemical characterisation and quantitation, identifying a wide range of compounds including but not limited to including phthalates, PAHs, and phenols. These compounds are known for their potential carcinogenic and endocrine-disrupting properties, and their detection in residential air emphasises the importance of monitoring indoor exposure. The methodology described in this chapter proved effective for targeted chemical analysis, resulting in a dataset that captures real-world indoor conditions and which in turn provides a valuable foundation for future investigations into indoor air composition and other classes of compounds found therein. Importantly, chemical composition was found to vary significantly between sampling sites, suggesting that location and occupant activities are key determinants of overall chemical profiles. This reinforces the need for monitoring in real-home environments in order to capture the variability introduced by the inherent differences in behaviour amongst humans. Only by accounting for these real-world factors can indoor air quality assessments provide an accurate reflection of human exposure and inform effective mitigation strategies.

The use of GC×GC–ToF-MS was critical in achieving this level of chemical characterisation. Compared to conventional one-dimensional GC-MS, the enhanced peak capacity and two-dimensional separation enabled improved resolution of co-eluting compounds, particularly within complex mixtures such as PAHs and their derivatives. This was evident in the ability to distinguish structurally similar compounds that would likely overlap in a single-dimension system, as demonstrated in the chromatographic comparisons presented in earlier chapters. As such, GC×GC provided clear advantages in resolving complex indoor particulate matter samples and improving confidence in compound identification. However, it must be acknowledged that there are practical constraints to this method, namely involving the complexity and constraints of the software.

In addition, other analytical approaches, such as liquid chromatography linked to mass spectrometry (LC-MS) and supercritical fluid chromatography–mass spectrometry (SFC-MS), would offer complementary chemical selectivity [160, 161]. The absence of these techniques represents a limitation of the current work and suggests that the assessment presented here may not fully account for all relevant compounds present in indoor particulate matter.

Chapter 4 extended this analysis by evaluating the toxicological implications of the measured concentrations of PAHs and phthalates, emphasised for their known toxicity. Using established toxic equivalency frameworks such as benzo[a]pyrene (BaP) equivalents for PAHs and DEHP-based metrics for phthalates, the project assessed the relative contributions of key compounds to total potential toxicity. For PAHs, benzo[a]pyrene, dibenz[a,h]anthracene, and indeno[1,2,3-cd]pyrene were the primary drivers of calculated exposure risk, reflecting their high toxic equivalency factors despite their relatively low mass contributions. In contrast, higher-mass PAHs such as chrysene and benzo[ghi]perylene contributed more substantially to total PAH mass but less to overall toxicity. For phthalates, DEHP and dibutyl phthalate were the dominant contributors both in mass and toxicity, consistent with their widespread presence and well-documented toxicological profiles.These results indicated that while overall particulate mass concentrations are an important indicator of exposure, chemical composition plays an equally critical role in determining risks to health. The analyses carried out provided a more complete understanding of these pollutants and their relevance to human health.

However, these results should be interpreted within the limitations of the study. Sampling was conducted over only three days per household, limited the ability to capture long-term variability. Future studies should aim to extend sampling durations, increase sample size, sample in multiple rooms of the households, and include other measurements such as outdoor sampling or source apportionment modeling to better distinguish between indoor and outdoor contributions. The last two points may be carried out by researchers in other INGENIOUS work packages using the data from this thesis.

Statistical analysis further highlighted that most demographic and behavioural factors considered in this study, including deprivation (IMD), smoking status, and cooking fuel type, did not show statistically significant associations with either total PAH concentrations or estimated cancer risk. The only statistically significant relationship observed was between housing tenure and total PAH concentration ($p = 0.009$), with rented properties exhibiting higher exposure levels.

While the analytical methodology developed for this work was effective for identifying and quantifying a wide range of compounds, its quantitative performance and limitations must be considered. Recovery experiments indicated variability across analytes, reflecting the challenges associated with replicating real-world sampling processes using spiked filters. As discussed, such approaches may not accurately represent gas–particle partitioning or the

physicochemical interactions occurring in indoor environments.

As a result, while the method is fit for comparative and exploratory analysis, caution should be applied when interpreting concentrations and their subsequent risk estimates. This limitation highlights the need for continued amendments and refinements to the methodology in future work.

Despite these limitations, this chapter's toxicological assessments served as a link between analytical results and real-world exposure concerns. Together, these chapters contribute to a more in-depth understanding of how indoor particulate matter composition and concentration affects human health. They highlight the importance of integrating compositional analysis with concentration measurements to more accurately evaluate exposure and its associated risks. In addition, they emphasise the significance of conducting sampling within real-home environments to reflect the complexity and variability of modern indoor living conditions.

## 6.3 DOMESTIC Cooking Experiments

The controlled cooking experiments conducted within the DOMESTIC facility provided an exploratory extension to the core research. These studies were designed to examine how household cooking activities contribute to particulate and VOC emissions under standardised yet realistic conditions. Although this work was undertaken in part due to delays in the primary study, it provided valuable additional insight into cooking behaviours and their effects on indoor particulate matter composition. Future studies could build on this methodology by testing a wider range of cooking styles and ingredients to better capture real-world variability. Applying targeted chemical and quantitative analysis could also help identify specific compounds of concern and their potential health implications. Together, these developments would enhance understanding of cooking-related exposures and strengthen the framework for future indoor air quality research.

## 6.4 Future Work

Future research should focus on refining indoor exposure assessment through the integration of real-time monitoring, chemical speciation, and personal exposure tracking. The methodological approach developed in this project could be expanded through deployment in a larger number of households, across different building types and climates, to improve the representativeness of data.

In addition to chromatographic techniques, spectroscopic approaches such as Fourier-transform infrared spectroscopy (FTIR) or Raman spectroscopy could be used to further characterise particulate matter composition, particularly for distinguishing between organic material, inorganic components, and emerging contaminants such as microplastics [162, 163]. The integration of these techniques would provide a more thorough understanding of particulate composition and improve source identification.

Further studies should also investigate the toxicological effects of specific compounds identified in this research, particularly PAHs and phthalates, under realistic exposure conditions. Controlled facilities such as DOMESTIC offer unique opportunities to study a wider range of everyday activities, such as cleaning, heating, and the use of consumer products, allowing a more comprehensive understanding of emission sources.

Collaboration between environmental scientists, toxicologists, and policymakers will be essential for translating these findings into effective strategies for improving indoor air quality. As awareness of indoor pollution grows, the development of evidence-based guidelines and standards will be critical to reducing public health risks associated with everyday exposures.

Critical to this work was the foundation of trust that Born in Bradford had already cultivated with the Bradford community. From participant recruitment to sampler deployment, dissemination of information, and the suggestion of potential interventions, the community played an active and engaged role throughout. This type of eager participation is rare and invaluable in public health research.

Ultimately, while advancing scientific understanding of indoor air pollution (e.g. sources, composition, and mechanisms of exposure, health risks) is vital, the true purpose of this work is to improve lives. Achieving this requires collaboration, transparency, and respect between researchers and the communities they study. This was the biggest lesson learned throughout my PhD, and served as a reminder that behind every York ID number, chromatogram peak, and LECR value are real people whose lives these data represent. It underscored the fact that our work should not be, at its core, just about understanding and assessing exposure, but about improving people's lives.

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# A Appendix A: Recipe Instructions

**Completed by:**

# Beef chilli

(loosely based off protocol for bolognese (Meal 3) from https://doi.org/10.1111/ina.12542)

**Test ID:** **Test Date:** **Entry Time:**
**Cook:** **Exit Time:**

## Ingredients

| Ingredient | Target Quantity | Actual Quantity | Prep | Detailed info, product code and link | Checklist |
|---|---|---|---|---|---|
| Vegetable Oil | 1 tbsp | | | Morrisons Vegetable Oil 1L | |
| Diced onions | 115 g | | Pre-prepared | Morrisons Diced White Onion 400g | |
| Garlic | 10 g | | Mini-chopper | Morrisons Garlic Pack x4 | |
| Hot chilli powder | ½ tsp | | | Schwartz Hot Chilli Powder 38g | |
| Ground Paprika | ½ tsp | | | Morrisons Ground Smoked Paprika 48g | |
| Ground Cumin | ½ tsp | | | Morrisons Ground Cumin 33g | |
| Beef mince | 200 g | | | Morrisons British Beef Lean Mince 5% Fat 500g<br><br>250g version | |
| Chopped tomatoes | 400 g (1 tin) | | | Morrisons Italian Chopped Tomatoes 400g | |
| Dried oregano | 1 tsp | | | Morrisons Oregano 12g | |
| Salt | ½ tsp | | | | |
| Tinned kidney beans | 1 tin, drained | | | Morrisons Red Kidney Beans (400g) | |

## Cooking Equipment

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| Pan | | Tbsp measure | | 1 plate | | Mini chopper | |
| Cooking utensil | | Pan lid | | Knife | | Scales | |
| Spatula | | Chopping board | | Bin bag | | | |

## Other Equipment

| | | | | | |
|---|---|---|---|---|---|
| Blue Roll | | Thermocouple | | Pen | |
| Canister | | | | | |

## Process

**Heat setting: 3 then 4** **Ring: Back right (1.7 kW)**

| | **Instructions** | **Time Elapsed** (mm:ss) | **Target Pan Temp** (°C) | **Time** |
|---|---|---|---|---|
| 0 | Peel **garlic** | | | |
| 0 | 2 pulses of **garlic** in mini chopper | | | |
| 0 | Open mini chopper and remove container ready to use | | | |
| 0 | Open the tub of onions | | | |
| 1 | Preheat pan on **setting 3** to target temperature | - | 120 | |
| 2 | Add **1 tbsp oil** and heat | 00:00 | | |
| 3 | Add **onion and garlic** to pan, continuously stir | 01:00 | | |
| 4 | Add **chilli powder, ground paprika and ground cumin** to pan and stir through | 02:00 | | |
| 5 | Add **beef mince** to the pan and cook, stirring occasionally until browned | 06:00 | | |
| 6 | Add the **chopped tomatoes, kidney beans, dried oregano and salt** and stir through. Increase ring to **setting 4.** | 11:00 | | |
| 7 | **End**: cover pan with lid to seal, remove pan from heat | 26:00 | | |
| 8 | **Canister Sample (optional).** | | | |
| 9 | **Wipe up any obvious spills** | | | |

**Completed by:**

# Meat free chilli

(loosely based off protocol for bolognese (Meal 3) from https://doi.org/10.1111/ina.12542 and https://www.bbcgoodfood.com/recipes/collection/healthy-chilli-con-carne)

**Test ID:** **Test Date:** **Entry Time:**

**Cook:** **Exit Time:**

## Ingredients

| Ingredient | Target Quantity | Actual Quantity | Prep | Detailed info, product code and link | Checklist |
|---|---|---|---|---|---|
| Vegetable Oil | 1 tbsp | | | Morrisons Vegetable Oil 1L | |
| Diced onions | 115 g | | Pre-prepared | Morrisons Diced White Onion 400g | |
| Garlic | 10 g | | Mini-chopper | Morrisons Garlic Pack x4 | |
| Hot chilli powder | ½ tsp | | | Schwartz Hot Chilli Powder 38g | |
| Smoked Paprika | ½ tsp | | | Morrisons Ground Smoked Paprika 48g | |
| Ground Cumin | ½ tsp | | | Morrisons Ground Cumin 33g | |
| Chopped tomatoes | 400 g (1 tin) | | | Morrisons Italian Chopped Tomatoes 400g | |
| Dried oregano | 1 tsp | | | Morrisons Oregano 12g | |
| Salt | ½ tsp | | | | |
| Tinned kidney beans | 1 tin, drained | | | Morrisons Red Kidney Beans (400g) | |
| Tinned mixed beans | 1 tin, drained | | | Morrisons Mixed Bean Salad (400g) | |

## Cooking Equipment

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| Pan | | Tbsp measure | | 1 plate | | Mini chopper | |
| Cooking utensil | | Pan lid | | Knife | | Scales | |
| Spatula | | Chopping board | | | | | |

## Other Equipment

| | | | | | |
|---|---|---|---|---|---|
| Blue Roll | | Thermocouple | | Pen | |
| Canister | | Bin bag | | | |

## Process

**Heat setting: 3** **Ring: Back right (1.7 kW)**

| | **Instructions** | **Time Elapsed** (mm:ss) | **Target Pan Temp** (°C) | **Time** |
|---|---|---|---|---|
| 0 | Peel **garlic** | | | |
| 0 | 2 Pulses of **garlic** | | | |
| 0 | Open mini chopper and remove container ready to use | | | |
| 0 | Open the tub of onions | | | |
| 1 | Preheat pan on **setting 3** to target temperature | - | 120 | |
| 2 | Add **1 tbsp oil** and heat | 00:00 | | |
| 3 | Add **onion and garlic** to pan, continuously stir | 01:00 | | |
| 4 | Add **chilli powder, ground paprika and ground cumin** to pan and stir through | 02:00 | | |
| 5 | Add the **chopped tomatoes, kidney beans, dried oregano and salt** and stir through. Increase ring to **setting 4.** | 11:00 | | |
| 6 | **End**: cover pan with lid to seal, remove pan from heat | 26:00 | | |
| 7 | **Canister Sample** | | | |
| 8 | **Wipe up any obvious spills** | | | |

**Completed by:**

# Chicken curry

(loosely based off the recipe shown in https://www.nigella.com/recipes/members/annauks-chicken-in-a-fried-onion-sauce) scaled down for a meal of 2.

**Test ID:** **Test Date:**

**Entry Time:** **Exit Time:**

## Ingredients

| Ingredient | Target Quantity | Actual Quantity | Prep | Detailed info, product code and link | Checklist |
|---|---|---|---|---|---|
| Vegetable Oil | 3 tbsp | | | Morrisons Vegetable Oil 1L | |
| Diced chicken breast | 250g | | Pre-prepared | Morrisons Diced Chicken Fillet 400g | |
| Diced onions | 150g | | Mini Chopper | Morrisons Diced White Onion 400g | |
| Fresh garlic | 10 g | | Mini Chopper | Morrisons Garlic Pack x4 | |
| Fresh ginger | 10 g | | Mini Chopper | Morrisons Ginger 125g | |
| Fresh green/red chillies | 10 g | | Mini Chopper | Morrisons Mixed Chillies 50g | |
| Ground coriander | 1/2tbsp | | Pre-prepared | Morrisons Ground Coriander 36g | |
| Ground Cumin | 1/2tbsp | | Pre-prepared | Morrisons Ground Cumin 33g | |
| Turmeric | 1/4tsp | | Pre-prepared | Morrisons Ground Turmeric 45g | |
| Cayenne pepper | 1/4tsp | | Pre-prepared | Morrisons Cayenne 48g | |
| Chopped tomatoes | 100g | | Pre-prepared | Morrisons Italian Chopped Tomatoes 400g | |
| Garam masala | 1/4tsp | | Pre-prepared | Schwartz Garam Masala Jar 30g | |
| Cooking Salt | 1tsp | | Pre-prepared | Morrisons Cooking Salt 1.5kg | |
| Water | 250mL | | | | |

## Cooking Equipment

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| Pan | | Tbsp measure | | Paper Plates | | Mini chopper | |
| Cooking utensil | | Pan lid | | Knife | | Scales | |
| Spatula | | Fork | | Chopping board | | Scissors | |

## Other Equipment

Canister if needed

## Process

**Heat setting: 4 and 2** **Ring: Larger one**

| | **Instructions** | **Time Elapsed** (mm:ss) | **Target Pan Temp** (°C) | **Time** |
|---|---|---|---|---|
| 0 | Peel **ginger** then **garlic** and de-stem **chilli** | | | |
| 0 | Chop **ginger, garlic and chilli** in the mini chopper and set aside | | | |
| 0 | Put the coarsely sliced **onions** in the mini chopper and chop it to smaller pieces and set aside. Skip this if using diced onions. | | | |
| 0 | Coarsely chop the coriander leaves (optional) | | | |
| 0 | Weigh the required amount of chopped tomatoes and set aside | | | |
| 1 | Preheat pan at **heat level 4** to target temperature | - | 180 | |
| 2 | Add **3 tbsp oil** and heat to >150ºC | 00:00 | | |
| 3 | Add onions to heated oil and stir continuously till golden brown | 01:00 | | |
| 4 | Add **garlic**, **ginger**, and **chillies** and stir continuously (raw smell should disappear) | 06:00 | | |
| 5 | Add chopped tomatoes and cook until tomatoes start turning mushy. | 07:00 | | |
| 6 | **Lower the heat level to 2** and add the spices (coriander, cumin, turmeric, and cayenne). Also add a sprinkle of water to prevent burning/overcooking the spices. Stir continuously for a minute. | 09:00 | | |
| 7 | Add **chicken pieces** to the pan, **increase the heating level to 4** and continuously stir. Stir in this stage till chicken starts turning pale | 10:00 | | |
| 10 | Add **water** to pan and mix well. Allow the water to reach boiling stage | 12:00 | | |

| | | |
|---|---|---|
| | | |
| 11 | Cover pan with lid to seal, and allow to cook for 5min. **Reduce the heat level to 2** | 15:00 |
| 12 | **Reduce the heating level to 2**. Add garam masala and salt (water if required) and mix well. | 17:00 |
| 13 | Simmer with lid closed and occasional stirring. Oil layer separating out of the gravy is an indication that the curry is cooked. Check if chicken pieces are cooked from inside (if not leave at low heat for 2min more). | 20:00 |
| 11 | **End**: Remove pan from heat | 21:00 |
| 12 | **Canister sample, if needed** | |
| 13 | **Wipe up any obvious spills, pack up** | |
| 14 | **Leave** | |

**Completed by:**

# Paneer curry

(Vegetarian version of chicken curry) scaled down for a meal of 2.

**Test ID:** **Test Date:**

**Entry Time:** **Exit Time:**

## Ingredients

| Ingredient | Target Quantity | Actual Quantity | Prep | Detailed info, product code and link | Checklist |
|---|---|---|---|---|---|
| Vegetable Oil | 3 tbsp | | | Morrisons Vegetable Oil 1L | |
| Diced Paneer | 200g | | Diced manually in 1” cubes | Pakeeza Paneer 226g | |
| Diced onions | 150g | | Mini Chopper | Morrisons Diced White Onion 400g | |
| Fresh garlic | 10 g | | Mini Chopper | Morrisons Garlic Pack x4 | |
| Fresh ginger | 10 g | | Mini Chopper | Morrisons Ginger 125g | |
| Fresh green/red chillies | 10 g | | Mini Chopper | Morrisons Mixed Chillies 50g | |
| Ground coriander | 1/2tbsp | | Pre-prepared | Morrisons Ground Coriander 36g | |
| Ground Cumin | 1/2tbsp | | Pre-prepared | Morrisons Ground Cumin 33g | |
| Turmeric | 1/4tsp | | Pre-prepared | Morrisons Ground Turmeric 45g | |
| Cayenne pepper | 1/4tsp | | Pre-prepared | Morrisons Cayenne 48g | |
| Chopped tomatoes | 100g | | Pre-prepared | Morrisons Italian Chopped Tomatoes 400g | |
| Garam masala | 1/4tsp | | Pre-prepared | Schwartz Garam Masala Jar 30g | |
| Cooking Salt | 1tsp | | Pre-prepared | Morrisons Cooking Salt 1.5kg | |
| Water | 250mL | | | | |

### Cooking Equipment

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| Pan | | Tbsp measure | | Paper Plates | | Mini chopper | |
| Cooking utensil | | Pan lid | | Knife | | Scales | |
| Spatula | | Fork | | Chopping board | | Scissors | |

## Other Equipment

Canister if needed

## Process

**Heat setting: 4 and 2** **Ring: Larger one**

| | **Instructions** | **Time Elapsed** (mm:ss) | **Target Pan Temp** (°C) | **Time** |
|---|---|---|---|---|
| 0 | Peel **ginger** then **garlic** and de-stem **chilli** | | | |
| 0 | Chop **ginger, garlic and chilli** in the mini chopper and set aside | | | |
| 0 | Put the coarsely sliced **onions** in the mini chopper and chop it to smaller pieces and set aside. Skip if using diced onions. | | | |
| 0 | Coarsely chop the coriander leaves | | | |
| 0 | Weigh the required amount of chopped tomatoes and set aside | | | |
| 1 | Preheat pan **at heat level 4** to target temperature | - | 180 | |
| 2 | Add **3 tbsp oil** and heat | 00:00 | | |
| 3 | Add onions to heated oil and stir continuously till golden brown | 01:00 | | |
| 4 | Add **garlic**, **ginger**, and **chillies** and stir continuously (raw smell should disappear) | 06:00 | | |
| 5 | Add chopped tomatoes and cook until tomatoes start turning mushy. | 07:00 | | |
| 6 | **Lower the heat level to 2** and add the spices (coriander, cumin, turmeric, and cayenne). Also add a sprinkle of water to prevent burning/overcooking the spices. Stir continuously for a minute. | 09:00 | | |
| 7 | Now add **paneer pieces** to the pan and stir, **increase the heating level to 4** and continuously stir for a minute. | 10:00 | | |
| 8 | Add **water** to pan and mix well. Allow the water to reach boiling stage. | 11:00 | | |

| | | |
|---|---|---|
| 9 | **Reduce the heating level to 2**, Add garam masala and salt, and mix well. | 14:00 |
| 10 | Simmer with lid closed and occasional stirring. Oil layer separating out of the gravy is an indication that the curry is cooked. | 17:00 |
| 11 | **End**: Remove pan from heat | 18:00 |
| 12 | **Canister sample, if needed** | |
| 13 | **Wipe up any obvious spills, pack up** | |
| 14 | **Leave** | |