The University Of Sheffield.

Understanding the role of inequality in creating and sustaining the Alcohol Harm Paradox.

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Thesis overview

In this thesis, I present my work which explores how a mechanism-based approach can be applied to test whether mechanisms described in health inequality theory can explain the alcohol harm paradox. This thesis brings together two novel contributions; the use of health inequality theory and the application of complex systems methods specifically agent-based modelling, to understand the causes of socioeconomic inequalities in alcohol harm.

Chapter one

The thesis begins by providing background on the alcohol harm paradox specifically discussing different definitions of the paradox and introducing evidence to support its existence. I also discuss the prominent explanations for the alcohol harm paradox and situate it as an issue of health inequality.

Chapter two

Chapter two outlines the research aims, questions and objectives of this PhD thesis and aligns them with each chapter.

Chapter three

In chapter three I provide background on inequalities in health more generally, by first defining how the term health inequality will be used in this thesis. I also introduce and discuss three distinct concepts: theories, pathways and frameworks, which have been used to explain inequalities in health, before introducing some further theoretical considerations.

Chapter four

Chapter four presents a systematic review of the explanations for the alcohol harm paradox and is included in publication format.

Title: "*Causal mechanisms proposed for the Alcohol Harm Paradox: A Systematic review.*"

Published in *Addiction.*

Authors: Jennifer Boyd, Olivia Sexton, Colin Angus, Petra Meier, Robin C. Purshouse, John Holmes.

Chapter five

Chapter five presents a theoretical review paper which explores how health inequality theories could be used to understand the alcohol harm paradox and is included in publication format.

Title: "*Beyond Behaviour: How Health Inequality Theory Can Enhance Our Understanding of the 'Alcohol-Harm Paradox'*."

Published in the *International Journal for Environmental Research and Public Health.*

Authors: Jennifer Boyd, Clare Bambra, Robin C. Purshouse, John Holmes.

Chapter six

Chapter six provides a detailed introduction to the modelling principles and methodologies applied in this thesis. Specifically, it describes what a realist mechanism-based approach is and introduces agent-based models as a particularly well-suited method to test mechanisms. This chapter also provides a detailed overview of the six step processes taken to use ABM to test a candidate mechanism.

Chapter seven

Chapter seven illustrates how the mechanism-based social systems software architecture was used to develop two independent conceptual models which could be implemented as ABMs. This chapter describes this process for two example theories; the Social Support Model and Fundamental Cause Theory. The reasons for selecting fundamental cause theory to be taken forward to the model implementation stage is also discussed at the end of this chapter.

Chapter eight

Chapter eight describes the process of constructing a static microsimulation model which represents the Scottish population in 2001, required to populate the agent-based model. This details the data cleaning procedure; the software and algorithm used to generate the population and presents the internal and external validation results of this model. Given that data on alcohol consumption was also required and not available from the survey used to generate the microsimulation model the process of integrating alcohol consumption data into the model from an alternative data source is described. Microsimulation population demographics, information on how the variables used to represent features of fundamental cause theory were constructed and a discussion of the strengths and limitation of the microsimulation model are also presented.

Chapter nine

Chapter nine presents an agent-based model which tests whether fundamental cause theory can explain the alcohol harm paradox in Scotland from 2001-2014. This chapter details the construction of the agent-based model and presents various diagnostic results which illustrate the functionality of the model and calibration results. This chapter is presented in publication format; however, it has not yet been submitted for publication.

Chapter ten

The final chapter of this thesis discusses the unique scientific contribution of this work by exploring alternative explanations for the alcohol harm paradox and applying agent-based model to test a candidate explanation, a method novel to this particular topic. This chapter

highlights the main findings, discusses the strengths and limitations, outlines some potential future directions for the research field and considers the implications for policy and practice.

Acknowledgement of collaborative work within the thesis

The thesis contains two manuscripts that have been accepted to peer-reviewed journals. The candidate (JB) confirms that the work submitted in this thesis is her own, except from work that has formed part of jointly authored journal publications. Each authors' contribution is indicated explicitly below. The candidate confirms that appropriate credit has been given within the thesis where reference has been made to others work.

Chapter four: Causal mechanisms proposed for the alcohol harm paradox – a systematic review. [Accepted for publication in *Addiction*].

Author: Jennifer Boyd

Co-authors: Olivia Sexton, Colin Angus, Petra Meier, Robin C. Purshouse, John Holmes.

Author contributions: Jennifer Boyd: Conceptualization; data curation; formal analysis; investigation; methodology; writing – original draft preparation. Olivia Sexton: Validation; writing – review and editing. Colin Angus: Investigation; writing – review and editing. Petra Meier: Writing – review and editing. Robin Purshouse: Conceptualization; formal analysis; methodology; supervision; writing – review and editing. John Holmes: Conceptualization; formal analysis; methodology; supervision; writing – review and editing.

Chapter five: Beyond behaviour: How health inequality theory can enhance our understanding of the 'alcohol-harm paradox'. [Accepted for publication in the *International Journal of Environmental Research and Public Health*].

Author: Jennifer Boyd

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Declarations from authors:

I, Olivia Sexton, declare that the above information regarding my contributions to the included manuscripts is a true record.

Signed:  Date: 29/09/2022

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I, John Holmes, declare that the above information regarding my contributions to the included manuscripts is a true record.

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Acronyms and Abbreviations

ABM: Agent-based Modelling

ABMs: Agent-based Models

AD: Alcohol Dependence

AHP: Alcohol Harm Paradox

AR: Alcohol Related

AUD: Alcohol Use Disorder

AXIS: Appraisal tool for Cross-Sectional Studies

BAC: Blood Alcohol Level

BHPS: British Household Panel Survey

BMI: Body Mass Index

CASCADE: Calibrated Agent Simulations for Combined Analysis of Drinking Etiologies

CASP: Critical Appraisal Skills Programme

CI: Confidence Interval

CSDH: Commission on Social Determinants of Health

DH: Determinants of Health

DSM: Diagnostic and Statistical Manual of Mental Disorders

ES: Employment Status

FCT: Fundamental Cause Theory

FMF: Flexible Modelling Framework

GHS: General Household Survey

ICD: International Classification of Diseases

IERPH: International Journal of Environmental Research and Public Health

LTC: Long Term Conditions

MBSSM: Mechanism-based Social Systems Modelling

MC: Military Conscripts

MP: Male Patients

MV: Motor Vehicles

NCDs: Non-Communicable Diseases

NGOs: Non-Governmental Organisations

NR: Not Reported

ODD: Overview, Design concepts and Details

OECD: Organization for Economic Cooperation and Development

OM: Occupational Mobility

ONS: Office for National Statistics

PECOS: Populations, Exposures, Comparisons, Outcomes and Study designs

PES: Partner Employment Status

PI: Parental Indicators

SAE: Standard Absolute Error

SC: Social Class

SDH: Social Determinants of Health

SEP: Socioeconomic Position

SES: Socioeconomic Status

SHeS: Scottish Health Survey

SHS: Scottish Household Survey

SIMD: Scottish Index of Multiple Deprivation

SRMSE: Standardised Root Mean Square Error

UK: United Kingdom

USA: United States of America

WHO: World Health Organization

Chapter 1 Introduction

Alcohol is a causal factor in over 200 disease and injury conditions (1). The World Health Organization (WHO) 2018 report on alcohol and health estimated 5.3% of all deaths globally and 5.1% of the global burden of disease and injury are attributable to alcohol (2). In the UK it is estimated that alcohol-related problems cost £3.5 billion to public healthcare, and £7.3 billion to the economy from lost productivity (3). Evidence from Scotland suggest that the burden of alcohol-related costs is not equally distributed, with 40% of health, social, crime and labour costs arising from the 20% most deprived areas (4).

1.1 What is the Alcohol Harm Paradox?

On average low socioeconomic positioned (SEP) groups drink the same or less than high SEP groups yet experience greater rates of alcohol related[1] harm (5,6). For example, in the UK a higher percentage of people in the least deprived group drink more than 4/3 (45%) or 8/6 (23%) units per day, almost double the number of people compared to those in the most deprived group (22% and 10% respectively) (7). Despite this, the risk of alcohol-specific[2] mortality is 5.5 times higher amongst the most deprived (8). The Alcohol Harm Paradox is the observation that low SEP individuals experience greater alcohol-related problems (9–11), despite drinking the same or less on average than their high SEP counterparts (12–15). This relationship is not only evident in the UK, and specifically Scotland (16) (which will be the setting for this PhD), but also exists in many other countries including Canada (6), Australia (17,18), the Netherlands (19) and Finland (20).

Evidence of the AHP has been found across a number of alcohol-related problems including *negative consequences* (e.g., alcohol-related disorders, injuries, the need to drink first thing in the morning, remorse and guilt, and alcohol-related violence) (5,19,21–24), *alcohol related morbidity* (the development of long term health conditions or hospitalization associated with

[1] Alcohol related: harm which is partially attributable to alcohol consumption.
[2] Alcohol specific: harm which is wholly attributable to alcohol consumption.

alcohol use) (11,13,16,25–34) and *alcohol related mortality* (8,11,13,16,25,28,32,35–37). SEP has also been measured in several ways; and the measure selected has an impact on the relationship observed between SEP, alcohol consumption and harm. For example, using a composite measure of SEP (e.g., comprised of education, employment, home and car ownership, income and social grade) leads to an observed social gradient in harm (5). Whereas other singular measures (e.g., occupational grade, income and education) show a u-shaped or inversed u-shape relationship – it's a phenomenon that disproportionately effects the most deprived (5). Generally, all measures point to some form of paradoxical relationship whereby those of the lowest SEP experience greater rates of alcohol harm despite drinking the same or less on average.

The AHP is an epidemiological phenomenon as each unit of alcohol consumed appears to be associated with a higher risk for low SEP individuals than high SEP individuals. This may be directly attributable to alcohol, caused by some other factor affecting the harmfulness of drinking or may be the result of confounding (or another bias) and explained by a pathway that does mediate the effects of alcohol consumption. It should be noted that this PhD does not take the causality of this relationship for granted. The main aim is draw on theory to seek candidate explanations for the AHP, in addition to seeking moderators or mediators if alcohol consumption is causal in this relationship.

1.2 Different Definitions of the Alcohol Harm Paradox

The AHP is a commonly discussed phenomenon within population health, alcohol research and epidemiology, however there is a discrepancy in how the term is used. Sometimes the term is used to mean general inequalities in alcohol-related outcomes without referring to alcohol consumption. In this sense it is taken to mean that lower SEP groups suffer higher rates of alcohol-related harm compared to high SEP groups. However, for this relationship to be paradoxical the inclusion of alcohol consumption is crucial. For clarity, this PhD will define the AHP as the finding that *lower SEP groups have higher rates of alcohol related and specific harms despite drinking the same or less on average as high SEP groups*. It should also be

noted that this pertains to their alcohol consumption across an extended period for example average weekly, monthly, or yearly units of alcohol consumed, as opposed to average consumption per drinking occasion. This is an important distinction because SEP groups have different drinking patterns. Evidence suggests that per occasion lower SEP groups drink more than higher SEP groups, while high SEP drink more frequently but less per occasion (38). The volume of alcohol in absolute terms is the same or less for those of a low SEP, but key differences as to how they consume this quantity of alcohol could be causal to the AHP. The remainder of this thesis will only refer to the AHP when discussing the relationship between SEP, alcohol consumption and alcohol-related harm.

1.3 Overview of the Current Literature

Several reviews and meta-analyses have been published pertaining to differences in alcohol-related harm between SEP groups. Most of these have focused on establishing socioeconomic differences in alcohol-related or attributable harm based on existing evidence or available survey data (6,12,13,39,40), but do not address whether this association is paradoxical. Only a subset of reviews and meta-analyses investigated the AHP by exploring the role of alcohol consumption or drinking patterns in explaining these differences (12,13,39). One also investigated whether the AHP holds across gender and investigated differences between high income and low-income countries (12).

The review level evidence on the AHP is mixed. Of those which investigated consumption, only one meta-analysis concluded that even when alcohol consumption patterns were the same between SEP groups, those of a low SEP had an elevated risk of reporting negative alcohol-related consequences (12). However, in another the level of consumption did attenuate the relationship between SEP and some disease specific outcomes including stroke, and when combined with smoking, could explain low SEP individuals' greater risk of developing head and neck cancer (13). Although ultimately this review was limited as it was unable to explore the relationship between alcohol consumption, SEP and alcohol-attributable harm. Another review which investigated consumption explored the differences between

evidence produced from cross-sectional and longitudinal studies and found mixed evidence for the existence of the paradox (39).

A more recent meta-analysis aimed to address issues associated with previous meta-analyses by accounting for alcohol consumption (41). The meta-analysis not only aimed to update the evidence to account for the role of alcohol consumption in socioeconomic inequalities in mortality but also investigated whether consumption and/or drinking patterns could explain differences in alcohol-related outcomes between SEP groups. This meta-analysis found that the quantity of alcohol consumed could only explain between -5% and 15% of the variance in the relative risk of both all-cause and alcohol-attributable morality between socioeconomic groups (41). However, drinking patterns had greater explanatory value; with frequency of heavy episodic drinking explaining 15% to 30% of the difference in mortality outcomes. This study does present evidence to support that per unit of alcohol low SEP groups experience an elevated risk of alcohol-related harm, while indicating that drinking patterns play a role.

Exploration of factors other than alcohol consumption in existing reviews was mainly hypothetical and presented in the discussion section. These factors included methodological (e.g., measurement error), environmental, social support, stress/chronic stress, clustering of health behaviours, differential access to healthcare, neighbourhood deprivation and materialist explanations (6,12,13,41).

The focus of reviews regarding the AHP thus far have been to establish its existence, whether it holds across different contexts (e.g., gender and countries), and if it can be explained by drinking patterns. Whilst each review mentions or theorises other mechanisms which may explain the existence of the paradox, consideration of these are often brief and not included in the study design.

1.4 What are the explanations for the Alcohol Harm Paradox?

An overview of the alcohol-harm paradox written in 2014 (42) proposed five key explanations for the existence of the paradox: drinking patterns, underreporting of consumption,

participation in multiple ‘unhealthy’ behaviours, access to healthcare and the effects of poverty on health inequalities.

Much of the existing research has focused on investigating the role of proximal individual-level factors only touching on the first three examples highlighted above. This research has examined the role drinking patterns, participation in multiple unhealthy behaviours, drinking history and underreporting (failing to accurately report alcohol consumption either intentionally or unintentionally) (38,43). Evidence from cross-sectional studies indicates that low SEP groups consume greater quantities of alcohol per occasion and are more likely to exceed recommended drinking limits, in contrast to high SEP groups whom drink frequently but drink less on these occasions (38,43–45). Engaging in heavier drinking patterns may lead to worse health outcomes - this has been a prominent explanation for the disproportionate rates of alcohol-related morbidity and mortality experienced by low SEP groups (5,38,43–45). There is also evidence that lower SEP groups are more likely to engage in multiple unhealthy behaviours including smoking, poor diet, and lack of exercise (38), which also might compound harm. However, these studies do not investigate alcohol-related harm and therefore it is unclear whether these factors can explain the AHP.

Contrary to evidence from cross-sectional studies, a record-linkage study using Scottish data indicated that per unit of alcohol consumed low SEP was associated with a greater probability of alcohol-related harm relative to high SEP (16). This relationship was only partially attenuated when adjusting for weekly consumption, binge drinking, BMI, and smoking (16). Therefore, these behavioural risk factors could not fully explain the AHP – following adjustment the risk of harm for lower SEP remained three-fold greater when compared to those of a high SEP (16).

Another group of prominent explanations are those related to methodological artefacts, including measurement error and underreporting. Measurement error regarding alcohol consumption needs to be noted as a potential limitation of research to date, given the mismatch between alcohol sales data and self-reported consumption; self-reported

consumption is often much lower compared to sales data (46). In terms of underreporting, research in fact suggests that high SEP groups are more likely to underreport by discounting special occasions (e.g., weddings and holidays) when self-reporting their alcohol consumption (38). Additionally, recent evidence using alcohol biomarkers as a measure of alcohol consumption further discounts concerns that the AHP is only found due to methodological artefacts. Alcohol biomarkers are an objective measure of alcohol consumption, they can indicate heavy or chronic alcohol use and abnormal liver function, and are not subject to the bias (e.g., recall, social desirability) associated with self-report measures (47). A paper which compared self-report measures of alcohol consumption to biomarkers found that the use of alcohol biomarkers in addition to self-report measures only slightly attenuated the AHP (47). Therefore, it is unlikely that measurement error or underreporting play a key role in explaining the AHP.

The socioeconomic circumstances of individuals are also hypothesised to cause the AHP (42); however empirical research has yet to explore this. Two factors which stem from socioeconomic inequality: access to healthcare and material explanations are purported to underlie the AHP (42). Although it is well known those living in deprivation face greater barriers when accessing healthcare services (e.g., costs, distance, and availability) (42) – little has been done to understand the role of these barriers in creating and sustaining the alcohol harm paradox. Material (social, economic, and environmental) circumstances are also thought to provide a protective benefit and therefore those with fewer resources are less protected from the experience of a problem or the impact of a stressful life event (48). These mechanisms and others associated with inequality may be intrinsic to the AHP and therefore should be addressed by empirical research.

It is clear that causes of the AHP are not fully understood. Alcohol-related harm and health inequalities in general are closely related, with recent evidence to suggest that alcohol-specific deaths greatly contribute to inequalities in life expectancy and variation in age of death between socioeconomic groups (49). It is therefore crucial to understand which factors drive

the alcohol harm paradox to inform public debate and the design of policies that can reduce alcohol-related harm and inequality in society.

1.5 The Alcohol Harm Paradox – an issue of health inequality

Health inequalities associated with alcohol have detrimental impacts for society. Due to the presence of the AHP, those living in deprivation shoulder the burden of alcohol harms more than any other group. These harms range from health (e.g., cancer, heart and liver disease) to social concerns including dangerous driving, crime and domestic abuse (42). Given that the most deprived are disproportionately affected relative to their alcohol consumption, aiming to reduce inequalities associated with alcohol could be central in tackling health inequalities more generally (49). It is critical to note that increased general welfare spending has been found to decrease alcohol related mortality (42), therefore tackling upstream determinants of health inequalities is likely to support efforts to reduce alcohol-related harms (50,51). Many researchers investigating the AHP discuss the potential impact of material resources or access to healthcare (16,42), however these discussions are often brief and hypothetical.

The AHP is clearly an issue of health inequality, yet research has not yet attempted to apply what is known about the causes of health inequalities to this phenomenon. Much of the research to date regarding the AHP has focused on establishing its existence (6,12,13,39,40) and investigated whether it holds across contexts (e.g., countries) or population groups (e.g., gender) (12,40). However, in terms of explanation, prominent research has thus far focused on testing the individual behavioural factors (e.g., drinking patterns and engaging in multiple unhealth behaviours) proposed to drive the existence of this phenomenon (42). Although explanations pertaining to individual behaviour play a role, evidence suggests these behavioural factors cannot fully explain the excess harm experienced by low SEP groups (16). Therefore, additional causal mechanisms are likely to be implicated.

Due to the current political and economic environment in the UK and globally; a combination of the effects of austerity, the impact of the global pandemic COVID-19 and the exponential

rise in the cost of living, it is expected that inequality will continue to rise. Therefore, it is critical to gain a deeper understanding of the causes of AHP to reveal key pathways where policy action could relieve the burden on low SEP groups. Drawing on theories of health inequality and associated factors when attempting to understand and tackle this phenomenon could shed light on the causal mechanisms.

Very little theory has been applied to understand the AHP and as a result explanations lack structure or detailed mechanisms. Applying theory from the well-developed area of health inequality has the potential to shed light on the mechanisms that create and sustain the AHP. As the causes of the paradox remain unknown it has remained difficult to design effective interventions or policy to reduce inequalities in alcohol-related outcomes. Identifying the key mechanisms that drive worse alcohol harm outcomes for low SEP groups is critical to mitigate the effects of the paradox. Generally, this PhD will take a mechanism-based approach to understand the AHP. A mechanism-based approach involves acknowledging that underlying the association between two variables is a mechanism (52). Mechanisms are comprised of "entities" and the "activities" those entities engage in collectively or independently which result in the observed outcome, or phenomenon (53). Computer simulation models particularly agent-based models (ABMs) are a useful tool for exploring the role of mechanisms in generating phenomenon such as the AHP, as they essentially encode mechanisms (54). This PhD will also draw on a wider health inequalities perspective, by looking to existing theories of the causes of health inequalities in general to identify mechanisms which could explain inequalities in alcohol harm.

1.6 Structure and contribution of the thesis

The remainder of this thesis will first outline the research aims, questions and objectives posed by this PhD project (Chapter 2). Then it will provide a background to health inequality theory, describing the most prominent theories in detail and outlining the frameworks used to understand the causes of health inequalities (Chapter 3). Then it will present a systematic review published in *Addiction* which synthesizes potential explanations for the AHP from the

existing literature (Chapter 4). This paper contributes to knowledge of the causes of the paradox given previous reviews only briefly mention or theorise mechanisms which may explain the existence of the paradox and have mainly focused on individual behavioural explanations. It will then present a review published in the *International Journal of Environmental Research and Public Health* (IJERPH) which brings together what we know about the AHP from the existing literature and attempts to enhance this understanding using health inequality theory (Chapter 5). This is the first paper to explicitly draw on theories of health inequality in the context of the AHP and provide suggestions for future empirical work. It will then outline and describe the modelling principles and methodology implemented to build an ABM which will test one candidate mechanism as an explanation for the AHP (Chapter 6) and describe how the selected theory or theories are translated into a conceptual model (Chapter 7). Then it will describe the process of developing a static microsimulation model of the population of Scotland in 2001 which will be used to initialize individuals in the ABM at baseline and provide them with the necessary attributes for the simulation (Chapter 8). It will then present an ABM developed to test whether the selected candidate theory can reproduce the trends associated with the AHP (Chapter 9). This is a Scottish case study over the period of 2001-2019, which will highlight the merits and limitations of using ABM to understand the causes of complex public health phenomenon such as the AHP. Finally, it will provide a general discussion of the thesis as a whole (Chapter 10).

Chapter 2 Research Aims, Questions and Objectives

This chapter will outline the main research aims of this PhD, followed by the research questions and objectives associated with each chapter of the PhD.

2.1 Research Aims

To advance understanding of the causal mechanisms behind the AHP by undertaking literature-based and secondary research to develop and validate a theory driven computer simulation model. This model will attempt to reproduce the trends associated with the AHP using mechanisms derived from health inequality theory.

2.2 Research Questions

1. What are the mechanisms and combinations of mechanisms that exist in the current literature on the AHP?
2. Are the mechanisms embedded in theories of health inequality present in the existing literature investigating the AHP and how could these theories be used to explicitly frame research on the AHP?
3. Can a mechanism-based explanation derived from health inequality theory be represented as an agent-based model?
4. Is the simulation model a candidate explanation for the existence of the AHP in Scotland?

2.3 Research Objectives

Systematic Review (Chapter 4)

1. To establish a comprehensive set of explanations proposed or tested by researched as explanations for the AHP.
2. To categorise these explanations into wider domains and describe the relationships that exist between explanations.

3 To provide an overview of the evidence base for the explanations which have been empirically tested.

Theoretical Paper (Chapter 5)

1. To explore how theories of health inequality could be used to understand the AHP and how these fit with explanations presented in the existing literature on the AHP.
2. To select a theory or theories to inform the creation of a conceptual model which has the potential to explain the AHP.

Model Development (Chapters 7-9)

1. To redescribe the selected theory or theories as a mechanism-based explanatory model, in terms of macro-micro middle-range theory suitable for computational modelling.
2. To build a microsimulation which is representative of the Scottish population in 2001.
3. To build an ABM which represents one mechanism-based explanation and initialize it with a representative sample from the Scottish microsimulation.
4. To use a calibration process to calibrate the parameters in the model to target data for Scotland from 2001-2014.
5. To validate the ability of the calibrated model to reproduce trends in the AHP observed in Scotland from 2015-2019 outside of the calibration setting.

Chapter 3 Health Inequality: background and synthesis

3.1 Chapter Overview

Firstly, this chapter will outline the definition of health inequality that will be implemented throughout the thesis. Then it will describe the prominent empirically informed theories which have been used to understand health inequality, before going on to discuss a set of theoretical frameworks which have been developed to deepen understanding of how these explanations work.

It should be noted that it is not within the aims of this PhD to complete a systematic review of theories but to give a broad representation of those developed by academics and NGOs that can be useful for studying the alcohol harm paradox. Selection of included frameworks stemmed from solicited expert opinion (Clare Bambra) and snowball methods of literature searching. This chapter identifies theories that may provide insight into the underlying causes of the AHP which are discussed in greater detail in relation to the AHP in chapter 5.

3.2 Defining Health Inequalities

The impact of health inequalities remains a priority as disparities between the rich and poor in high-income countries increasingly widen, with the disadvantaged in society suffering higher rates of serious illness and premature death (55–57). A widely accepted definition describes health inequalities as "*systematic differences in health between different socioeconomic groups within a society. As they are socially produced, they are potentially avoidable and widely considered unacceptable in a civilized society*" (58). This definition alludes not only to a quantitative difference in health outcomes, but also a moral and ethical dimension. Given the moral and ethical dimension implied in this definition, the definition of health inequality adopted in this PhD is closely aligned with the term health inequity, often thought of as referring to differences that are unjust and avoidable (59).

This PhD will also use the term SEP instead of socioeconomic status (SES), in line with recommendations made by Krieger and colleagues (60), as it represents the positions

individuals or groups hold in society relative to others based on social and economic factors as opposed to their absolute status (61). The term SES muddles two distinct aspects of SEP: 1) resources available and 2) status (meaning rank or prestige) (60). Therefore, to maintain clarity the term SEP is preferred. Numerous indicators from the individual level to the area level can be used to accurately represent SEP including but not limited to parents' education, education, income, housing, occupation, indices of multiple deprivation and so on (61).

3.3 What are the causes of health inequality?

From the outset it is critical to note that when discussing the causes of health inequality, it is to an extent possible and helpful to define three main concepts: ***theories***, ***pathways***, and ***frameworks***. A ***theory*** refers to an idea, or group of ideas, which are used to explain something observable, in this case health inequality. For example, simply put the culture-behaviour theory proposes that cultural and societal norms determine health behaviour which in turn determines health outcomes (62,63). ***Pathways*** on the other hand refer more specifically to the different ways or directions in which these theories (groups of ideas) create health inequality. For example, social selection is a pathway which proposes that health is determined by things like health behaviours, and health in itself determines an individual's SEP – those in poor health become or remain of lower SEP due to their ill health (64). The last concept to be aware of is ***frameworks***, and these draw on the theories and pathways to create a conceptual representation of the causes of health inequality. They may also attempt to combine different theories and portray interactions between the associated factors. It is important to understand all three of these concepts to understand not only the factors that influence health inequality, but how they do and the relationships between these factors. While this chapter aims to keep these terms and concepts distinct from one another to improve readability, in the literature none of these terms are tightly and unambiguously defined. Therefore, in discussing these terms they can inevitably bleed together. The next three sections will in turn discuss each of these concepts.

3.4 Empirically informed ‘theories’ of health inequality

Since the publication of the UK Black Report in 1980 (65), there has been an extensive research effort to understand the causes of health inequalities, a major health concern in the global north. Several theories have been developed to explain the causes of health inequalities. These have been termed theories of disease distribution as they integrate social and biological explanations and as a result call for intervention that tackles broader factors, not just health behaviour (66–68). A seminal report produced by the WHO outlines the theories they believe can explain the causes of health inequality within a social determinants’ framework (64). However, there are some discrepancies between this report and key texts from the academic literature.

According to key texts from the academic literature, the field has taken five main groups of theories: culture-behaviour, material (and neo-material) resources, psychosocial, lifecourse and more recently political economy (62,63). The report produced by WHO on the other hand lists only three main theoretical directions: psychosocial, social production of disease/political economy of health and eco-social (64). These three theoretical explanations are specific to the field of social epidemiology, whereas the previously mentioned five arise from a combination of work in epidemiology and general public health. As social epidemiology tends to focus on the impact of social and structural factors on health, the one major difference between the WHO perspective and key texts in the academic literature is the emphasis on the assumption that societal characteristics determine disease and health distribution (69). This work relies heavily on the social determinants of health framework (70), which at its core focuses on the economic and social conditions which influence differences in health. In contrast, theories at the intersection of epidemiology and public health focus on broader determinants of health, which additionally encompass the physical environment and individual behaviour (71). Despite this discrepancy there is overlap between the groups of theories outlined by the key texts in the academic literature and WHO which will be discussed in combination in the next this section.

3.4.1 Culture-Behaviour

Culture-behaviour theory focuses on the impact of health-related behaviours, which are more or less culturally acceptable depending on the group you belong to, as the cause of differences in health outcomes (62,63). Alcohol consumption, smoking, exercise, and diet are linked to health, with studies consistently finding that some groups more likely to engage in multiple unhealthy behaviours – men, younger age groups and there is a gradient by SEP (72). The 'hard' version of this approach argues that these behaviours completely account for variations in health outcomes (63). However, it is generally accepted that health behaviours are simultaneously a cause and a symptom of health inequalities, they are themselves a product of the socioeconomic environment (63). Cultural-behaviour theory does not appear in the theoretical background presented in the WHO report (64), however it is present in the final conceptual framework (see section 3.6 for further detail).

3.4.2 Social production of disease/Materialist theory

Materialist explanations, also referred to as the social production of disease, assert the link between wealth and resources, and health (62,63). The term materialist is often used to encompass not only individual material wealth but to implicitly refer to neo-materialist arguments which concern access to and the availability of public services (primarily healthcare) (62). This theory is highlighted in both the academic literature (62,63) and WHO report (64). Differences in material wealth determine access to transport, a healthy diet, good education, housing, and ability to engage in social participation, all of which are positively associated with health (63). Material resources can also be protective as they mean you are less likely to live in health damaging conditions or work in risky environments (63). There is a strength of evidence to support that material factors do impact health (50,63). However, a major critique is that health inequalities persist in countries with increased standards of living indicating that absolute material wealth cannot fully explain the relationship between SEP and health (63).

3.4.3 Psychosocial

Recent developments investigating the causes of health inequalities have revealed that health more closely reflects how equal a society is as opposed to the overall economic wealth of a country (63,73). The '*income inequality hypothesis'*, as it is termed, has increased interest in psychosocial explanations for health inequality (63). This theory is outlined in both the academic literature and the WHO report (62–64). These types of explanations shift the focus from material factors alone to how people view themselves in comparison to others in their society (their perceived social status), how this impacts them psychologically and the subsequent biological consequences (62–64). Feelings of subordination or inferiority have been shown to stimulate a physiological stress response which worsens health outcomes (74,75). This originates from work exploring the impact of the social environment on biological functioning which increases susceptibility to disease (76). These negative feelings which stem from the experience of living in unequal societies where people compare their status to others, are thought to weaken social cohesion, alongside chronic stress which too has health damaging effects (70,77–81).

Psychosocial factors address the biggest criticism of materialist theory, by providing an explanation as to why countries with increasingly good living standards but higher levels of inequality still have persistent health inequalities (63). There is however criticism regarding the evidence for psychosocial factors as many argue that this theory detracts focus from the underlying material causes of inequality which are inherent to the structure of society (63). In other words, psychosocial factors provide policymakers with evidence-based rhetoric for tackling health inequalities at the individual level (e.g., stress relief), instead of altering the structures that then cause negative psychological experiences.

3.4.4 Political Economy

A relatively recent development in understanding health inequalities argues that much of the existing research has focused on downstream causes, sometimes referred to as "*symptoms*", of inequality (63), even within a discipline that already emphasises the need to look upstream.

A political economy account aims to draw on the idea that cultural-behavioural, material, and psychosocial explanations are rooted in larger societal structures (e.g., politics, the economy, work, and labour market) (63). This theory emphasizes the influence of policy actors on which types of policies are introduced, which are to an extent determined by the attitudes they possess (e.g., individualistic versus environmentally or socially focused) (63). There is some evidence to support the impact of these upstream causes on health given observed differences in population health between countries. Those with a greater economic and social policy orientation (e.g., Scandinavian countries) have significantly better health outcomes compared to other political orientations (82).

3.4.5 Ecosocial

The ecosocial approach, developed by Krieger (66), is presented in the WHO report (64). This multi-level theory seeks to "*develop analysis of current and changing population patterns of health, disease and well-being in relation to each level of biological, ecological and social organization*" (66). Key to this theory is the idea that biology and biological changes are determined by the social environment. This theory places a strong emphasis on the role of biology which is broadly missing from most other theories discussed in this section. To give an example, an ecosocial viewpoint argues that observed biological differences between racial groups are the direct result of occupational and residential segregation (83). It also aims to explore how the social influences biological change over time and how genetic inheritance also plays a role in determining health inequalities (64).

3.4.6 Summary

Despite discrepancies in labelling, it is generally agreed that these theories do all play a role in creating and sustaining health inequalities. The theories outlined in key texts in the academic literature and by WHO are not necessarily incompatible with one another, but are different perspectives which concentrate on different factors. However, there is still some discussion regarding the relative importance of these factors and the pathways through which they influence health.

3.5 'Pathways' influencing health inequality

In the prominent literature there are three possible pathways through which the factors discussed above can impact health: Social Selection, Social Causation, and the Life Course. As with the theories discussed above, these pathways are not necessarily mutually exclusive. For example, social selection and causation can undoubtedly operate throughout the lifecourse. The life course pathway is also defined as a theoretical direction in key texts from the field of public health and epidemiology (62,63). However, it can usefully be thought of as a pathway through which the empirically informed theories outlined above create health inequalities (64). This section will briefly outline all three pathways.

3.5.1 Social Selection

The social selection pathway presents reverse causation as the reason for socioeconomic differences in health outcomes. In other words, it is health that determines an individual's socioeconomic position rather than socioeconomic position having an influence on health (64). This relationship results in downward social mobility such that those in poor health move down the socioeconomic gradient while those in good health move up the gradient (64). Social mobility reflects the dynamic nature of an individual's social position, which can change throughout their life, both in comparison to their parents' position and their own in earlier life (64).There is mixed evidence to support the existence of this pathway: in general, health does impact social mobility (84,85). However, some argue that social selection is not the primary pathway that produces health inequalities. They argue that those who are downwardly mobile will have better health than those already in low SEP, driving up the average health for that group (64). In contrast those who are upwardly mobile having previously been of lower SEP are more likely to be in worse health and therefore will drive down the average health when they move up in SEP (64). Given this and evidence to suggest that low SEP is present prior to the development of health problems (as discussed in the next section), some studies have concluded that social selection is not the main pathway in determining health inequalities (86,87).

3.5.2 Social Causation

The Social Causation pathway argues the opposite of social selection – that social position does determine health via several related factors (64). It is based on numerous cohort studies which reveal that low SEP is present long before the occurrence of health problems (64). This pathway of causality has been central to research investigating the causes of health inequality. This relationship between SEP and health is not direct but is argued to be due to the distribution of intermediary risk factors throughout the population (64). These "intermediary factors" relate back to the main theoretical directions discussed in the previous section: material, psychosocial, behavioural and resulting biological factors (e.g., access to services, experiences of inequality, health-related behaviours etc.).

3.5.3 Life Course

It is generally agreed among researchers that to understand health inequalities it is crucial to recognise that events and exposures occur across the life course (63). This pathway is not in itself an explanation; however, it emphasises the importance of timing and looking at the whole picture of someone's life in contrast to static timepoints (62–64). The life course pathway is not limited to a single generation and has an element of familial inheritance (64). Using this pathway to understand inequalities in health requires examination of risk factors from the very early stages in life to old age (62,63). The lifecourse pathway is particularly important given the rise in incidence of NCDs which often develop in later life and may be partially attributable to early life experiences (63).

Two main classifications of life course pathway have been presented in previous research: the *critical periods* model and the *accumulation of risk* model (88).

The first classification proposes that exposure to an event at a specific timepoint may have lifelong impacts on biological make up, which can only minimally be altered by later life experiences (64). This "*critical periods*" model is based on research that indicates the detrimental impact of adverse events during fetal or early childhood development. One extreme example of this is the deleterious effects of social deprivation in early childhood on

the brain, which leads to mild impairment, impulsivity, and attention and social deficits which persist in later life (89). It should be noted that while this model places emphasis on early life it does also acknowledge the importance of mediating factors in later life (64).

The second classification, the "*accumulation of risk*" model is complementary to the critical periods model; however, this second model suggests that individuals may accumulate risk factors for disease over time and this prolonged exposure is detrimental to biological systems within the body (64). There is good evidence that deprivation clusters and accumulates over time; those living in deprivation are more likely to experience multiple types of disadvantage (63). Shifting the focus from proximal risk factors and health outcomes to the accumulation of risk factors may have greater explanatory power.

3.5.4 Summary

There is little evidence to support that social selection is a major causal pathway of health inequalities. In contrast there is evidence to suggest that SEP influences exposure to certain risk factors which then leads to health outcomes. This has led to the development of an alternative pathway, social causation. The Life Course pathway has also been developed to enhance our understanding of the complex relationship between SEP and health. This pathway emphasizes the importance of critical timepoints (e.g. early childhood development), the cumulative impact of deprivation and the associated experiences of living in deprivation. Therefore, social causation and life course pathways may be key to fully understanding the causes of health inequalities.

3.6 Conceptual 'Frameworks'

Several frameworks exist which attempt to understand the relationship between theories, the pathways through which they are enacted, and health. These frameworks attempt to join up theories and pathways to provide an overall picture of the causes of health inequalities. A major driver in the development of these frameworks has been the WHO. Although it is generally agreed that the theories discussed in section 3.2 to some extent cause health inequalities, and that a combination of social causation and life course are the predominant

pathways, there remains debate regarding which framing, if any, should be used to understand interactions between factors discussed in theory. This section will discuss the developments of the WHO to create theoretical frameworks that can explain health inequality, specifically The Determinants of Health 'Rainbow Model' and The Social Determinants of Health frameworks. It will also discuss the main criticisms of these frameworks.

3.6.1 The Determinants of Health 'Rainbow Model'

The first notable attempt to develop a framework to contextualise the causes of health inequalities was the 'Rainbow Model' in 1991 (90). The document outlining this framework was produced for the WHO and remained influential in informing strategy papers to reduce health inequity in Europe until 2006 (91).

Dahlgreen and Whitehead developed a five-tier figure depicting the Rainbow Model to illustrate the main determinants of health (90). This included a top tier which represented the macro-structural environment. Followed by material and social conditions; the living and working conditions influenced by housing, education, health care, agriculture etc. Then people's social and community networks, specifically the support they receive from those networks. Finally, individual lifestyle factors such as diet, smoking and alcohol consumption. The bottom layer represents individual-level factors such as age and sex, which are fixed and outwith policy control.

In their initial report Dahlgren and Whitehead mapped the layers from this model to four levels of policy for intervention. At the macro-level, policies should aim to bring about "*long-term structural changes*"; some example policies given were economic strategies and tax policies (90). Policy level two focused on a multi-sectorial approach to improving the material and social conditions in which people live and work. This included policies targeting welfare benefit provision and improving healthcare. The subsequent level aimed to tackle the local community by changing social support and community cohesion to improve health outcomes. The last policy level focused on changing individual behaviours and attitudes, specifically concerning lifestyle choices with a focus on health education and additional support for groups with the

unhealthiest lifestyles. Dahlgren and Whitehead argue that policies focusing on only one level will be ineffective and it is a combination of policies throughout this spectrum that will reduce health inequalities (90).

This framework, although highly influential, has several drawbacks. The framework is implicitly static: while the authors do emphasise the importance of tackling multiple levels they do not consider interactions between factors at each level or how these may dynamically shift throughout the lifecourse. Further, whilst the framework does account for many of the social determinants of health, it lacks clear theoretical underpinning, relies heavily on social causation as a pathway and segments efforts to reduce health inequality.

3.6.2 Social Determinants of Health Framework

More recently, the WHO formed the Commission on Social Determinants of Health (CSDH) which developed a conceptual model of the SDH framework (64). This aimed to update and enhance the original Rainbow model in line with new evidence such as the influential work from Marmot and Wilkinson (55,92), among others, who have shifted the focus from behaviour to the wider social determinants of health.

There is consensus that differences in health associated with socioeconomic inequality primarily stem from social determinants, which have become central to research investigating the causes of health inequality (93). The World Health Organizations' seminal report outlining the SDH framework was an empirically led endeavor which aimed to identify risk factors from a social epidemiological perspective (64). In the creation of this framework they brought together the theoretical directions and pathways discussed in the previous sections to develop a comprehensive SDH framework. The purpose of this framework was to ensure a clear picture of the relevant factors, their relationships, and the pathways through which they cause inequalities.

In outlining their framework, they note the important contextual information which does not fit with theoretical directions and the pathways previously discussed. The first such information

is the need to acknowledge that social power is a key driver of the structure of society and therefore cannot be ignored (64). They state that developing a comprehensive theory of power and its relationship with health inequality is outside the scope of their report, but that aspects of accounts of power from philosophy and politics will be integrated into the framework (64). Based on these insights, they conclude that key to reducing inequalities is shifting the distribution of power to create a more equal society.

Additionally, the WHO acknowledge the influence that Diderichsen's model of the social of production of disease had on the development of their theoretical framework (64,94). This model puts social position at the core. There are two levels: society and individual. At the societal level social contexts are created and, in turn, form a social gradient along which individuals are positioned (64). Position on the gradient then determines an individual's exposure to certain conditions (e.g., poor housing conditions), some of which may be more health damaging. It also determined in turn the consequences of experiencing these conditions (64), which are not limited to health but can be social and economic.

The CSDH conceptual framework splits the drivers of health inequality into two sections: structural determinants and the intermediary determinants which are presented as sub-categories of social determinants (64). In the conceptual framework the structural determinants are shown to influence the intermediary determinants. The top level of the structural determinants includes governance, macroeconomic policies, social policies, public policies, and culture and societal values. The socioeconomic and political context shares a bidirectional relationship with socioeconomic position including measures of social class, gender and ethnicity, and influence education occupation and income. Socioeconomic position impacts on the experience of intermediary determinants and determines social cohesion and social capital. The intermediary determinants: material circumstances, behaviours and biological factors, and psychosocial factors then impact equity in health and well-being either directly or indirectly via the health system. Finally, equity in health and wellbeing closes the loop by feeding back to socioeconomic position and the wider

socioeconomic and political context. It is important to note that structural determinants only affect health and wellbeing through the intermediary determinants and have no direct effects in this framework.

This complex framework accounts for many of the SDH and brings together social causation and life course pathways, therefore addressing many concerns regarding the Rainbow Model. The CSDH framework recognises the importance of power in terms of how people can influence structures and how structures limit people's actions. The CSDH framework also has a clear role for SEP itself, which is missing from the Rainbow Model, therefore acknowledging how structures influence position and in turn influence the intermediary factors individuals are exposed to. However, arguably the use of theory in the CSDH framework is vague (e.g., in the documentation they outline that they use power in the conceptual framework, however power relationships are not explicated in the final diagram) and heavily based on the social epidemiological research. There is also an overreliance on the intermediary determinants as causal to health and wellbeing as opposed to a greater focus on direct pathways from structural factors to health. While the intermediary determinants may be impacted by structural factors, it is easy to see how this framework could be used to focus on intervening at the intermediary level.

3.7 Further Theoretical Considerations

Researchers have acknowledged that the prominent way of thinking of the causes of health inequality are focused on the intermediary causes of health inequalities (95). Structural factors are seen to be immovable and therefore little attention is paid to intervening at a structural level. However, theories attempting to understand the relationship between people and society often indicate a co-dependent relationship which changes over time. While an element of this is included in the CSDH framework there is a lack of explication of the fluidity of the relationship between society and individuals. To rectify this there has been a conscious effort by some to bridge the gap between the empirically informed explanations, which inform the CSDH

framework, and sociological theory. This work has drawn on theories from outside the field of social epidemiology including fundamental cause theory.

3.7.1 Fundamental Cause Theory

Much like other theories discussed in section 3.4 (e.g., political economy) FCT theory shifts the focus from proximal individual level factors to context, what puts people "at risk of risks" (96). Central to FCT are resources defined as money, knowledge, power, prestige and social connections. It is proposed that high SEP groups have increased access to these flexible resources and can employ them to avoid risks, reduce the consequences of disease and improve the uptake of treatment to improve health. Conversely, these resources are not readily available to low SEP groups. In establishing SEP as a "fundamental cause", research investigating risk factors oversimplifies the relationship between SEP by not acknowledging that these risk factors themselves are generated by social conditions. Crucially this theory does not deny the role of social determinants but instead suggests that SEP determines whether individuals can adapt to the introduction of new disease, risks or treatment (97). Øverseen and colleagues argue that the application of this theory to understanding health inequalities would see that the risk factors are rooted in SEP (97).

Those in favour of the application of FCT point to the fact that social epidemiology has exerted its efforts investigating the proximal level factors (e.g., diet and exercise) (96). This aligns with the specifically Western belief that outcomes can be individually controlled and therefore health is the responsibility of the individual (96). They do acknowledge the influence of structural factors, however given that an individual approach to public health fits with cultural values in countries such as the UK, structural factors tend to be treated as fixed. Neglecting the social conditions, from which other risk factors are formed, has arguably slowed progress in reducing health inequalities (97). Focusing on these social conditions as fundamental causes could be key to not only understanding the causes but to intervention development.

FCT is to an extent reflected in the CSDH conceptual framework developed by the WHO (64). While they do not explicitly refer to fundamental cause theory, they present a clear bi-

directional relationship between SEP and intermediary risk factors (indicating that SEP, which is generated by structural causes then leads to exposure to intermediary factors). They also explicitly state that behavioural factors are the result of fundamental causes (64). One concern is that the use of fundamental cause as a term is vague and not well-explained. The lack of explicit use of FCT has been argued to lead to the belief among social epidemiologists that social position is unchangeable and therefore is treated as such (97). The major additional concern raised by FCT is to look at the 'causes of causes' or the 'causes of structures' (96,97). Health inequalities research shaped by the social determinants of health is critically lacking this. While it is accepted that the structures that shape SEP are at the root of health inequalities (e.g., the CSDH WHO framework), most research has focused on intermediary factors. In addition to considering the traditionally used theories of health inequalities (e.g., the social determinants of health), this PhD will also consider whether alternative theories (e.g., FCT) are better placed to explain the existence of the AHP.

3.8 Summary

This chapter aimed to provide a broad overview of the literature investigating the causes of health inequality which could be applied to understand the AHP. The WHO have been a trailblazer for health inequality research and have developed frameworks including the DH and SDH which conceptualise the pathways through which these factors result in health inequalities. The theories at the core of these frameworks: cultural/behaviourial, materialist, psychosocial, lifecourse, political economy and ecosocial, have the potential to enhance our understanding of the causes of the AHP. Additionally, FCT has been identified as an alternative theory which is worth consideration. It is important to assess whether these theories have been used in the field of alcohol epidemiology – specifically in research investigating the AHP, to identify and address any gaps in their use and discuss what these alternative (non-behavioural) framings mean for a new alcohol-related harm research agenda. Therefore, the first step towards gaining an understanding of how health inequality has been used to understand the AHP should be to systematically review the literature on the explanations

provided for the AHP (Chapter 4) and then to contextualise these explanations using the theories of health inequality outlined in this chapter (Chapter 5).

Chapter 4 Causal mechanisms proposed for the Alcohol Harm Paradox: A Systematic review

4.1 Chapter Overview

This chapter presents a systematic review which explores the explanations for the AHP proposed in the existing literature. Full details of the rationale, methods, results, discussion and conclusions from this review are presented in this chapter.

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Title: Causal mechanisms proposed for the Alcohol Harm Paradox – A Systematic Review.

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4.2 Abstract

Background and Aims: The Alcohol Harm Paradox (AHP) posits that disadvantaged groups suffer from higher rates of alcohol-related harm compared with advantaged groups, despite reporting similar or lower levels of consumption on average. The causes of this relationship remain unclear. This study aimed to systematically review the scientific literature to identify explanations proposed for the AHP. Secondary aims were to review the existing evidence for those explanations and investigate whether authors linked explanations to one another.

Methods: Systematic search of MEDLINE (1946-January 2021), EMBASE (1974 – January 2021) and PsycINFO (1967 – January 2021), supplemented via manual searching of grey literature. Included papers explored the causes of the AHP, OR investigated the relationship between alcohol consumption, alcohol-related harm, *and* socioeconomic position. Papers were set in OECD high income countries. Explanations extracted for analysis could be evidenced in the empirical results or suggested by researchers in their narrative. Inductive thematic analysis was applied to group explanations.

Results: Seventy-nine papers met the inclusion criteria and initial coding revealed these papers contained 41 distinct explanations for the AHP. Following inductive thematic analysis, these explanations were grouped into 16 themes within six broad domains: Individual, Lifestyle, Contextual, Disadvantage, Upstream and Artefactual. Explanations related to risk behaviours, which fit within the Lifestyle domain, were the most frequently proposed (*n*=51) and analysed (*n*=21) by included papers. Evidence gathered in this review confirms that risk behaviour only plays a partial role while explanations in other domains, specifically the individual, contextual, disadvantage and upstream, lack empirical testing.

Conclusions: While there are many potential explanations for the AHP, risk behaviours were found to dominate the scientific literature. Applying well-established theories of health inequality and the use of computer simulations, which move from testing relationships between variables to understanding the underlying mechanisms, could advance our understanding of the paradox.

4.3 Introduction

Alcohol accounts for 5.3% of deaths and 5.1% of the burden of disease and injury globally (98). However, alcohol-related harms (e.g., deaths, illnesses and hospitalisations due partly or wholly to alcohol) are not equally distributed across socioeconomic positions (SEP) – the social and economic factors that determine an individual's position in society (60).

Disadvantaged groups suffer from higher rates of alcohol-related hospital admissions and deaths compared with advantaged groups, despite reporting similar or lower average levels of consumption (16,41). For example, in the UK, the proportion of people in the highest SEP group drinking more than 4/3 (45%) or 8/6 (23%) units per day is almost double compared to the lowest SEP (22%, and 10% respectively) (7). Despite this, the alcohol-specific mortality rate among the most deprived is 5.5 times higher (8). This relationship termed the alcohol harm paradox (AHP) is found internationally, including in the UK (16), Australia (17), the Netherlands (19) and Finland (9) and across measures of SEP (e.g. social grade, income, education, car ownership, employment and housing tenure) (5). Prior to 1980, findings suggest a clear dose-response relationship between alcohol consumption and alcohol-related hospitalization and mortality, irrespective of SEP (99–101). However, in the last 40 years the AHP has become a consistent and longstanding finding (42). Despite this there is a paucity of research attempting to understand the underlying causes of the AHP.

Several reviews and meta-analyses describe socioeconomic differences in alcohol-related harms based on existing evidence or available survey data (6,12,13,39–41). However, only a subset also focuses on the contribution of alcohol consumption to this relationship, measured as average consumption (e.g. grams or units weekly, monthly or yearly) or drinking patterns (how often and how much people drink) (12,13,41). This evidence highlights that neither average alcohol consumption nor heavy drinking patterns can explain differences in alcohol attributable outcomes between SEP groups. At best, heavy drinking occasions partially attenuate the link between SEP and, hospitalisations or mortality by 15-30% (41). Put simply, the most disadvantaged consistently suffer disproportionate risks of harm from their alcohol

consumption when compared to their advantaged counterparts, which is not only a health burden on society but contributes to increasingly widening health inequalities (49).

Empirical studies of the AHP have largely focused on proximal individual-level factors as potential explanations. The role of unrecorded alcohol consumption has to an extent been investigated, and results suggest underreporting is similar across socioeconomic groups (38). Cross-sectional studies have also tested differences in drinking patterns, behavioural clustering, and drinking histories (38,43). Although there is evidence that low SEP groups tend to have heavier drinking patterns (38,43) and engage in multiple risky health-related behaviours (38), fewer studies go on to test the degree to which lifestyle risk factors explain differences in alcohol-related harm. One study highlighted that the rate of alcohol-attributable mortality and hospital admissions was three times higher for the most disadvantaged compared with the most advantaged, this association remained after adjusting for weekly consumption and heavy drinking occasions, and it was only slightly attenuated after further adjusting for BMI and smoking (16). While investigation of lifestyle factors is prominent, other potentially fruitful avenues of explanation, such as social and economic causes (e.g. social support, housing and employment), have been neglected.

Substantial socioeconomic gradients in health exist across countries and contexts (50,102). There is a critical need for evidence to support public health policies that tackle not only behaviour, but also the broader social determinants of health to mitigate the AHP. This study aimed to review explanations for the paradox put forward in relevant scientific literature. Secondary aims were to review the existing evidence for or against these explanations, and to explore how authors combine different explanations to shed light on potential relationships between different causal factors. To our knowledge this is the first review to collate explanations for the AHP.

4.4 Methods

Search strategy

We followed the Preferred Reporting Items for Systematic Reviews and Meta-Analyses guidelines (PRISMA) (103). The protocol for this study can be found at: http://doi.org/10.13140/RG.2.2.25606.60489. MEDLINE (1946 – January 2021), EMBASE (1974 - January 2021) and PsycINFO (1967 – January 2021) were searched to identify peer-reviewed literature on the topic of the AHP or studies that investigated the relationship between alcohol-related harm, socioeconomic position, and alcohol consumption. An extensive list of search terms was used (see Table S1, appendix A) to capture the themes of alcohol (e.g., alcohol adj3 drink*) and socioeconomic factors (e.g., disadvantage*). Given the large number of results returned during test searches, further specifications were made by focusing on papers with alcohol in the title and some exclusory terms were included (e.g., NOT therapeutics). Terms were tailored dependent on database requirements. For grey literature, Google and Google Scholar were searched, and this was supplemented via expert identification of relevant reports (CA).

Inclusion and exclusion criteria

The population, exposures, comparisons, outcome, and study designs (PECOS) criteria for inclusion are listed in Table 4.1. Studies were included if they: (i) were full papers published in English (ii) explicitly explored the AHP *OR* investigated the relationship between: alcohol-related harm, socioeconomic position, and alcohol consumption (Table 4.1). We focused on high income OECD countries as classified by the World Bank (104), primarily due to differences in alcohol environments between high- and low-middle income countries (e.g., greater availability of informally produced alcohol in low-middle income countries) (105). A range of study designs were eligible for inclusion. Systematic reviews and meta-analyses were included, as it is equally possible to extract 'explanations' for the paradox from these studies. However, intervention and treatment studies were outside the scope of this review.

Additionally, empirical studies which analysed data exclusively collected *pre*-1980s were excluded.

Table 4-1: *Population, exposures, comparisons, outcomes, and study design criteria for study inclusion.*

Criteria	Definition
Population	OECD high income countries only
Exposures	Alcohol consumption (any measure including both self-report (e.g., quantity/frequency, heavy drinking occasions), biological indicators (e.g., blood alcohol concentration) and aggregate sales data (e.g., per capita consumption)
Comparisons	Socio-economic position (any measure including area-level deprivation and individual measures (e.g., educational attainment, occupation, and income level))
Outcomes	Alcohol-related harm (any measure which relates to health harms (e.g., morbidity and mortality), clinical diagnosis of alcohol use disorder using ICD codes or DSM-IV manual or negative alcohol-related consequences (e.g., had an accident))
Study designs	All designs were considered both quantitative and qualitative - including secondary research, intervention studies were excluded

Screening

All records were imported to EndNote Online and duplicates were removed. Titles and abstracts were screened to identify papers matching the inclusion criteria. Full-text versions of the papers were then screened to determine inclusion. Initial screening was carried out by one reviewer (JB). A second reviewer (OS) then randomly screened a sample of the included studies (n=20) to validate that papers were correctly included. There was no disagreement between reviewers regarding inclusion.

Data extraction

Data from the papers were extracted by one reviewer (JB). A second reviewer (OS) independently assessed the accuracy of data extraction for a sample of the included studies (n=20). In the case of disagreement both reviewers referred to the paper in question, and a consensus was reached. A data extraction matrix was developed, which included

characteristics of the studies (design, year of data collection and location), participants (age, target population and sample size), measures (unit of analyses, SEP, alcohol consumption and alcohol harm measures) and outcomes (main findings and explanations for the AHP). Both tested and hypothetical explanations were extracted. 'Explanations' were any reasons identified from the empirical results or proposed by the authors which explain why alcohol-related harm outcomes were worse for those of a low SEP. Explanations were commonly taken from the results and discussion sections of empirical papers or the main body of other types of included paper. Hypothetical explanations were extracted verbatim. The evidence for these explanations was also extracted from included primary research or from authors citing other research findings when proposing an explanation.

Quality assessment

Quality appraisal of the included studies was conducted by one researcher (JB) to assess risk of bias. The AXIS critical appraisal tool (106), CASP Qualitative, CASP Systematic Review, CASP cohort study and CASP case-control study checklists (107) were used depending on the study design. Commentaries, author replies, discussion papers and reports were not critically appraised. Overall, the quality of included papers was assessed as good. More information on critical appraisal can be found in Table S2, Appendix A.

Analysis

Descriptive summary statistics were used to describe search results and study characteristics. An inductive thematic approach was taken to analyse the explanations provided by included papers. This aimed to group explanations within broader themes. Explanations were coded and initially analysed by one researcher (JB) in consultation with co-authors (RP & JH). In the instance where an author meaningfully linked multiple explanations in the text, this was recorded as a connection. A narrative synthesis of the findings providing evidence for or against the extracted explanations was also conducted.

4.5 Results

Descriptive analysis

Searching of electronic databases returned 20,828 records. A further 13 records were identified from the grey literature. Total records reduced to 18,790 following de-duplication. Of the 18,790 records, following title and abstract screening, 195 were selected for full-text screening and 79 of these met the inclusion criteria for data synthesis (Figure 4.1). Attempts to retrieve inaccessible papers were made through the search databases, University Library services and Google Scholar. Study characteristics are displayed in Table 4.2.

***Figure 4-1**: PRISMA Flow Diagram.*

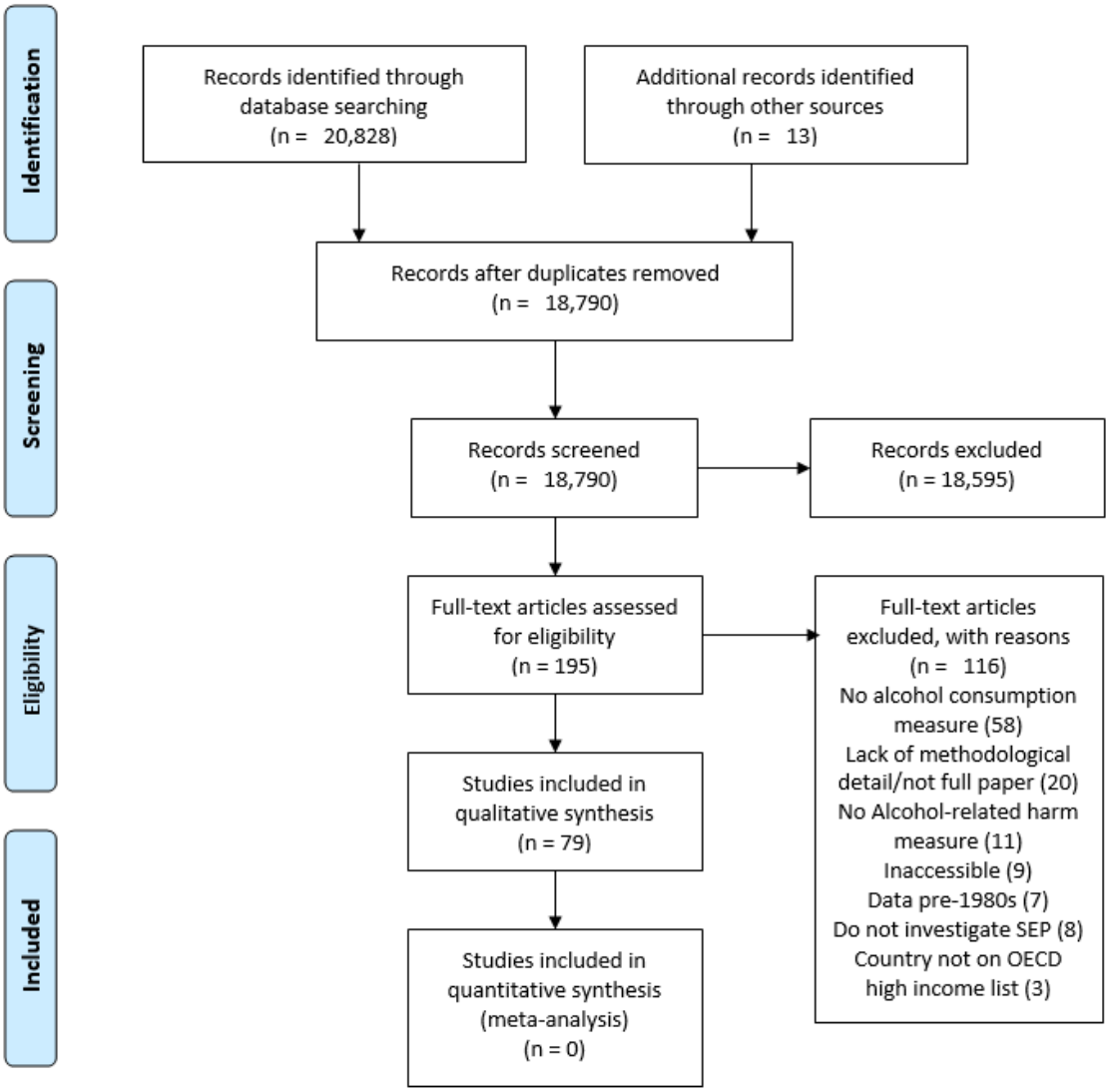


Table 4-2: *Characteristics of included papers.*

Author, Year	Country	Study Design	Study Year	Population	Sample Size	Age	Measurement Level	Harm Measure	SEP Measure	Consumption Measure	Evidence of the AHP
Alcohol Research UK, 2015 (108)	UK	Report	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Backhans et al., 2016 (26)	Sweden	Cohort	2002-11	G	15,841	18-84	I	AR Hosp & Death	ES, E	Last 12 months; Drinks/week; Binge drinking	Yes
Beard et al., 2016 (5)	UK	Cross-sectional	2014-15	G	1,700	16+	I	AUDIT-H, AUDIT-D	O, I, E, ES, H	AUDIT-C	Yes
Bellis & Hughes, 2009 (109)	UK	Report	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Bellis et al., 2016 (38)	UK	Cross-sectional	2013-14	G	6,015	18+	I & AG	N/A	A	Last 12 months, Units/week	N/A
Bloomfield, 2020 (110)	Denmark	Commentary	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Boyle et al., 2014 (111)	Australia	Case-Control	2005-2007	G	918 (cases), 1,021 (controls)	40-79	I & AG	Colorectal Cancer	A	g/week	N/A
Breakwell et al., 2007 (34)	UK	Cross-sectional	1991-2004	G	N/A	15+	AG	AR Death	A	Units/week	Yes
Brown et al., 2014 (112)	USA	Cross-sectional	2010-11	G	663	19-91	I	Somatic Complaints	E	Drinks/month	N/A

Chick, 1998 (113)	UK	Review	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Collins, 2016 (39)	USA	Review	N/A	N/A	28 studies	N/A	N/A	N/A	N/A	N/A	N/A
Connor et al., 2010 (114)	New Zealand	Cross-sectional	2006-2007	G	1,770	18-70	I & AG	Negative AR consequences	E, A	Drinking days last 12 months, drinks/occasion, binge drinking	N/A
Conway et al., 2015 (27)	EU, Americas	Case-control	1988-2007	G	23,964 cases, 31, 954 controls	NR	I	Head and neck cancer	E, I	Drinker status, drinks/day	Yes
Degerud et al., 2018 (35)	Norway	Cohort	1960-2011	G	207,394	NR	I	Cardiovacular Disease, Ischemic Heart Disease, cerebrovascular and all-cause mortality	H, I, E	g/day, Heavy drinking episodes	Yes
Evans-Polce et al., 2016 (115)	UK	Cohort	1958-2006	G	11,469	7-55	I	All-cause mortality	PI, H, O	Units/week	N/A
Fair Foundation, 2015 (116)	Australia	Report	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Fillmore et al., 1998 (36)	USA & Sweden	Meta-analysis	1964-1982	N/A	31 studies	16+	I	All-cause mortality	E, ES, I	Drinks/occasion, occasions/month, drinks/month	Yes
Gartner et al., 2019 (117)	Wales	Record-linkage	2013-2016	G	11,038	16+	I	AR Hosp	A, SC, E, ES, H	Units/heaviest drinking day, last 12 months	Yes

Hall, 2017 (118)	UK	Commentary	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Hart, 2015 (119)	Australia	Qual	N/A	Young Adults	N/A	18-24	I	N/A	N/A	N/A	N/A
Herttua et al., 2007 (120)	Finland	Cohort	1985-2003	G	70.1 million	15+	I & AG	AR Death	E	Litres/capita	N/A
Huckle et al., 2010 (121)	New Zealand	Cross-sectional	1995, 2000 & 2004	G	3,848, 4,295 & 5,477	18-65	I	Negative AR Consequences	E, I, O	Litres/year	Yes
Jonas et al., 1999 (122)	Australia	Cross-sectional	1995-96	G	NR	N/A	AG	AR Hosp	ES, O, H, I, MV	Litres/capita	N/A
Jones et al., 2015 (13)	EU & Americas	Systematic Review	2012	N/A	31 studies	N/A	I & AG	AR Morb & Death	E, O, I, A, ES, H, OM	g/year, g/day, drinks/week, units/week, drinks/day, drinking status, ml/day, days drank/week, glasses/day, binge drinking, years vodka consumption, drinks/last 12 months	Yes
Karriker-Jaffe et al., 2012 (22)	USA	Cross-sectional	2000 & 05	G	7,613 & 6,919	18+	I & AG	Negative AR consequences, AD	A	Drinks/last 12 months, Heavy drinking	Yes

Karriker-Jaffe et al., 2013 (21)	USA	Cross-sectional	2000 & 05	G	7,613 & 6,919	18+	I & AG	Negative AR consequences	A	Drinks/last 12 months, Heavy drinking	Yes
Katikireddi et al., 2017 (a) (16)	UK	Record-linkage	1995-2012	G	50,236	M=48	I & AG	AR hosp, death and prescription	E, A, O, I	Units/week, binge drinking	Yes
Katikireddi et al., 2017 (b) (123)	UK	Commentary	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Kuendig et al., 2008 (124)	EU	Cross-sectional	1997-2002	G	N/A	25-60	I	Negative AR consequences	E, ES	Grams/day, binge drinking	Yes
Lawder et al., 2011 (28)	UK	Cohort	1998-2008	G	8,305	M=47	I & AG	AR hosp	ES, B, A	Units/week	Yes
Lewer et al., 2016 (43)	UK	Cross-sectional	2008-2013	G	51,498	18+	I & AG	N/A	I, E, ES, A	Heavy episodic drinking, Heavy weekly drinking	N/A
Livingston, 2014 (18)	Australia	Cross-sectional	2010	G	21,452	12+	I & AG	N/A	A, I	Drinks/year, risky drinking	N/A
Lundin et al., 2012 (29)	Sweden	Cohort	1969-1991	MC	37,798	18+	I	AR hosp	PI, O, E, I	Risky alcohol use	Yes
Major et al., 2014 (30)	USA	Cohort	1995-2006	G	4,814,247	M=63	I & AG	Hepatocellular carcinoma incidence, chronic liver disease mortality	A	Drinks/day	Yes
Makela & Paljarvi, 2007 (9)	Finland	Cohort	1969-2000	G	6,406	25-69	I	AR hosp & death	O	CI/year	Yes

Makela, 2008 (125)	Finland	Commentary	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Marmot, 2001 (126)	UK	Commentary	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Mayor, 2016 (127)	UK	Commentary	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
McDonald et al., 2008 (31)	UK	Record-linkage	1995-2005	G	23,183	30+	I & AG	AR discharge diagnosis	A	Units/week	Yes
Meier et al., 2017 (128)	UK	Discussion	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Menvielle et al., 2004 (32)	France	Case-control	1989 & 1991	MP	504 cases, 242 controls	<50 - >70	I	Laryngeal or hypopharyngeal cancer	E, O, OM	Glasses/day	Yes
MESAS, 2016 (129)	UK	Report	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Moller et al., 2019 (130)	Denmark	Cross-sectional	2014	Young Adults	70,566	M= 17.9	I	Negative alcohol consequences	PI	Standard drinks/week	Yes
Mulia & Karriker-Jaffe, 2012 (131)	USA	Record-linkage	2000 & 2005	G	13,231	24+	I & AG	Negative alcohol consequences, AD	E, A	Drinking status, risky drinking, monthly drunkenness	N/A
Mulia & Zemore, 2012 (132)	USA	Cross-sectional	2005	G	4,080	18+	I	AD	Poverty status	Frequency of drunkenness in the last year	N/A
Nielsen et al., 2004 (133)	Denmark	Cohort	1976-2001	G	14,223	20+	I	All-cause mortality	E, I	Frequency of types	N/A

Norstrom & Landberg, 2020 (134)	Sweden	Cohort	1994-2017	G	N/A	N/A	AG	Alcohol-specific mortality & violent deaths	E	Per capita consumption	Yes
Norstrom & Romelsjo, 1999 (135)	Sweden	Cross-sectional	1990 & 1991-1995	M	2,817	20-64	I	AR death	O	Litres/year	No
Nweze et al., 2016 (136)	USA	Cross-sectional	2013	P	738	15-70	I	AR hosp	ES, IN	BAC	N/A
Parkman et al., 2017 (137)	UK	Qual	2015	P	30	16+	I	AR hosp	E, H, ES	Current & previous use	N/A
Pena et al., (2020) (47)	Finland	Eight Cohort studies	1978-2016	G	52,164	25+	I	AR death	I, E	g/week, Alcohol Biomarkers	Yes
Pena et al., (2021) (138)	Finland	Eight Cohort Studies	1978-2016	G	53,632	25+	I	AR death	I, E	g/week	Yes
Probst et al., 2020 (41)	Canada	Systematic Review/ Meta – analysis	2020	N/A	10 studies	N/A	N/A	N/A	N/A	N/A	Yes
Public Health Wales, 2014 (139)	UK	Report	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Rehm & Probst, 2018 (140)	Canada	Discussion	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A

Rhew et al., 2020 (141)	USA	Cohort	N/A	Young Adults	746	18-23	I	Negative alcohol consequences	PI	Standard drinks/week	N/A
Roberts et al., 2008 (142)	UK	Record-linkage	1998-2003	P	52,096	<35 - >75	I & AG	Pancreatitis incidence & death	A	Binge drinking	No
Roberts et al., 2013 (143)	UK	Record-linkage	1999-2010	P	19,196	<35 - >75	I & AG	Pancreatitis incidence & death	A	Units/day in the previous week	No
Roche et al., 2015 (144)	Australia	Review	N/A	N/A	138 studies	N/A	N/A	N/A	N/A	N/A	N/A
Romelsjo & Lundberg, 1996 (145)	Sweden	Cross-sectional	1967-1993	G	NR	25-64	I	AR hosp & deaths	O	g/day	Yes
Sadler et al., 2016 (44)	UK	Cross-sectional	2010-2013	P	9.6 million HES alcohol admissions	18+	AG	AR hosp	A	N/A	N/A
Salom et al., 2014 (23)	Australia	Cohort	1981-2002	Young Adults	2,399	0-21	I	Mental health & AD	ES, PI, PES	Drinks/occasion	Yes
Sargent, 1989 (146)	Australia	Discussion	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Shaper et al., 1988 (147)	UK	Cohort	1978-1987	M	7,735	40-59	I	All-cause mortality	O	Units/week	Yes
Singh & Hoyert, 2000 (37)	USA	Cohort	1979-89 & 1990-92	G	370,500	25+	I & AG	Cirrhosis and chronic liver	ES, E, PI, O	Per capita consumption	Yes

								disease mortality			
Skogen et al., 2019 (148)	Norway	Cross-sectional	N/A	G	4,311	16-72	I	AUDIT	O, I, ES	AUDIT-C	N/A
Smith & Foster, 2014 (42)	UK	Report	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Stanford-Moore et al., 2018 (149)	USA	Case-control	2002-2006	P	1,153 cases, 1,267 controls	20-80	I	Squamous cell carcinoma of the head and neck	I, E, IN	Drinking status, years drank, g/lifetime	Yes
Stewart et al., 2017 (33)	UK	Cohort	2000-2014	Adults with LTC	95,991	18+	I & AG	All-cause mortality	A	Drinking status, units/week	Yes
Syden et al., 2017 (25)	Sweden	Cohort	2002-2011	G	17,440	25-64	I	AR hosp & death	O	g/week, Heavy drinking	Yes
Thern et al., 2019 (150)	Sweden	Cohort	2013-2014	Young Adults	1,005	17-29	I	AUD	ES	Weekly binge drinking	Yes
Thor et al., 2019 (151)	Sweden	Cross-sectional	2015-2016	Young Adults	6,153	17-18	I	Negative alcohol consequences	PI, A, Academic Orientation	Binge drinking	Yes - for 2/3 SEP measures
Trias-Llimos et al., 2020 (152)	Europe	Cross-sectional & Cohort	2011-2015	G	159,132 person – years at risk	50-85	I	All-cause mortality	E	AUDIT-C	Yes

Van Oers et al., 1999 (19)	The Netherlands	Cross-sectional	1994	G	3,537	16-69	I	Negative alcohol consequences	E	Type, days/month, glasses/occasion	Yes
Whitley et al., 2014 (153)	UK	Cohort	1990-2008	G	C1= 1,444, C2= 1,550	35+	I	All-cause mortality	O, I, E	Units/week	Yes
WHO, 2014 (a) (1)	Global	Report	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
WHO, 2014 (b) (98)	Global	Report	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
WHO, 2018(154)	Global	Report	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Wood & Bellis, 2015 (155)	EU	Report	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A

NR, not reported. N/A, not applicable. G, general population. MC, military conscripts. P, patient. MP, male patients. M, males. LTC, long-term conditions. I, individual. AG, aggregate. AR, alcohol related. AD, alcohol dependence. AUD, alcohol use disorder. ES, employment status. E, education. O, occupational social grade. I, income. H – home ownership. A, measure of area-level deprivation. OM, occupational mobility. IN, insurance. PI, parental indicators. PES, partner employment status. MV, Motor vehicles. B, benefits. SC, social class. G, grams. CL, centilitres. BAC, blood alcohol content.

The largest number of papers came from the UK (n=27). Other countries providing several papers included the USA, Sweden, Australia, New Zealand, Finland, France, Denmark, Canada, The Netherlands, and Norway. Some studies were set at a continental (e.g., Europe) or global level. Of the included empirical studies, cohort (n=26), cross-sectional (n=21), case-control (n=4) and qualitative (n=2) designs were employed. One used both cross-sectional and longitudinal data. Included reviews and meta-analyses (n=5) contained a total of 238 studies. Commentaries (n=6), debate/discussion papers (n=4) and reports (n=10) were also included.

Empirical studies covered the general population (n=37), patients only (n=7), young adults (n=6), men only (n=2), adults with long-term health conditions (n=1), and military conscripts (n=1). The existence of the alcohol harm paradox was explicitly explored in 39 of the empirical studies. Of the identified papers, only seven included explicit theoretical discussion.

Of the empirical studies the majority used at least one quantity/frequency measure of alcohol use (n=36). Other measures included hazardous consumption, heavy drinking episodes, per-capita consumption, alcohol biomarkers and blood alcohol concentration (Table 4.2). Measures of SEP included individual-level (e.g., education) and area-level deprivation measures (Table 4.2). Most studies used physical health harm outcomes, including deaths, hospitalisations or disease states wholly and/or partially attributable to alcohol (n=36). Other harm outcomes included negative alcohol-related consequences and alcohol use disorder or dependence (Table 4.2).

Evidence of the AHP

Only three of the included empirical studies found that those of a lower SEP had higher alcohol consumption which then led to increased harm, two of which were specifically focused on pancreatitis (135,142,143). Therefore, generally the evidence base supported the existence of the AHP (n=36, including 3 meta-analyses of a total of 72 studies), excess harm among those of lower SEP could not be explained by the volume of alcohol consumed.

Thematic Analysis

Initial coding revealed 41 explanations for the AHP. The explanations were often presented in discussion sections, did not draw on existing theory and often appeared to be post-hoc explanations for findings. Following inductive thematic analysis of the 41 explanations, we identified sixteen themes and then grouped these themes into six domains: Individual, Lifestyle, Contextual, Disadvantage, Upstream and Artefactual. Domains, themes and explanation definitions are shown in Table 4.3. The number of papers suggesting each theme as an explanation is presented, however it should be noted that this is a metric of popularity rather than merit. There was no obvious connection between study design or population, and the type of explanation given (Table 4.3). Themes were not mutually exclusive, and authors often combined or indicated interactions between explanations. These relationships are highlighted in a network diagram (Figure 4.2).

Table 4-3: *Thematic Table of explanations for the AHP extracted from included papers with information on type of study design and population.*

Domain	Theme	Explanation	Definition	Study Design	Population
Individual	Biological	Biological Characteristics (35,41,113)	SEP groups have a different biological or genetic make-up related to ethnicity or due to experiencing inequality which leaves them more susceptible to harm.	Systematic Review, Discussion Paper, Cohort	General Population
		Behavioural related alterations (35,38,126,138,154)	Engaging in multiple risk behaviours has a biological impact: (i) nutritional deficiencies and metabolic consequences which alter protein and vitamin absorption, (ii) an adverse effect on the immune system and (iii) they interact with live enzymes, all leading to greater risk of disease (e.g. liver disease) and harm.	Cross-sectional, Cohort, Commentary, Report	General Population
	Psychological	Stress (16,21,36,37,41,113,138)	Low SEP groups experience more psychological stress and a greater number of stressful events: (e.g., marital breakdown, dangerous environment, immigrant status, unemployment and living in poverty). This is thought to reduce resilience to disease.	Systematic Review, Cohort, Discussion Paper, Meta-analysis, Cross-sectional	General Population
		Coping (19,22,29,37,44,112,113,119,120,131,132,135,137,150)	Differences in coping strategies: Low SEP groups use alcohol as a coping strategy which can lead to alcohol dependence. They are also more likely to use resigned acceptance as a coping strategy and are less likely to use cognitive avoidance and emotional discharge which independently negatively impact wellbeing.	Cross-sectional, Discussion Paper, Cohort, Qualitative	General Population, Men, Young People, Military Conscripts, Patient
		Stereotypes/Stigma (21,22,131,132,144,146)	Lower SEP groups experience more labelling and discrediting which leads to social rejection and exclusion. This	Discussion Paper, Cross-sectional, Cohort, Review	General Population

			could result in a self-fulfilling prophecy, whereby members of that group enact the behaviours they are expected to possess. This could also increase group and individual tensions which find an outlet via harmful drinking. This may also lead to fewer social resources, increasing psychological vulnerability.		
		Attribution (19,124)	There are a higher number of abstainers in low SEP groups, therefore the alcohol problems faced by those who do drink in this group may seem worse by comparison. This only holds true for subjective measures of alcohol-related harm.	Cross-sectional	General Population
	Health & Wellbeing	Physical Health (9,19,27,28,33,36,44,113,115,117,137,139,142,144,147)	There is a higher prevalence of pre-existing physical health conditions, poorer general health, multi-morbidities or being overweight/obese in low SEP groups which could explain disproportionate effects of alcohol.	Cross-sectional, Cohort, Review, Case-control, Meta-analysis. Qualitative, Report	General Population, Patient, Men, Adults with Long-Term Conditions
		Mental Health (19,28,33,36,43,44,108,113,117,130,137,144,151)	Low SEP individuals tend to be more psychologically vulnerable and have a greater prevalence of pre-existing mental health conditions, mental distress, or psychological symptoms (e.g., nervousness, irritability, helplessness, loneliness) which could exacerbate the effects of alcohol. There is also an independent association between poor wellbeing and worse health outcomes.	Cross-sectional, Review, Cohort, Report, Meta-analysis, Qualitative	General Population, Patient, Adults with Long-Term Conditions, Young Adults
Lifestyle	Risk Behaviour	Drinking Patterns (5,13,16,19,21,25,28,30,31,33,34,37,38,41–44,47,98,108–	Although overall or average alcohol consumption may be similar, or lower for low SEP groups, they consume greater	Report, Systematic Review, Meta-	General Population,

	110,113,114,117,121,122,124,126,127,134,135,138–142,147,148,152,155)	quantities of alcohol per drinking occasion.	analysis, Cross-sectional, Cohort, Review, Commentary, Discussion Paper	Men, Patient, Young Adults
	Clustering of Health Behaviours (5,13,16,21,27,28,30,32,33,35,36,38,41,43,47,108,111,117,133,138,140,149,150,153,155)	Those in low SEP groups engage in multiple health risk behaviours for example smoking, poor diet, a lack of exercise and concurrent drug use which exacerbate the impact of alcohol.	Systematic Review, Meta-analysis, Cross-sectional, Cohort, Case-control, Report, Discussion Paper	General Population, Adults with Long-Term Conditions, Young Adults, Male Patients, Patient
	Type of Beverage (5,13,16,38,117,127,133,137,142,154)	Beers, ciders, and spirits are more commonly consumed by low SEP, while wine is often associated with higher SEP. The quality and price of alcohol consumed may impact harm outcomes.	Cohort, Systematic Review, Meta-analysis, Cross-sectional, Commentary, Qualitative, Report	General Population, Patient
	Drinking History/Future Drinking (16,31,38,43,118,147,155)	Drinking is temporal and may change throughout the lifecourse. Although those of low SEP may have reduced consumption upon measurement, increased susceptibility to harm could be due to previous drinking. There are several reasons why people may reduce consumption (e.g., developing an illness). This explanation was extended to an increase in consumption in the future, as some studies only measure consumption at baseline and outcomes in following years.	Cohort, Cross-sectional, Commentary, Report	General Population, Men

	Drinking Practices	Norms (1,108,116,119,126,129,136,141,144)	Group and neighbourhood norms including drinking pattern, expected volume, how to drink certain beverages (e.g., shot a spirit) and norms around the permissibility of excessive alcohol use differs by SEP.	Cohort, Report, Qualitative, Review	Young Adults, Patient
		Culture (116,119,131,144)	Drinking culture attached to certain places of employment or neighbourhoods may lead to poorer health and difficulties maintaining employment, which could then exacerbate stress and increase consumption.	Report, Qualitative, Cohort, Review	Young Adults, General Population
	Health-Consciousness	Health literacy (38,118,137,139,145,153)	Engagement with health promotion campaigns and preventative services. It was proposed that low SEP may not make use of available services or are slower to access these services.	Cross-sectional, Cohort, Commentary, Qualitative, Report	General Population, Patient, Men
		Healthy Behaviours (9,36,113,126,153)	Those of a high SEP adopt healthy behaviours (e.g., good diet and exercise) which may protect against negative impacts of drinking.	Cohort, Review, Meta-analysis, Commentary	General Population
Contextual	Social	Social Support (1,9,13,23,25,34,36,37,108,113,115, 116,132,137,141,144,154,155)	Social support may buffer the negative impacts of alcohol consumption. Those of high SEP have a wider “social margin” which insulates them from the negative consequences of their actions while low SEP lack social support and are often socially isolated.	Systematic Review, Meta-analysis, Report, Cross-sectional, Review, Cohort	General Population, Young Adults
		Social Exclusion (36,98,116,131,144)	The marginalisation of low SEP groups is greater due to several factors including a higher number of abstainers, stigmatization that comes with having an alcohol use disorder and intersections	Report, Meta-analysis, Cohort. Review	General Population

		between multiple minority status (e.g., ethnic, refugee, homeless and LGBT+).		
	Peer Influence (1,9,108,116,144,154)	Negative influence from peers and family in low SEP groups may impact harm outcomes. There is evidence that men of high SEP are more likely to be married and therefore long-term partners may be an important agent of social control for excessive drinking. Not only would a partner provide social control but also additional financial support via combined income and this influence was extended to others in their social network.	Cohort, Report, Review	General Population
Drinking Context	Dangerous Environment (9,13,44,98,119,121,125,128,130,151,154)	Low SEP are more likely to drink in dangerous environments with a lack of policing and safety, which may lead to a higher risk of violence, police encounters and unintentional injury.	Report, Systematic Review, Meta-analysis, Cohort, Discussion Paper, Cross-sectional, Qualitative, Commentary	General Population, Young Adults, Patient
	Exposure (154)	Drinking in public places is common amongst the most deprived groups (e.g., the homeless). This leaves them exposed to certain infectious diseases (e.g., TB and HIV) which may compound harm.	Report	N/A
Place	Neighbourhood Deprivation (5,13,21,108,122,125,132,138,149,151,154)	A lack of resources, treatment facilities or preventative/educational programs, an increased police presence, neighbourhood disorder, low educational ethos and a lack of community institution negatively impact harm outcomes.	Systematic Review, Meta-analysis, Cross-sectional, Report, Case-control, Cohort, Commentary	General Population, Patient, Young Adults

		Alcohol Outlet/Advertising Density (21,30,41,108,110,114,116,129,132, 144,154)	Increased outlet density has an impact on patterns of drinking and harmful consequences. The density of alcohol advertising in deprived areas was also considered to potentially influence the excess harm experienced by those of a low SEP.	Systematic Review, Report, Cross-sectional, Cohort, Commentary, Review	General Population
Disadvantage	Intersectionality	Multiple Minorites (1,21,22,36,37,136,144,149)	The impact of belonging to multiple minority groups (e.g., SEP, race, gender, and sexuality), and how experiencing multiple aspects of disadvantage may amplify inequalities in alcohol-related harm.	Case-control, Cohort, Meta-analysis, Cross-sectional, Review, Report	Patient, General Population
	Lifecourse	Cumulative Effects (9,23,27,29,37,125,144,149,153,154)	The accumulation of negative/stressful life events over time or additive effects of prolonged risky health behaviours which negatively impacts health and potentially employment itself.	Cohort, Case-control, Commentary, Review, Report	General Population, Patient, Military Conscripts
		Early risk factors (9,23,29,116,144,151,155)	The experience of ACE's in childhood, childhood household dysfunction and a disadvantaged start in life (including prenatal factors) perpetuates a vicious cycle of poverty and poor health which impacts on social participation, wellbeing, their ability to cope and access to available support or treatment.	Cohort, Cross-sectional, Report, Review	General Population, Young Adults, Military Conscripts
		Family Influence (21,23,154)	Limited family income restricts material resources and creates stress given the inability to meet basic needs. Family history of alcohol problems could impact alcohol consumption and health in later life. Parental education is shown to negatively impact on health literacy and	Cohort, Cross-sectional, Report	General Population

			children's employment aspirations, opportunities, and adulthood income.		
	Material	Material resources (16,23,27,34,42,98,121,137,141,144)	A lack of resources could negatively impact on harm due to the inability to protect themselves from the experience of a problem or stressful life event and could exacerbate poor health through poor housing conditions, homelessness, and unemployment.	Report, Cohort, Cross-sectional, Case-control, Qualitative, Review	General Population, Young Adults, Patient
	Neo-Materialist	Access, Quality and Barriers (5,13,18,23,36–38,41,42,44,47,98,108,116,129,137,138,144,149,154,155)	Depending on geographical distribution, services in disadvantaged areas may be fewer and more difficult to access or of a lower quality. Low SEP groups face several potential barriers when attempting to access healthcare including cost, transport, availability (in terms of opening hours), mobility issues and stigma which may deter them from using services. Dependent on country there were additional considerations for example the cost of health insurance.	Report, Systematic Review, Cohort, Meta-analysis, Qualitative, Review, Cross-sectional, Case-control	General Population, Patient
Upstream	Structural	Economic (25,39,98,108,112,116,120,129,144,145,154)	Trickle-down effects of the economy were thought to contribute to excess harm. Economic stressors (e.g., economic downturns or recession) are more closely associated with morality in the lowest SEP groups. Gross national income and changes in minimum or disposable income has increased the buying power of low SEP groups, which has led to an equalization of alcohol consumption.	Report, Review, Cross-sectional, Cohort	General Population
		Socio-political (18,22,116,125,138,144,149)	The attitudes and decision making of residents and policy makers. Politicians focusing on individual behaviours rather	Cross-sectional, Case-control, Cohort, Report,	General Population, Patient

	than tackling the social determinants of health which increases inequalities. Political context is extremely important as countries with poor minimum living standards, limited public investment in social goods (particularly in deprived areas) and worse social system responses are likely to worsen health outcomes for low SEP groups.	Commentary, Review	
Alcohol Policy (33,116,129,144,146)	The mutually beneficial economic relationship between the state and the alcohol industry shapes policy decisions. Although it is hoped that this is counterbalanced by 'helping professions' it is also in their interest to continue the expansion of treatment and this is deflected by each entity casting blame on the another. Additionally, a lack of policy that aims to reduce harmful consumption, alcohol availability, pricing and promotion, and global market liberalization (changes in affordability), production, importation, distribution, and pricing of alcohol were hypothesised to contribute to the AHP.	Cohort, Discussion Paper, Report, Review	Adults with Long-Term Conditions
Corporate Influence (146)	The alcohol industry funds alcohol research which may misinform policy decision making. Privately owned media was also argued to play a role via diffusing true or false information.	Discussion Paper	N/A
Employment (9,21,27,32,37,42,116,144)	There were several mechanisms through which employment could worsen alcohol-related harms for low SEP groups. This included the working conditions or occupational exposures faced by low SEP individuals. Job type,	Cohort, Report, Case-control, Cross-sectional, Review	General Population, Male Patients

	low wages and inflexible employment, and job alienation, stress and low satisfaction are all thought to negatively impact harm outcomes. Those from more deprived backgrounds with insecure employment may also be less able to take time off work when they get ill, compounding the problem. This contrasts with the idea that high SEP individuals may get more support from their employers, whereby employers are more willing to invest energy in solving their alcohol problems. Relatedly issues of unemployment were also discussed including the issue of receiving additional help of benefits related to a long-term condition or disability which may discourage some people from getting better as they would lose this additional help as a result.		
Power (146)	Dominant groups in society may suppress subordinate groups via different means (e.g., variable wages, segmented social status), therefore fragmenting groups. These subgroups would then experience greater discrimination and stigma, while the status quo is maintained by the dominant groups having individualistic beliefs. This coupled with social control: the idea that the most powerful individuals have an interest in subordinate groups adopting deviant or socially problematic behaviour which in turn is defined by the powerful, facilitates a 'revolving door' system by which the same individuals pass through	Discussion Paper	N/A

			a multitude of institutions including hospitals, jails, and clinics.		
		Broad Determinants (25,126,133,138)	Other broad factors such as social and commercial determinants of health are the causal factors associated with low SEP which may explain the AHP.	Cohort, Commentary	General Population
Artefact	Downward Drift	Reverse Causation (16,21,25–27,38,98,108,126)	Heavier drinkers are more likely to lose their job or move to deprived areas due to their heavy drinking. The existence of an alcohol problem is the driving force behind low SEP, rather than low SEP having an independent association with increased harm.	Report, Cross-sectional, Cohort, Report, Case-control, Commentary	General Population
	Methodological	Underreporting/Measurement error (27,30,31,41,42,47,110,117,133,139, 149,150,155)	The use of self-report measures allows the opportunity for response bias and memory limitation to impact the results. Measures which rely on binge drinking beyond a threshold instead of individual units is not accurate at capturing differences in the proportions of non-drinkers between SEP groups.	Systematic Review, Meta-analysis, Report, Cohort, Case-control, Cross-sectional, Commentary	Young Adults, Patient, General Population
		Unmeasured factors (149)	Not all confounders are measured. For example, the way cigarette smoke is inhaled, or the type of cigarette could have an impact on harm.	Case-control	Patient
		Study Design (138)	Need to use more longitudinal data when investigating the AHP particularly to account for time dependent effects.	Cohort	General Population
		Underrepresentation (38,41,42,126,139)	The heaviest drinkers in deprived areas are often underrepresented in studies. This is a potential confounder for cross-sectional studies using aggregate data, as once the heaviest drinkers are	Systematic Review, Meta-analysis, Report, Commentary,	N/A

	accounted for higher rates of harm are no longer paradoxical.

Individual

Individual explanations consisted of processes which take place within individuals that could increase their susceptibility to alcohol-related harm. Themes within this domain included biological (n=7), psychological (n=22), and health and wellbeing (n=19) (Table 4.3). Explanations within the individual domain were often not amenable to human intervention (e.g., genetic make-up or a pre-existing physical health condition).

Individual explanations for the AHP were only hypothesised and had not been tested within any causal or correlational analyses. In related areas, one author has used the tension reduction model to explain alcohol consumption (the idea that alcohol is consumed as a coping strategy to achieve tension reduction) (112). There was also some evidence to suggest coping strategies more broadly (19,113) and abstention due to pre-existing health conditions (113,115) differed by SEP. Another paper highlighted that the biological effects of social inequality which leads to higher mortality of lower social classes has been observed in primates (113). However, given the lack of evidence it is unclear whether these explanations contribute to the AHP.

Lifestyle

The lifestyle domain focused on health behaviour of individuals and groups. These were distinct from individual explanations as they involved an element of choice. Themes were risk behaviour (n=51), drinking practices (n=11) and health-consciousness (n=10) (Table 4.3). One paper explicitly referred to theories of social practice (the context, how and why of drinking) when discussing how drinking practices at the group level could contribute to the paradox (128). Another discussed diffusion of innovation theory, the idea that higher SEP groups are faster to adopt new and healthier behaviours (153).

There were several papers (n=21) which investigated the role of risk behaviour in explaining the AHP. One study highlighted higher rates of hazardous behaviour (e.g. creating a public disturbance or physically abusing someone) among the socioeconomically advantaged rather

than the disadvantaged (18). Another study also highlighted that for young adult's risky alcohol consumption and heavy drinking was more prevalent in the employed compared to the unemployed, while alcohol-related problems were greater for the unemployed (150). Otherwise, there was evidence to suggest drinking patterns and clustering of health behaviours may play some role, as several cross-sectional studies highlighted that those of a low SEP tend to engage in heavier drinking patterns and multiple unhealthy behaviours (19,33,35,38,43,109,135). Those testing the causal role of risk behaviour (n=13) found that these factors partially attenuate the AHP but could not fully explain excess harm experienced by lower SEP groups (9,13,16,25,32,41,117,130,138,149). For example, one record linkage study revealed that when adjusting for alcohol consumption, heavy drinking, BMI and smoking, the hazard ratio for the most deprived group compared to the least deprived was 2.71 (95% CI 2.01-3.64) (16). However, two studies did find that controlling for drinking pattern completely accounted for differences in alcohol-related problems in an adult and young adult population (121,151). In contrast, there was no evidence on the impact of drinking practices or the protective effects of health-consciousness.

Contextual

Contextual factors were those in the individual's immediate environment which may contribute to the AHP. Themes included social (n=20), drinking context (n=11) and place (n=18) (Table 4.3).

Although widely discussed, contextual explanations lacked empirical testing. One study using a within and between subjects design, found that when individuals live in neighbourhoods with higher levels of poverty they report 5% more negative alcohol consequences compared to when they lived in a wealthier area (CR = 1.05; 95% CI: 1.00, 1.11; p = 0.045) and those who on average reside in more impoverished areas also report more negative alcohol consequences (CR = 1.27; 95% CI: 1.10, 1.46; p = 0.001) (141). Some studies provided evidence that social factors, (e.g. marital status), provide a protective effect (9,37). However,

the limited evidence on other contextual factors, including the relationship between outlet density, consumption and harm, was mixed (108,114).

Disadvantage

Explanations in the disadvantage domain tended to focus on the lived experience of those in poverty and how different facets of this may contribute to the AHP. Themes included intersectionality (n=8), lifecourse (n=14), material (n=10) and neo-materialist (n=21) (Table 4.3).

Despite repeatedly appearing in the discussion sections of included papers, only a few explanations associated with disadvantage were empirically tested. Adjusting for material and behavioural factors (25) or cumulative behaviours over the lifecourse (153) attenuated the relationship between SEP and harm by 18-31% and 38-77% respectively. There was also evidence that early SEP, disadvantage during adulthood, and negative prenatal factors (e.g. maternal heavy drinking) all increased the risk of developing a comorbid mental health and alcohol use disorder, which was not attenuated when controlling for own adolescent drinking (23).

Upstream

The upstream domain captured explanations at the macro-level which were hypothesised to have effects on alcohol-related harm. Themes included economic (n=11), socio-political (n=7), alcohol policy (n=5), corporate influence (n=1), employment (n=8), power (n=1), and broad determinants (n=4) (Table 4.3). These explanations focused on the structure of society rather than factors associated with belonging to SEP groups. However, the pathways between these societal structures and alcohol-harm were not well explained.

None of the included papers attempted to empirically assess whether structural factors can account for the AHP. There was evidence to suggest that economic stressors are more closely associated with mortality in the lowest SEP groups (112,120). There is also mixed evidence that negative health effects associated with job loss are concentrated in those already at risk

due to pre-existing alcohol problems (26), and that SEP overlaps with harmful occupational exposures (32). However, the extent to which these contribute to the AHP is unknown.

Artefactual

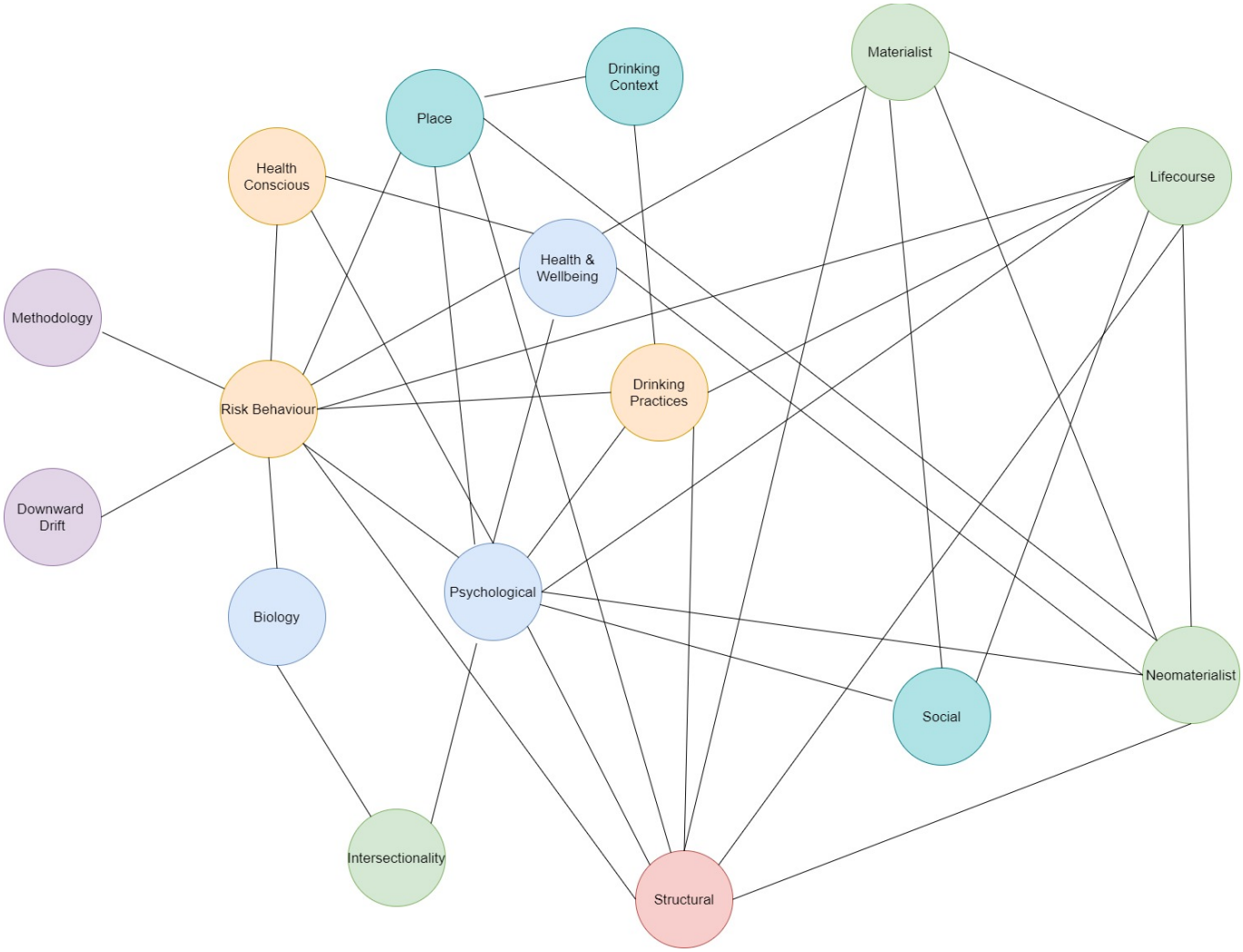
Artefactual explanations claimed the AHP was found due to error. Themes included downward drift (n=9) and methodological (n=16) (Table 4.3).

There was evidence which opposed artefactual explanations for the AHP. Although downward drift was commonly discussed, the only study to test it found it could not account for the AHP (16). Record linkage and longitudinal studies also support the existence of the paradox (9,16,23,25,26,28–31,33,35,37,146,147,153), and therefore diminished concerns of underrepresentation of low-income heavy drinkers in the alcohol consumption data. Another study highlighted that adjusting for alcohol biomarkers only slightly attenuated socioeconomic differences in alcohol mortality (1.0-12.1%), suggesting measurement error is not a likely explanation for the AHP (47). There was a lack of evidence investigating the impact of often unmeasured factors (e.g., type of cigarette).

Relationships between the thematic explanations

The relationships between all themes (colour coded for domain) are shown in Figure 4.2. The connections represent where authors have combined themes within a single explanation. For example, the methodology theme is connected to risk behaviour as one explanation argues that lower SEP groups drink more than they self-report and their heavy consumption leads to greater harm (31).

***Figure 4-2**: Network Diagram illustrating the connections between themes.*



Domain Key: Purple = Artefactual, Orange = Lifestyle, Blue = Individual, Green = Disadvantage, Turquoise = Contextual, Red = Upstream

It is clear risk behaviour is central to explanations for the AHP with the greatest number of connections to other themes (n=10) and links with every other domain (Figure 4.2). This is unsurprising given that health risk behaviours have been the focus of empirical efforts to understand the causes of the AHP.

Other themes, specifically within the upstream and disadvantage domains, were also well connected, possessing connections to four of the five domains. Despite this they lacked empirical testing.

However, some themes: biological, intersectionality, drinking context and those in the artefactual domain, only had one or two connections. This could reflect the characteristics of the explanation, for example one of the methodological explanations suggests that due to the use of self-report measures research has failed to capture accurate levels of alcohol consumption for low SEP groups, they consume more than they report. Alternatively, the lack of connectivity could reflect value, in terms of what researchers think are important explanations for the paradox.

4.6 Discussion

This review examined explanations for the AHP to identify potential pathways and mechanisms which result in differential risk of harm between SEP groups. This is a new approach and goes beyond previous systematic reviews and meta-analyses which have so far established the existence of the AHP and the contribution of alcohol to this relationship (13,41). We identified 16 themes within six domains used to explain the AHP. Risk behaviours were the most prevalent explanations. This finding, paired with the dominance of the behavioural paradigm in empirical work, suggests there has been a reliance on using risk behaviour to understand the AHP. Evidence found in this review opposed the idea that the AHP was artefact. There were many other, mainly hypothetical, explanations for the AHP proposed in the literature. This included individual-level mechanisms (e.g., biological, or psychological), contextual factors (e.g., place-based factors), the lived experience of disadvantage, and upstream structural factors (e.g., the economy and politics). In part this reflects an awareness that the AHP is complex; there is no simple explanation, and researchers do not view causes in isolation. However, it remains unclear why other reoccurring explanations (e.g., social support, or access to health care) have been neglected while researchers frequently return to risk behaviours. This is particularly puzzling given that quantitative evidence suggests risk behaviours only play a partial role (16,117).

There are two potential reasons for this: theoretical and methodological. The study of the AHP is rooted in alcohol epidemiology, which singularly focuses on the causes and effects of

alcohol consumption (156). More broadly the field of epidemiology has faced criticism regarding its approach to understand population health. One of the earliest critiques by Krieger points to fundamental errors in developing epidemiological methods rather than theory, with greater weight given to proximal risk factors, and a focus on causes without context (157). These limitations have led to an emphasis on individual disease susceptibility and individual-level interventions. Instead Krieger argues that eco-social theory (the idea that biology and biological changes are shaped by the social environment) should be used to understand health (157). Concerns regarding how causation is viewed in epidemiology have persisted in contemporary public health, with similar criticisms raised more recently (158). These concerns continue despite efforts to raise the profile of theories such as eco-social theory, and calls to adopt pluralist approaches to causality in epidemiology, which stipulate that causation is not a single connection between two things but the context in which you observe a causal relationship plays a role (158). Adopting such an approach would change the way alcohol researchers conceptualise and investigate the AHP.

The lack of clear theoretical structuring in epidemiology, which is argued to have led to a focus on proximal risk factors (e.g., risk behaviours), could also be a symptom of a lack of methods to carry out more complex analyses of distal factors. Possible solutions to this include the use of complex system modelling methods, which have gained traction within public health and are now being implemented in a UK based project to gain insight into the causal relationships between policy and health-related outcomes (159). Software architecture has also recently been devised to address how theory can be systematically incorporated into individual-level and agent-based computer simulations to understand health and health behaviours (160). Applying these computer simulation methods to the AHP could provide the opportunity to shift the empirical focus from risk behaviours to wider determinants as they can capture complexity and are mechanism based rather than focused on testing relationships between variables.

Strengths and limitations

This is the first review to catalogue explanations provided for the AHP across a breadth of literature. In taking a broad approach to literature searching and inclusion criteria it was possible to review work from multiple disciplines employing varied methodologies. This led to the identification of a varied set of explanations. However, it is possible that some explanations are more appropriate depending on the study design, population, and measure of harm. As the primary aim of this review was to collate and review explanations more generally, we did not conduct an in-depth exploration of this issue. However, upon examination there was no evidence that study design or population influenced which explanations were presented. In terms of measures we found one clear example of an explanation only applicable when using a subjective measure of alcohol harm - those in low SEP groups who drink may feel their outcomes are worse because their peers are more likely to be abstainers (19). This issue awaits further examination.

This review was restricted to high income countries. The results and conclusions are therefore only applicable to this context. Furthermore, most papers focused on the UK, which may limit generalisability. This was justified given substantial differences in alcohol environments. However, given that alcohol is a global issue (98), future research should gain insight into how alcohol affects the disadvantaged in low-middle income countries to help address the deepening of local and global health inequalities.

Another limitation is that only one reviewer screened and extracted data from the papers. We recruited an independent researcher to re-assess a sample of papers for inclusion and extraction. Cross-checking between the two reviewers demonstrated good reliability.

Research & Policy Implications

The lack of explicit theory used to present explanations is a barrier to understanding the causes of the AHP. The development or application of theory may be fundamental to identify the true causal mechanisms which create and sustain the AHP. Several explanations have

been proposed which align with the vast literature detailing theories of health inequality more generally. Eco-social theory, amongst those more commonly discussed (e.g., the materialist (the link between wealth and resources, and health) or political economy theory (the idea that risk factors for health inequalities are rooted in structures)) (63), are just some examples of health inequality theory which could be applied to understand the AHP.

The AHP is well-evidenced and behavioural-related explanations play a partial role. However, these explanations fall short in understanding the complex causes of inequalities in alcohol-related harm. There is a current lack of evidence investigating other explanations found in this review which makes it difficult to suggest potential interventions to mitigate the AHP. Future research should empirically investigate these alternative explanations for the AHP. Computer simulation models offer one potential way of achieving this aim in the short-term and for relatively low-cost.

Based on the evidence from this review the key policy implication is that tackling drinking alone will not reduce inequalities in alcohol-related harm. While there is some evidence that improving multiple health behaviours may attenuate the risk of alcohol-related harm, it is critical that policy makers look to policies outside the scope of public health to mitigate the inequality produced by the paradox.

4.7 Conclusions

There are many proposed explanations for the AHP, however efforts thus far have revolved around risk behaviours as the main cause. Other potentially promising explanations associated within the individual, contextual, disadvantage and upstream domains have remained hypothetical and understudied. Implementation of health inequality theory and complex modelling techniques could provide the opportunity to explore the role of wider determinants in creating and sustaining the AHP.

Chapter 5 Beyond Behaviour: How Health Inequality Theory Can Enhance Our Understanding of the 'Alcohol-Harm Paradox'.

5.1 Chapter Overview

This chapter presents a theoretical review paper which draws on the previously presented systematic review while exploring how health inequality theory could be used to frame the causes of the AHP and providing suggestions for future empirical work.

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5.2 Abstract

There are large socioeconomic inequalities in alcohol-related harm. The alcohol harm paradox (AHP) is the consistent finding that lower socioeconomic groups consume the same or less as higher socioeconomic groups yet experience greater rates of harm. To date, alcohol researchers have predominantly taken an individualised behavioural approach to understand the AHP. This paper calls for a new approach which draws on theories of health inequality, specifically the social determinants of health, fundamental cause theory, political economy of health and eco-social model. These theories consist of several interwoven causal mechanisms including genetic inheritance, the role of social networks, the unequal availability of wealth and other resources, the psychosocial experience of lower socioeconomic position, and the accumulation of these experiences over time. To date, research exploring the causes of the AHP has often lacked clear theoretical underpinning. Drawing on these theoretical approaches in alcohol research would not only address this gap but would result in a structured effort to identify the causes of the AHP. Given the present lack of clear evidence in favour of any specific theory, it is difficult to conclude whether one theory should take primacy in future research efforts. However, it is clear that drawing on any of these theories would shift how we think about the causes of the paradox from health behaviour in isolation, to the wider context of complex interacting mechanisms between individuals and their environment. Meanwhile, computer simulations have the potential to test the competing theoretical perspectives, both in the abstract and empirically via synthesis of the disparate existing evidence base. Overall, making greater use of existing theoretical frameworks in alcohol epidemiology would offer novel insights into the AHP and generate knowledge of how to intervene to mitigate inequalities in alcohol-related harm.

Keywords: Alcohol; Alcohol-related harm; Socioeconomic position; Health Inequality, Social Determinants

5.3 Introduction

Systematic socioeconomic inequalities in health persist and continue to widen across the globe, including in countries ranked highly on indices of economic prosperity and human development (161,162). Alcohol-related health outcomes are not only an example of health inequality but also contribute to inequalities in both life expectancy and death age between socioeconomic groups (49). There is a large body of evidence to suggest that although those of lower socioeconomic position (SEP) tend to drink the same or less on average as those in higher SEPs, they still experience greater rates of alcohol-related harm (41). One record linkage study found that despite controlling for alcohol consumption and other risk behaviours, the most deprived group still maintain a three-fold higher risk of alcohol-related harm (16). This phenomenon, termed the alcohol-harm paradox (AHP), treats alcohol use as a risk factor for health-related harm, although when alcohol use crosses into alcohol dependence this social/health state of dependence that arises is viewed as a harm outcome (163). The AHP is found consistently across several outcomes, including alcohol dependence (22), alcohol-related morbidity (13) and mortality (41). Yet, the causal mechanisms remain unclear.

Despite socioeconomic inequalities in alcohol-related health outcomes, health behaviour has been central to research investigating the AHP (164). This reflects wider public health trends, as for decades epidemiological research has been criticised for its emphasis on using individual-level proximal risk factors to predict population-level health (157,158). Arguably this has led to the most affluent reaping the health benefits due to their increased access and uptake of behaviour change interventions (162).

Cross-sectional research has demonstrated that low SEP groups tend to drink on fewer occasions but drink more heavily per occasion compared to high SEP groups (38,43). They are also more likely to engage in multiple health-risk behaviours (e.g., smoking, poor diet) (38). However, these studies do not measure harm outcomes.

Conversely two record-linkage studies found that behavioural factors, including drinking pattern, smoker status and BMI, could not fully explain the paradox (16,117). These factors

attenuated inequalities, but low SEP groups still had a persistently higher risk of alcohol-related harm [ibid.]. These findings were confirmed by a recent meta-analysis, which found that quantity of alcohol consumed and drinking patterns could not explain socioeconomic inequalities in the relative risk of both all-cause and alcohol-attributable mortality (41). This suggests health behaviours are unable to fully explain the AHP.

While empirical research on the AHP has been limited in exploring other factors associated with socioeconomic circumstances (164), there is an increasing appetite to draw on explanations used to understand health inequalities. A report summarising the AHP discusses access to healthcare and material resources as potential explanations (42). However, at present, there is a lack of theoretical structure to research investigating the AHP. Our understanding of the causes of the paradox remains stagnant due to a continual focus on individual behaviour. This is reflected in recent calls for exploration of contextual factors (e.g., characteristics of drinking environment) and how they not only influence health behaviour but may also directly impact harm (110).

The aim of this paper is to address this gap by identifying alternative approaches rooted in health inequality theory which could be used to design future research into AHP. To achieve this, we review theories of health inequality and their potential to understand the causes of–and therefore potential solutions to–the AHP. We do not aim to synthesise these theories or recommend any one theory. In the context of the AHP, drawing on any of these approaches would be a novel way to conceptualise the problem or inform research design. In Section 2 we introduce prominent theories including the social determinants of health (SDH), fundamental cause theory (FCT), the political economy approach and the eco-social model and discuss the extent to which these approaches are present in the existing AHP literature. We do so by explicitly drawing on a recent systematic review which presents an overview of the explanations for the AHP (164). We then examine how these theories could be used to explicitly frame research on the AHP. In section 3 we discuss the potential use of computer

simulations to assess their explanatory value. In section 4 we discuss what adopting a health inequality lens could mean for the wider alcohol-harm research agenda.

5.4 Drawing on theories of health inequality to understand the AHP

Since the publication of the UK Black Report on Inequalities in Health (65), several theories have been developed which seek to explain how SEP drives health outcomes. Most have a common focus: to shift attention away from the individual-level and behavioural factors, and instead take a multi-level approach. In this section we outline four main theoretical approaches: the SDH, FCT, the political economy approach and the eco-social model (see Table 5.1 for descriptions of each theory), and referring to a recent review (164), discuss how these approaches fit with explanations for the AHP used within academic literature. The review highlighted which explanations had remained hypothetical, and which were present in the empirical research. We aim to highlight how explicitly drawing on theories of health inequality could support research aiming to identify the causal mechanisms that drive the AHP.

Table 5-1: *Health Inequality Theories with Descriptions.*

Theory	Description
Social Determinants of Health	Contains four sub-theories (culture-behaviour, materialist, psychosocial and lifecourse). The social determinants of health specify the interacting role of factors from the narrowest sphere (e.g., individual biological mechanisms) to the broadest (e.g., the structure of society) (64). These determinants can be distinguished into upstream factors (e.g., socioeconomic structure of society), and downstream factors, (e.g., individual factors, health policy and healthcare) (165,166). The structures in society not only impact health directly but also indirectly by creating mechanisms (or SDH), which are then distributed to reflect the socioeconomic stratification of society (167).
Fundamental Cause Theory	Central to FCT are resources defined as money, knowledge, power, prestige, and social connections. It is proposed that high SEP groups have increased access to these flexible resources, and can employ them to avoid risks, reduce the consequences of disease and uptake available treatment to improve health. Conversely these resources are not readily available to low SEP groups. FCT opposes individualistic beliefs,

	emphasising that health cannot be individually controlled and is to some extent the responsibility of the state (96).
Political Economy of Health	The political economy account draws on the idea that cultural-behavioural, material, and psychosocial explanations are rooted in structures (e.g., politics, the economy, work, and labour markets) (63,168). It is the wider macro-economic and political context that determines the distribution of the SDH, population health and inequalities (169,170). This often occurs through public policy decision making, which is impacted by the corporate and business sector, labour, civil society, and political attitudes (e.g., individualistic versus environmentally or socially focused) (63).
Eco-social Model	The eco-social approach developed by Krieger is a multi-level theory which seeks to "develop analysis of current and changing population patterns of health, disease and well-being in relation to each level of biological, ecological and social organization" (83). Key to this theory is the idea that biology and biological changes are determined by the social environment (157). For example, alleged racial differences in biology (e.g., kidney function, blood pressure) posited by biomedical research are instead seen as the modifiable and embodied biological result of occupational and residential racial segregation (83).

The Social Determinants of Health: Current evidence and future directions

The SDH refers to the social and economic factors that shape health at the individual and population level (92,171). This approach originated from the Rainbow Model (90), was refined by the WHO in the 2000s (64) and continues to be central to public health research. It attempts to shift the focus from individual-level behaviours as the cause of health inequality to social determinants which themselves determine not only health but also behaviour (172). Drawing on this theoretical approach to understand the AHP could be the first step to shift from an individual approach.

Within the SDH theoretical approach there are four underlying - interrelated - explanations: culture-behaviour, materialist, psychosocial and lifecourse (62).

Culture-Behaviour. Norms and cultural practices associated with socioeconomic groups have been hypothesised to impact alcohol-related harm. There has been discussion, but no formal hypothesis testing, for how normative differences in drinking patterns between socioeconomic groups might contribute to AHP (35,126). Culture and norms may also influence help-seeking and engagement with preventative healthcare services (139). There is further scope to examine occasion-level risk factors, such as drinking contexts and their association with acute alcohol harm (173).

Materialist. The materialist approach is not present in empirical work investigating the AHP. Researchers have hypothesised that some of the mechanisms associated with materialism lead to socioeconomic inequalities in alcohol-related harm without explicitly drawing on theory. This includes individual material deprivation (e.g., housing and employment), which results in individuals having worse health and a lack of resources to protect themselves from a problem or stressful life event (42,121,144). Additionally, place-based materialist mechanisms, such as a lack of environmental resources (e.g., treatment facilities and preventative services), alcohol outlet density and barriers to accessing healthcare have all been hypothesised contribute to the AHP (2,108).

When providing materialist explanations for the AHP researchers tend to focus on the mechanisms which impact the most deprived in society, without considering the material advantages available to wealthier socioeconomic groups. Additionally, material explanations are typically discussed in isolation in the AHP literature, meaning the link between materialist explanations and societal structures (e.g., the welfare state and benefits system) is missing from the current narrative.

Historically, the contribution of individual material factors (car ownership) to health inequalities has been well evidenced (174–176). Subsequent research also includes resources available in the environment (e.g., access to destinations, transportation systems) (177). Applying these measures to identify material differences within and between socioeconomic groups could reveal the contribution of material mechanisms to the AHP.

Psychosocial. The psychosocial approach has yet to be used in research investigating the AHP. Stress-related mechanisms are hypothesised to play a role, particularly lower socioeconomic groups experience a greater number of stressful life events, negative stereotyping, stigma, and social isolation (22,37,144,146). The lack of social relationships is purported to lead to maladaptive coping strategies, consuming alcohol to cope and a reduced resilience to future negative events (113,125). Conversely, it is acknowledged that affluent individuals have a beneficial network of social connections and therefore a greater social 'buffer' against stressful life events (2,98). While these hypothesised mechanisms touch on components of the psychosocial approach, the role of social comparison (when lower socioeconomic groups compare themselves with others) and discussion of the biological consequences, both central to the psychosocial approach, are missing from the current AHP literature.

Explicitly using the psychosocial approach would reframe the discussion of psychological and social mechanisms to consider how people feel compared to others and the psychological and biological consequences of those feelings which may contribute to the inequalities expressed within the AHP. This concept of relative deprivation is particularly important given the presence of the AHP in high income social welfare state countries (164) where social inequality persists. There is a vast literature on psychosocial pathways which have been shown to contribute to health inequalities more generally (178), particularly in the form of social capital (capturing both social buffer and potential negative effects of social inequality and exclusion). Future work aiming to understand the AHP could usefully refer to the measures of social capital used in existing studies.

Lifecourse. The life-course explanation integrates aspects of several other explanations, allowing different causal mechanisms and processes to explain socioeconomic health inequalities. Risk factors associated with other SDH explanations have been situated in time by some researchers investigating the AHP. This work shows promise, with one study finding cumulative behaviours (those that persist over time) attenuate the link between SEP and all-

cause mortality by 38-77% compared to adjusting for proximal behaviours which attenuated the link by only 24-55% (153). Some literature on the AHP discusses the impact of experiencing material disadvantage at critical time periods (e.g., childhood) and accumulation of negative events, as having lasted negative health effects (9,23,29).

The life-course perspective has been adopted in research using event history analysis and retrospective data, for example in a study investigating the role of cultural capital and cultural health capital during childhood in the uptake of mammography in later life (179). There is a lack of application of these methods in the context of alcohol-harm, with only one similar example identified in the review which investigated factors associated with the development of a comorbid alcohol and mental health condition (23).

The overall SDH approach is however subject to criticism. It has been argued that those who adopt it remain focused on the intermediary causes of health inequalities despite the consensus that it is the macro-level structures that result in health inequality (95). These macro-level structures are viewed as outside of individual control and have become 'causes of causes' obscured by more proximate factors (e.g., health behaviour). This has resulted in theoretical and empirical research dedicated to describing the mechanisms that link socioeconomic inequalities to health, as opposed to identifying the source of socioeconomic inequality (95). One theory developed to address this gap is FCT (96,180).

Fundamental Cause Theory: Current evidence and future directions

FCT shifts the focus from individual-level causes of health inequalities, to looking at the context; what puts people "at risk of risks" (96). This means acknowledging that risk factors (e.g., alcohol consumption) are generated by social conditions, specifically the socioeconomic stratification of society. Crucially this theory does not deny the role of social determinants but suggests that base mechanisms associated with SEP determine whether individuals have the ability to adapt to the introduction of new disease, risks or treatment (180). Proponents of this theory highlight that SEP should be viewed as the fundamental cause of health inequality and

any downstream risk factors rooted within it (97). From this perspective, neglecting the social conditions which generate risk factors has slowed progress in reducing health inequalities.

FCT is not apparent in research investigating the AHP. Using this theory to frame the mechanisms underlying the paradox requires a focus on the societal structures which generate social inequality. Viewing SEP as a fundamental cause of health inequality requires the understanding that disparities are generated through multiple intervening risk-factor mechanisms which alter over time (96). Key to this is the role of resources (money, knowledge, power, prestige, and access to social connections), closely linked to the materialist approach (96,97). FCT asserts that health inequalities will remain despite societal and healthcare changes so long as the socioeconomic structure giving access to resources remains stable (97). Drawing on this perspective to understand the AHP would require acknowledging the existence of this structure and treating SEP as a meta-mechanism responsible for access to resources which could mitigate the effects of other factors associated with the SDH.

A comparative case-study based on FCT predicted that as lung cancer became more preventable, due to knowledge of the link between smoking and the disease, those with greater access to resources disproportionately benefited, thus health inequalities increased (181). Contrastingly, for a disease lacking in major prevention or treatment innovation (e.g., pancreatic cancer), there was found to be no mortality advantage associated with socioeconomic group and this trend was consistent across time [ibid.]. Alcohol-related harms (e.g., liver disease), are largely preventable. Trend analysis could test the role of FCT and investigate whether the introduction of prevention or treatment measures over time has resulted in socioeconomic inequalities in alcohol-related harm.

The Political Economy of Health: Current evidence and future directions

Sitting between the SDH and FCT is the political economy of health approach. The political economy explanation is an attempt to acknowledge the role of upstream factors in generating and distributing risk factors. It argues that the social - and behavioural - determinants of health

are themselves shaped by structural determinants: politics, the economy, the (welfare) state, political institutions, the organisation of work, and the structure of the labour market (182–184) and that population health is shaped by the "social, political and economic structures and relations" that may be, and often are, outside the control of the individuals they affect (83,169).

Structural influences within the political economy approach have only been tenuously linked to the AHP. The economic and socio-political conditions, alcohol policy, corporate influence, employment, and power relations are provided as potential explanations for the AHP (18,116,120,146), however authors do not clearly articulate the underlying mechanisms. They touch on the commercial determinants of health as key drivers of alcohol-related harm which aligns with recent calls to acknowledge the detrimental role of the private sector on both the environment and health behaviour which in turn determines health (185). The political economy perspective clearly defines the role of these structures as influencing the distribution of the other SDH. Drawing on a synthesis of these perspectives in the context of alcohol-related harm would highlight these mechanisms. For example, social and political attitudes of residents and decision makers influence the investment of public services in deprived areas which then determines the availability of services (21), a materialist determinant of health.

Studies investigating the role of political economy in the generation of health inequalities typically take a cross-national comparative approach. This involves comparing different economic and political systems to understand how these systems contribute to health inequalities, both within and between countries (186). This approach to research provides the opportunity to identify how the structure of the labour market, employment and welfare systems can prevent or increase health inequalities [ibid.]. There is a current lack of cross-national comparisons in the existing AHP literature.

The Eco-social Model: Current evidence and future directions

A recent commentary by Bloomfield has called for future research investigating the AHP to draw on the eco-social approach, acknowledging that inequalities in alcohol-related harms

cannot be explained by drinking patterns alone (110). The main distinguishing feature of the eco-social approach is the emphasis it places on biological and ecological analysis (83).

Biological mechanisms have been hypothesised to contribute to the AHP. Primarily these have been related to health behaviours and genetic alterations due to the experience of disadvantage (35,38). For example, engaging in certain patterns of behaviour (e.g., multiple unhealthy behaviours or drinking with meals) has metabolic effects which compound or protect against the effects of alcohol consumption (35). Biological alteration related to the experience of disadvantage or differences based on ethnicity were also more vaguely linked to the AHP (113).

Explicitly using the eco-social approach would shift the focus to how individuals biologically embody their social conditions. Achieving this in empirical research requires access to biological and social data. A recent paper which analysed data from several cohort studies investigated the relationship between social disparity and biology, finding evidence of biological changes in response to the environment (187). There may be opportunities for alcohol researchers to engage in collaborative projects or gain access to data sets, for example the UK Biobank (188), which would allow the opportunity to investigate the eco-social model in the context of alcohol harm.

5.5 Computer simulations can test the explanatory value of mechanisms specified in health inequality theory

Explicitly drawing on existing theories of health inequality may address the gap in identifying and extracting relevant variables and relationships in the pursuit to understand the AHP. However, the methods best placed to test these causal relationships requires further scrutiny.

To study these complex relationships which exist on a multi-level plane (e.g., individual, community and structural levels) and are dynamic in nature, suitable research methods are required. The “risk factor” approach to epidemiology explores decontextualized and independent relationships between dependent and independent variables and uses linear

reductionist models to test these relationships (189). To capture the features of complexity a mechanism-based approach is required which explicates the details of how regularities are brought about rather than focusing on statistical regularities between variables (53). Mechanisms consist of “entities” and the “activities” entities engage in, either as a collective or independently, to bring about a particular outcome [ibid.]. Computer simulation methods are a good candidate to test mechanisms, and complex system models have become increasingly attractive in public health research (190).

A review of the use of simulation models in the context of health inequality concluded that they enhance our understanding of socioeconomic health inequalities (191). Specifically, the class of techniques known as agent-based modelling (ABM) can flexibly model the multilevel, reciprocal, and indirect effects of socioeconomic inequalities [ibid.]. ABMs are computer simulations comprised of agents (e.g., individuals or households) and their interactions within the context of their environment (192). ABMs provide the opportunity to test mechanisms specified in theory (193). This ranges from abstract theory testing to more concrete applications which draw on empirical data to inform the properties and environments of agents (194). Like other types of simulation model, ABMs enable otherwise fragmented evidence to be synthesised in order to address research questions and inform decision making (195).

One example of an ABM implemented to understand socioeconomic health inequalities explored the role of bounded rational choice mechanisms (individual level) and spatial segregation (structural level) in the emergence of income gradients in healthy eating (196). This model represented both food stores and households as having agency—over decisions to supply and purchase, respectively, healthy, or unhealthy foods. The model equations define the mutual interactions between stores and households, enabling feedback loops to be represented. The model findings suggest that differences in diet between socioeconomic groups arise only when high income household and healthy stores are both spatially segregated from low income households and unhealthy stores. Once established, these diet

inequalities could only be overcome when both groups had favourable preferences for healthy foods and when healthy food was relatively cheap [ibid].

A similar approach could be taken to investigate the mechanisms specified in health inequality theory. Here, we sketch such a model. In psychosocial theory, one mechanism proposed to result in socioeconomic differentials in health is that high SEP groups have a protective social buffer (2,98). Hypothetically this mechanism could be represented in an ABM by simulating individuals as agents and defining a macro-level social network structure with connections based on agent attribute similarity (e.g., age, gender). Agents would possess the capability to give or receive support in the presence of a stressful event. However, this capability would be contingent on their own resources (e.g., income), type of support available to them (e.g., emotional support) and their own stress burden. Individuals whom receive support from their network would have a reduced stress burden and therefore reduced risk of harm. The network could also be responsive to changes in relationships (e.g., providing support strengthens ties while refusing support breaks social ties between agents). A simulation such as this would allow *in silico* experimentation with changes in resources, types of support and stress, to determine how these features impact not only individuals but potentially their social network structure.

Recent developments in computer model integration have also demonstrated that ABMs which combine mechanisms from multiple theories can provide an improved explanation for complex phenomena (in terms of parsimony and empirical goodness-of-fit) (160,197). These integration findings are particularly relevant given that theories of health inequality do not necessarily compete, but rather attempt to explain health inequality from different viewpoints.

Computer simulation methods such as ABM have yet to be applied to understand the AHP and would allow us to make best use of the available evidence to test the explanatory value of mechanisms described in existing theories of health inequality. When we extract the mechanisms from these theories and implement them in an ABM simulation, does the simulation generate inequalities in alcohol-related harm?

5.6 Discussion

It is clear research investigating the AHP eschews the use of theory. Many of the mechanisms specified in health inequality theory are touched on as hypothetical explanations for the paradox, mainly on an ad-hoc basis and in the absence of clear theoretical structure. Structure would be provided by drawing on any of these theories explicitly. In the one instance where one of the theories was present in the empirical work on the AHP this showed promise, as cumulative behaviours across the lifecourse could explain a greater proportion of harm experienced by lower socioeconomic groups (153). There is a lack of evidence which makes it difficult to conclude whether one theory over another can best explain the AHP, especially as these theories do not necessarily compete but examine causes of health inequality at different levels and with differing emphasis on certain factors. One thing is clear, the use of these theories will shift how we think about the causes of the paradox from health behaviour in isolation, to the wider context of complex interacting mechanisms between individuals and their environment.

Framing alcohol research using health inequality has significant implications for the study of the AHP and wider alcohol harm research agenda. In the past, behavioural framings have resulted in empirical work underpinned by individual proximal factors, specifically alcohol consumption and other health behaviours. In section 2 for each theory, we identify research designs implemented in social epidemiology which attempt to understand the causes of health inequality more generally (e.g., new measurements that capture social capital (178), or cross-national comparisons (186)). We can utilise the advances in social epidemiology, for example the introduction environmental resources in the materialist perspective (177), and apply this to the AHP.

Taking a behavioural approach has resulted in the implementation of policies which often rely solely on individuals acting to reduce their alcohol consumption (e.g., educational campaigns), which arguably increase inequalities (42,51). There have been attempts to reduce inequalities by introducing minimum unit pricing in several countries including Scotland, Wales, and

Australia's Northern Territory. In theory this policy reduces the consumption of alcohol particularly for those of a lower SEP, as they typically purchase alcohol at cheaper price points(198). However, the focus of this policy remains on reducing alcohol consumption which will not address the underlying causes of inequality.

Critically, shifting from this focus on alcohol consumption as the fundamental cause of harm in alcohol research requires researchers to acknowledge the causal processes driving harm are complex and that understanding of these processes requires different methodological perspectives drawing on ideas from complexity science (199).

While the focus of this paper has been on the AHP, a well evidenced phenomenon, it is possible that a harm paradox could exist for other health behaviours. Hypothetically at the same number of cigarettes smoked, those of a lower SEP may experience greater rates of smoking related harm; and there is evidence to support this hypothesis (200). This reflects a slight misnomer – the AHP is not particularly paradoxical if it simply reflects wider causes of health inequalities. This concern further reinforces the need to utilise theories of health inequality to understand the complex interactions between health behaviour, the environment and harm and explore why lower socioeconomic groups are more vulnerable to the negative effects of risk behaviour.

5.7 Conclusions

The existing research on the causes of the AHP lacks theoretical structure and relies heavily on analysing the contribution of health behavioural risk factors. Drawing on health inequality frameworks would result in a more structured effort which gets at the root causes of both alcohol-related harm and alcohol-related health inequalities. Using these multi-level frameworks would allow us to understand the role of other mechanisms, in addition to alcohol consumption, which exist in the wider socioeconomic environment. Simulation methods (e.g., ABMs) allow for the opportunity to explore the complexity captured in health inequality theory meaningfully. Combining these theories with simulation methods has the potential to inform

policy which not only reduces consumption but reduces harm, and in turn health inequalities more broadly.

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Chapter 6 Modelling Principles and Methodology

6.1 Chapter Overview

The previous two chapters presented review papers; the first identified explanations for the AHP in the existing literature and the second considered how the main theoretical directions from a health inequalities perspective could be applied to enhance our understanding of the AHP. The next step in this PhD's programme of research was to identify the causal mechanisms outlined in health inequality theory and, drawing on these mechanisms, develop an agent-based simulation model which tests the explanatory value of a selected theory.

The rest of this chapter outlines the methodological principles that were used to answer the final research question posed by this PhD project: can a quantitative simulation model derived from theory test a candidate explanation for the AHP? First, this chapter outlines the motivation for taking a realist mechanism-based approach, introduces the concept of agent-based modelling (ABM) and then provides an overview of the stepped process taken to test mechanisms using quantitative computer modelling. This chapter also provides a background on meta-models, which are frameworks used to redescribe theory and facilitate the development of agent-based models (ABMs), and introduces the mechanism-based social systems modelling (MBSSM) framework which was applied to extract mechanisms from theory (for more details see Chapter 7). Finally, the chapter introduces the microsimulation and calibration architecture that was used in this PhD.

6.2 A Realist Mechanism-based Approach

It is clear from the previous review chapters that the AHP is an example of health inequality. Research methods that can capture the multi-level and dynamic nature of explanations for health inequalities are required to appropriately examine the relationships that may drive inequalities in alcohol harm. Specifically, a realist mechanism-based approach and associated methodologies have the potential to provide explanatory insight into what is driving the AHP.

A realist mechanism-based approach is founded in critical realism, which among its key features focuses on understanding and explaining mechanisms (201,202). Mechanisms consist of entities, their properties, and the activities entities engage in either as a collective or independently, to bring about a particular outcome (53). The entities that give rise to mechanisms are not deterministic, rather they constrain or enable certain events (203,204). Therefore, critical realism focuses on understanding and explaining the underlying mechanisms that cause the events we may observe (201).

A realist mechanism-based approach explicates the details of how regularities with the potential to be observed in data (e.g., the AHP) are brought about as opposed to focusing on testing statistical associations between variables (53). Quantitative computer simulation methods are an ideal candidate for identifying and testing which mechanisms can explain particular regularities and are becoming increasingly used to understand public health problems such as socioeconomic inequalities in health (191). Specifically, ABMs have been identified as having the ability to flexibly model the multilevel, reciprocal, and indirect effects of socioeconomic inequalities (191) and are particularly well suited to test mechanism-based explanations for these inequalities that could inform policy appraisal and evaluation (205). Given its suitability to test mechanisms and capture key features associated with socioeconomic inequalities, ABM was selected for use in this PhD.

6.3 Agent-Based Modelling

Agent-based models (ABMs) are computational models that simulate autonomous agents (e.g., individuals, households) situated in space and time, who may interact with other agents and their environment (193,206). The interplay of individual differences, the interactions between agents, and agents' interactions with their environment allows us to identify causal mechanisms which generate emergent social phenomenon (192). The researcher assigns agent attributes (e.g., age, sex), and implements the rules which govern actions and interactions (207). ABMs can have two system levels, the micro or "agent-level" which consists of agents and often the collective actions of agents, and the macro-level which consists of the

broader social and structural entities (160). There are several key features of ABMs: the agents themselves, the rules, behaviours and relationships, and the environment.

6.3.1 Agents

Although there is not a universally agreed definition of an 'agent', agents tend to represent the entities that exist in the real-world system of interest (207). In the context of this PhD the micro-level agents of interest are people, as the project is interested in understanding the causes of the AHP. However, the macro-level agents of interest (if any) will depend on the theory selected for testing. While the type of agent varies based on the research question of interest, there are several features common to most agents which are described by Crooks & Heppenstall (207):

- Autonomy: agents are autonomous (they are self-governed), and can exchange and process information to make independent decisions.
- Heterogeneity: there are often differences between agents in terms of their characteristics or attributes. For example, a human agent could have attributes such as age, sex, motives, beliefs etc. Agents can form groups; however, these are "*amalgamations of similar autonomous individuals*".
- Active: agents are active as they engage in actions which independently influence the simulation. A few examples of active features that an agent may possess are: mobility (they can move around the environment in the model), adaptation/learning (they can change their behaviour based on previous states), interactive/communicative (they can communicate or interact with other agents and their environment).

6.3.2 Rules, behaviours, relationships

Agents, as described above, are bound by rules which impact their behaviour and their relationships with other agents and the environment (207). Rules can be determined from simple heuristics or defined based on theory, and are often obtained from the academic literature, expert elicitation, or analysis of data (207). Rules can apply to all agents in the simulation or groups of agents, and are typically implemented as 'if-else' statements in which

agents only carry out the action if a specified condition has been met (207). In recent years there has been an increased interest in drawing on psychological behavioural frameworks (e.g., dual process framework and the belief-desire-intention framework) to more accurately represent human behaviour (208,209).

Agents can share relationships with other agents and their environment which can be specified in several ways. For example, they can be reactive (e.g., agents perform actions based on the actions of other agents) or goal-directed (e.g., agents perform actions to meet a goal) (207). For agent-agent interaction to occur this often requires agents to be connected to one another. Connections between agents can be based on simple rules, for example in the predator-prey model connections are based on spatial proximity (210). Social networks can also be set up to represent more complex connections between agents. For example, in existing ABMs exploring alcohol consumption, a social network connected individuals in the model based on their age, sex, education group, level of deprivation and drinking status (211,212). Within a social network, agents can interact with other agents that they are connected to. Social networks can either be dynamic (connections between agents can be broken or formed over the course of the simulation) or static (connections between agents remain fixed) (213).

6.3.3 The environment

Environments situate agents in a representation of space, which can either be actual physical space, for example using Geographic Information Systems modelling of real-life cities (214), or abstract space. This space provides a context in which agents interact with other agents and the environment itself (207). The model environment can also be a useful tool to visually represent an ABM and facilitates the monitoring and observation of agents and their behaviours.

Given that ABMs are comprised of agents, their attributes and behaviours, and mechanisms consist of entities, their properties, and activities (205), as a method they essentially encode artificial mechanisms and provide the opportunity to test mechanisms specified in theory (193). ABMs are a particularly flexible means of testing mechanisms as developing an ABM is often

an iterative process in which modellers can vary the abstraction of the system of interest. As a result, ABMs can be easily adapted to new information about agents and their behaviour (215).

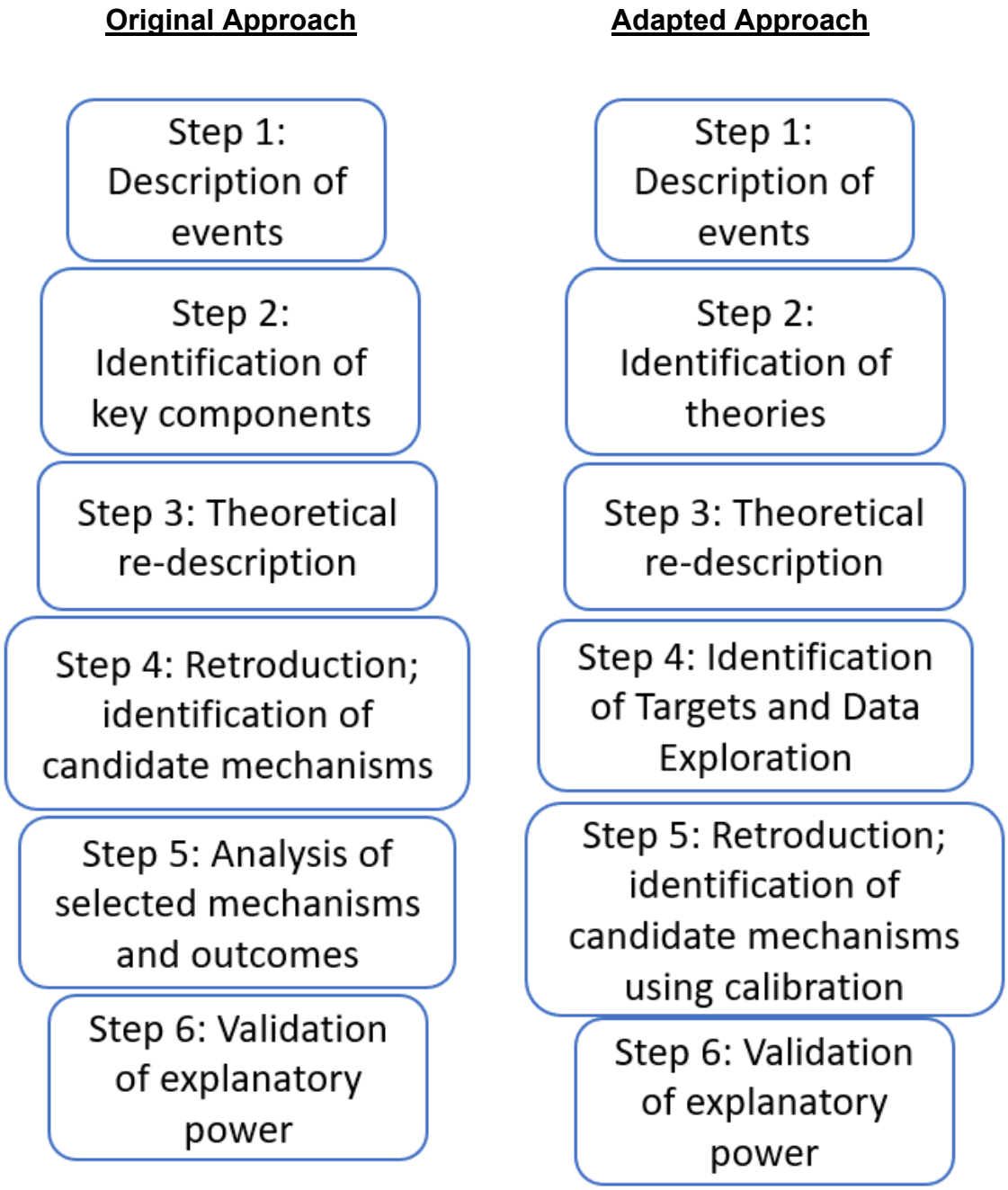
ABMs can also explicitly incorporate the features of complex systems (e.g., feedback loops, non-linearity, and adaptation), which are more difficult to represent using other modelling methods (215,216). In addition to deepening our understanding of complex phenomenon, ABMs can be used to test the impact of policy interventions and inform policy decisions (216).

6.4 A Quantitative Approach to Testing Mechanisms

Drawing on the work of Danermark (217) and Sayer (218,219), Bygstad and Munkvold (220) developed a stepwise framework for critical realist data analysis. For the purposes of this PhD project this six-step framework was adapted to fit within the context of ABM (see Figure 6.1 for the original six step framework and the adapted framework).

Step 2 was adapted to identify theories as opposed to key components of the AHP as a phenomenon in general. This step was added to the process to reflect the PhD's focus on testing the ability of a theory to explain the AHP and replaced the identification of key components step which aims to identify the real objects or entities involved in generating the phenomenon (220). The causes of the AHP are complex and as highlighted in chapters 4 there could be many potential entities involved in the generation of the paradox (e.g., social entities such as norms, structural entities such as the education system) depending on which explanation you choose to focus on. Identifying a set of theories first narrows the range of entities that would normally be identified in step 2. Another adjustment made to the existing six-step process was to add a step that identifies targets and explores the data available which could be used to implement a quantitative ABM, a key step when developing a computer simulation model.

***Figure 6-1**: Flowchart of the six-step approach for critical realist data analysis and the adapted approach for testing mechanisms using ABM.*



The adapted approach also expands on the retroduction step by specifying that candidate mechanisms are identified using a calibration procedure. As the method is a quantitative computer simulation step 5 in the original process (analysis of selected mechanisms and outcomes) was removed and instead consolidated in the retroduction step as model outputs are compared to targets in this stage of the adapted process.

6.4.1 Step One: Description of events

The first step in the process is simply identifying and describing the event, the event being the phenomenon of interest. For this PhD project this is the AHP. The first several chapters of this thesis have described the AHP as the consistent finding that despite consuming less alcohol on average those of a low SEP experience greater rates of alcohol-related harm. These chapters also presented numerous empirical observations of this phenomenon which establish its existence.

6.4.2 Step Two: Identification of theories

The second step of the adapted process involves the identification of theories which could be used to explain the phenomenon of interest. Chapter 5 of this thesis identifies and discusses in detail different health inequality theories in the context of the AHP. Due to time and resource constraints only one theory will be selected for testing. The process and reason for the selection of the candidate theory is discussed in detail in the next chapter (Chapter 7: Translating Theory to ABM using the MBSSM architecture).

6.4.3 Step Three: Theoretical re-description

Redescription is the process of "redescribing" the features and components of a theory into a format appropriate to implement in a simulation model (217,221). In other words, developing a conceptual model that represents the theory or theories of interest. To facilitate the redescription of mechanism-based theories, social theorists have constructed several frameworks, called meta-models, which can be used to identify the entities and behaviours described in theory (160).

Meta-Models

Traditionally, ABMs have been used for bottom-up modelling, whereby agent action and interaction give rise to emergent phenomena at the macro-level (54). A specific example of this is the Coleman Boat meta-model which only allows the micro-level to possess the capacity for action (160,222). This meta-model has been refined and adapted by Hedstrom & Swedberg (223), who suggest that there are two levels: the micro or "*agent level*" which consists of

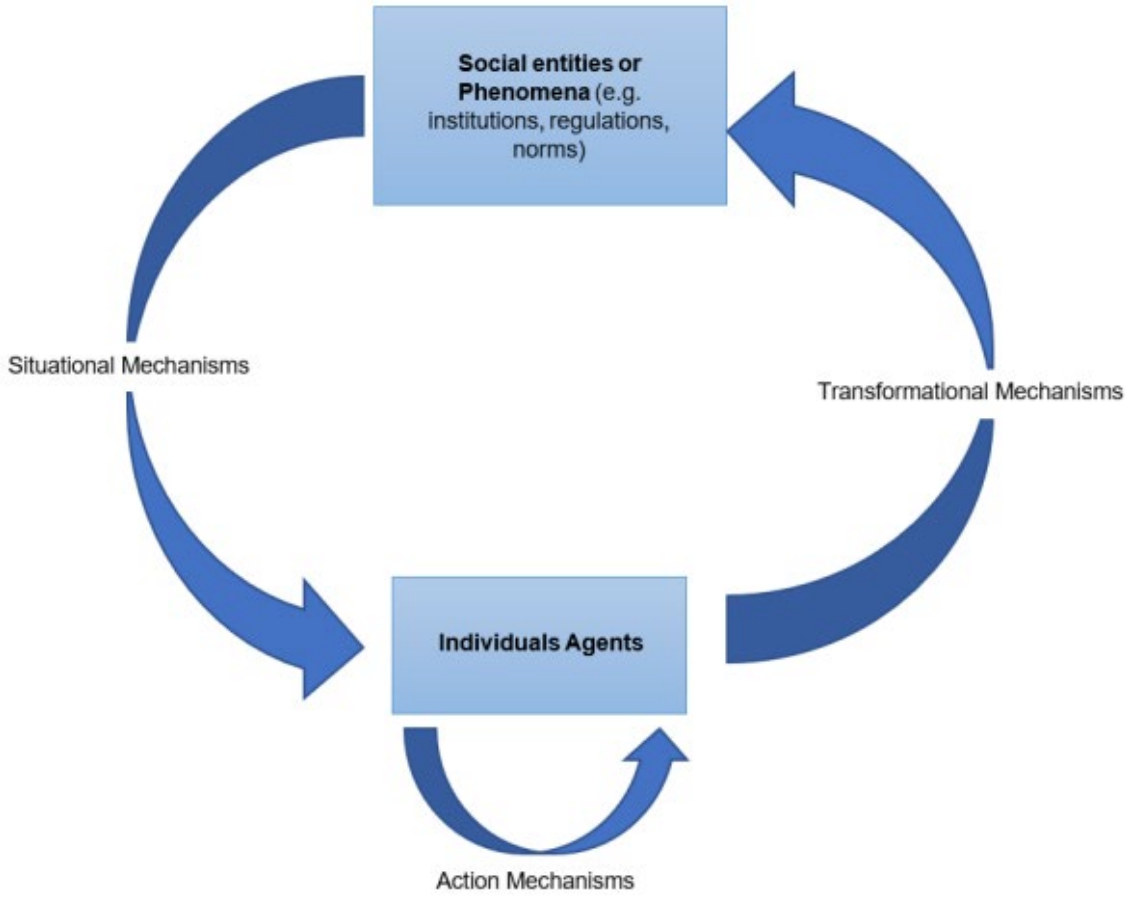
individuals or the collective action of individuals, and the macro level which consists of the broader social and structural entities (e.g., social norms or regulations). These levels then interact via three different forms of mechanism: *situational mechanisms* (macro-micro) the impacts of social structures on individuals' "*internal states*", *action mechanisms* (micro-micro) the impact of internal states on individual agent action/behaviour, and *transformational mechanisms* (micro-macro) how the actions of individuals then shape structural entities. Despite the presence of a macro-level in this meta-model, each mechanism defined by Hedstrom & Swedberg clearly remains focused on the impacts to, and actions of individuals at the micro-level. The Hedstrom & Swedberg meta-model structure has dominated the field of analytical sociology with a strong emphasis on the role of individuals in system dynamics while dampening the role of structures due to a lack of macro-macro mechanisms (160).

It has been argued that ABMs can be used to represent mechanisms not necessarily tied to individual's decision making. One example meta-model developed by Tilly takes a similar structure to the Coleman Boat, but outlines a different set of mechanisms (224). Environmental mechanisms defined as any external influence on individual agents (macro-micro), cognitive mechanisms which determine agent behaviour, and relational mechanisms which "*alter connections among people, groups, and interpersonal networks*" (micro-macro) (160,224). This meta-model diverges from those the Hedstrom & Swedberg meta-model given that relational and environmental mechanisms can impact the structural level "*without any necessary connection to individual-level cognitive mechanisms*" (224). Several other alternative meta-models have been developed including Sawyers five-layer ontology (225) and Archer's social morphogenetic approach (203) but key to these approaches is that structural processes are permitted to impact structures without interference from the individual and that the macro level can also impact agents without a pre-requisite of "*individual-to individual interaction*".

To address the discrepancies between use of meta-models in developing ABMs Vu and colleagues have developed a meta-model, the Mechanism-Based Social Systems Modelling

(MBSSM) architecture (160). In general, Vu et al. adopt a micro-macro level structure and use terminology taken from Hedstrom & Swedberg to name the mechanisms (223) (see figure 6.2). However, they provide several caveats that address the limitations of the Hedstrom & Swedberg meta-model discussed above: 1) that the micro level is not restricted to individuals (it can also be households etc.) and 2) interaction does not only occur between agents at the micro level (160).

Figure 6-2: *The MBSSM Macro-Microstructure adapted from Vu et al., 2020.*[3]



One of the aims of this project was to build and test a computer simulation model that draws on a theory of health inequality to explain the AHP. Therefore, this PhD project applied the

[3] Vu TM, Probst C, Nielsen A, Bai H, Buckley C, Meier PS, Strong M, Brennan A, Purshouse RC. A software architecture for mechanism-based social systems modelling in agent-based simulation models. Journal of artificial societies and social simulation: JASSS. 2020 Jun 30;23(3).

meta-model developed by Vu and colleagues to develop a conceptual representation of the selected theory. In general, conceptual model development in this PhD used the MBSSM architecture to extract both entities and mechanisms identified from theory (160), further details on this process can be found in Chapter 7.

6.4.4 Step Four: Identification of Targets and Data Exploration

The next step in the process of developing a quantitative ABM using a realist mechanism-based approach was to identify appropriate target data for model calibration. Target data is the data the model aims to reproduce (i.e. the targets are the specific cases of the AHP the model aims to explain), and for this PhD the required target data must show a socioeconomic gradient in alcohol harm. Another part of this process is to explore the secondary data available which could be used to implement the ABM. This should be data that contains variables which could be used to represent individuals and the attributes they require as described in the conceptual model. A summary of the data sources used in this PhD can be found in table 6.1 and are described in further detail in chapters 8 and 9.

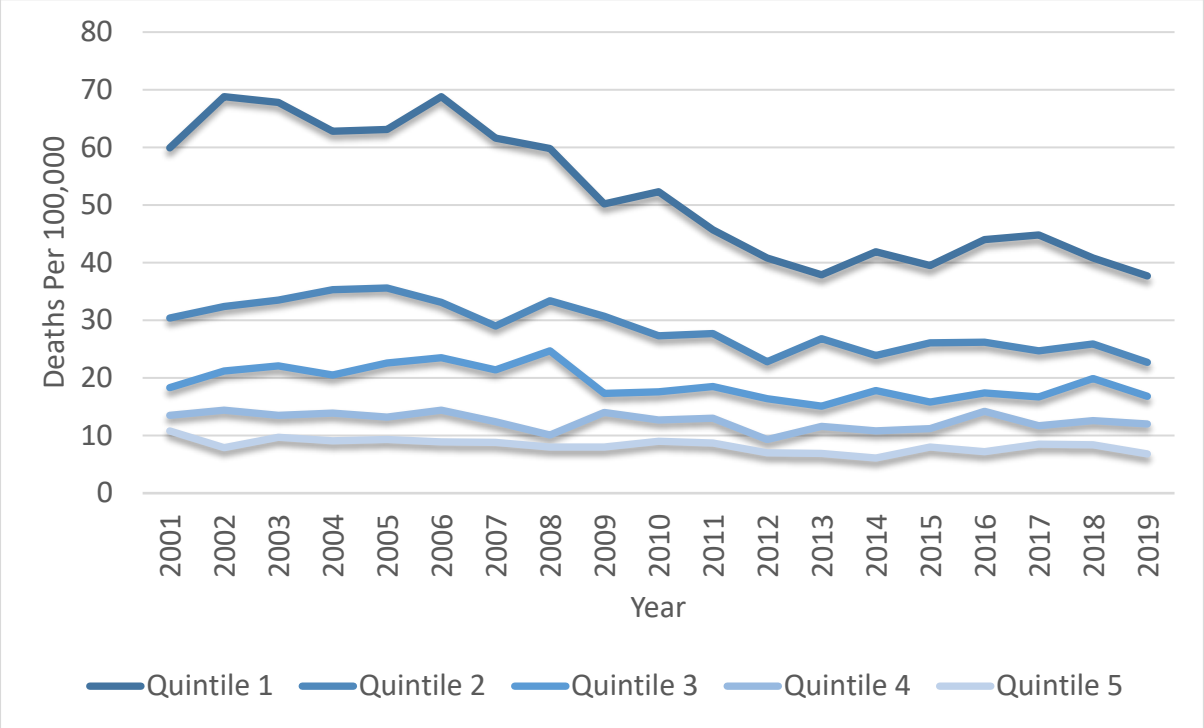
Table 6-1: *Summary of key data sources used in this PhD project.*

		Data Description and Source
Targets		Alcohol-specific death rates for Scotland split by deprivation quintile (SIMD1 – most deprived, and SIMD5 – least deprived). Data requested from the National Records for Scotland https://www.nrscotland.gov.uk/ Email: statisticscustomerservices@nrscotland.gov.uk
Microsimulation		
(i)	Area-level aggregate counts	Counts of individuals living in each DataZone in Scotland. Data obtained from the 2001 Scottish Census: https://www.scotlandscensus.gov.uk/search-the-census#/
(ii)	Attribute rich survey data	A survey with variables available to provide individuals with attributes (e.g., age, sex, income, educational attainment). The British Household Panel Survey was used which was available from the UK Data Service: https://ukdataservice.ac.uk/
(iii)	Alcohol consumption data	Average units of alcohol consumed per week. For years 2001-2006 data was sourced from the General Household Survey (GHS) and for years 2008-2019 data was sourced from the Scottish Health Survey (SHeS). All data obtained via the UK Data Service: https://ukdataservice.ac.uk/

To identify appropriate target and data sources to generate a microsimulation model first requires the selection of a geographical setting for the model. A key consideration when selecting a geographical setting is that the phenomenon of interest, the AHP, can be described to ensure its existence in that context. To describe the AHP, we require evidence from individual-level linked data which possesses a measure or multiple measures of SEP, alcohol consumption and health outcomes, specifically alcohol-specific morbidity and/or mortality. A prominent study by Katikireddi et al., using such linked data has demonstrated that the AHP does exist in Scotland between 1995-2012 (16). Katikireddi et al., show that even after adjusting for alcohol consumption those of a lower SEP had consistently greater risk of alcohol attributable admission or death. The data used in this study was the Scottish Health Survey (SHeS) which contains measures of alcohol consumption linked to hospital and death records. Theoretically, it is possible to acquire target data which empirically describes the AHP for a Scottish population. However, due to budget and time constraints it was not possible to obtain the linkage data for the purposes of this PhD project. This is because the cost to obtain the linked dataset was between £15,000-£30,000 and the data could only be accessed via the National Safe Haven, a secure environment only available to hospitals and universities across Scotland.

As it was not possible to acquire the ideal target data for this project, readily available secondary data for Scotland was identified. Specifically, data detailing the rates of alcohol-specific death split by deprivation quintile was provided by the National Records for Scotland (see Figure 6.3).

Figure 6-3: *Model target data: rates of alcohol-specific death per 100,000 in Scotland split by SIMD quintile, 2001-2019.*

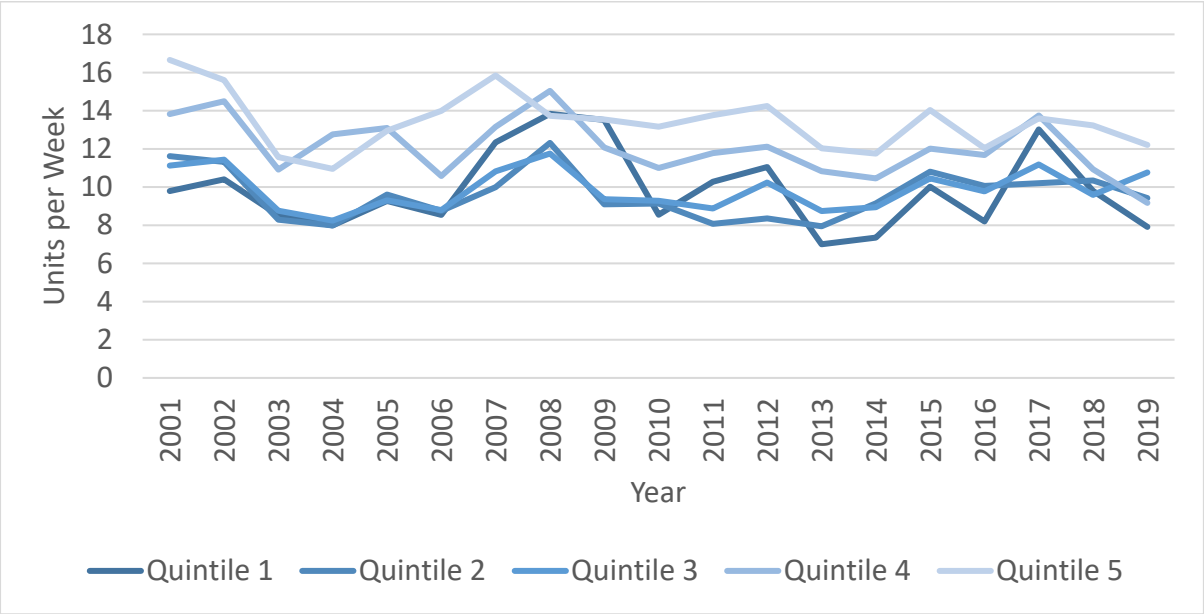


*Note: Quintile 1: Most Deprived, Quintile 5: Least Deprived

The alternative target data displayed in Figure 6.3 does show a consistent socioeconomic gradient in rates of alcohol-specific death in Scotland from 2001-2019, with the most deprived quintile (Quintile 1) experiencing the highest rates of death. A major limitation of using this target data is that it does not provide accompanying data on the alcohol consumption associated with each mortality. To overcome this limitation, we initialized each individual in the model with a value for average units of alcohol consumed per week for each year of the simulation taken from other data sources, specifically the General Household Surveys (GHS) (2001-2006) and Scottish Health Surveys (SHeS) (2008-2019). As highlighted in Figure 6.4 the alcohol consumption trends are what you would expect to observe in the presence of the AHP. The most deprived quintile (quintile 1) has consistently lower or similar consumption levels compared to the least deprived quintile (quintile 5). It should also be noted that from 2001-2019 there has been a decline in alcohol-specific deaths in Scotland which is particularly precipitous from the most deprived quintile. Therefore, there has been a substantial decline in the socioeconomic differences in harms over the period of study as the gap between the most

and least deprived quintiles has decreased. Therefore, the model constructed in this thesis will need to account for a decline in inequalities in alcohol specific death from 2001-2013. While this is not a grave cause for concern, this does impact the generalisability of the model results. The identified parameters used to operationalise mechanisms in the model will be unique to contexts where there is an observed decline in inequalities in alcohol harm. However, given inequalities in alcohol specific death are sustained over the time period in Scotland the modelling exercise will still have the ability to offer insights into the causes of the AHP.

Figure 6-4: *Average Units per week consumed by each deprivation quintile taken from the General Household Survey (GHS) and Scottish Health Survey (SHeS).*



A static microsimulation model was developed to provide a representative simulated – also known as 'synthetic' – Scottish population. Essentially this type of model simulates a system, in this case Scotland, which consists of millions of individuals created using a computer (226). The word 'static' simply refers to the time point, and for this PhD the microsimulation model generated a synthetic population of Scotland for the year 2001. Acquiring a representative sample of Scottish individuals is necessary to test the theory using an empirical ABM as it requires context-specific data on individual agents. A representative sample was obtained

from the microsimulation and was used to initialise the population in the ABM prior to simulation.

To generate a synthetic population from scratch requires two types of data, microdata (the individual level survey data that will inform the attributes of the individuals in the synthetic population (e.g., age, sex, education) and geographical constraint data (the count of individuals in each characteristic category for each spatial zone) (227). An additional benefit of selecting Scotland as the geographical context is that there are several surveys available via the UK data service including the Scottish Health Survey (SHeS) amongst others, which while not linked to hospitalization or mortality records are readily available and can be used to assign attributes to individuals. As the available target data starts in 2001, the baseline synthetic population was derived using the 2001 census as geographical constraint data and the British Household Panel survey collected in 2001 that is available via the UK data service.

The microsimulation model was generated using the Flexible Modelling Framework (FMF) and existing secondary data. The FMF is generic software which uses a static spatial microsimulation model algorithm based on simulated annealing (228). Simulated annealing is a combinatorial algorithm of deterministic and probabilistic methods used to allocate individuals to spatial zones (227). Full details of the method and data used to generate the synthetic population can be found in Chapter 8.

6.4.5 Step Five: Retroduction

Retroduction is the exploration of whether generative mechanisms, which operate under certain conditions, can in fact explain an empirical phenomenon (219). Therefore, if the mechanisms encoded in the ABM developed for this PhD project can reproduce the trends associated with the AHP; a positive deprivation gradient in alcohol-specific mortality, it is possible that they are the causal mechanisms underlying the AHP observed in Scotland and, potentially, elsewhere.

To identify these generative mechanisms using ABM requires model calibration. Regardless of the theory represented by the ABM there are likely to be several parameters which require calibration to the target data to produce accurate estimates. To calibrate the model, parameters will be sampled from prior distributions using Latin hypercube sampling in the *R* 'nlrx' package (229), and implausibility values will be calculated for each model run for every target k (see equation 6.1) (230).

$$I_{[k]=} \frac{(ym[k]-yt\,[k])^2}{(se_m[k]^2)+(se_t[k]^2)}$$

Equation 6-1

Implausibility I is a metric which captures the distance from the model outputs to the target data by calculating the difference between the simulated output ym and target data yt divided by the standard error of the model se_m and the standard error of the empirical target data se_t. Then the overall implausibility was taken to be the average implausibility over the k targets (see equation 6.2).

$$\frac{1}{K}\sum_{k=1}^{K} I[k]$$

Equation 6-2

Rejection sampling will also be used to calculate approximate posterior distributions using the *R* 'abc' package (231). Posterior distributions describe the probability that each model parameter will take a particular value from its input range and captures the uncertainty around each parameter given the observed data (232). Posterior distributions are estimated by accepting or rejecting a set of sample parameters by estimating how far the resulting model output is from the target data. The difference between the simulated model output summary statistics and the observed target data summary statistics is calculated using Euclidean distance, and parameter samples are accepted or rejected based on this distance. A tolerance threshold can be selected using the 'abc' package and determines the proportion of "best fitting" simulations that are selected to generate the posterior distributions.

Essentially model calibration compares the simulated output from the model to the chosen target data; and uses a metric (the implausibility value) to represent how close the model output is to the target data. In doing so, the calibration procedure will reveal if the mechanisms encoded in the ABM can generate the AHP, and if so under which parameterization settings this occurs.

6.4.6 Step Six: Validation of explanatory power

The final stage of the critical mechanism-based approach involves the validation of the explanatory mechanism or mechanisms identified in the previous stage. Validation involves identifying the explanatory power of a mechanism, often by comparing mechanisms, to identify the mechanism which can best explain the observed phenomenon (219). As this PhD proposes to test only one theory, a comparison of mechanisms will not be possible. However, if the calibration process identifies a set of parameters which closely reproduce the observed target data (e.g., implausibility value < 1), model validation can be conducted to test the explanatory power of the candidate mechanism. Model validation involves comparing the model output to additional target data, that has not been used in the calibration procedure. Therefore, the final five years of the target data (2015-2019) will be preserved for model validation. If the identified model can closely fit to the subsequent years of data, this indicates that the mechanisms encoded in the model have good explanatory power.

6.5 Summary

In reflection of this PhD's aim to test whether a candidate theory-based mechanism can create and sustain the AHP, it is critical to explicitly consider the choice of research paradigm. A realist mechanism-based approach allows a depth of understanding into how a mechanism or mechanisms generate observed phenomenon and in which context they exist. An appropriate methodological approach is to develop a computer simulation - specifically an ABM. The ABM will be built in a Scottish setting as there is clear evidence that the paradox exists in that geographical context, and that targets and secondary data to generate a synthetic population are readily available.

Chapter 7 Translating social support and fundamental cause theories to an agent-based model

7.1 Chapter Overview

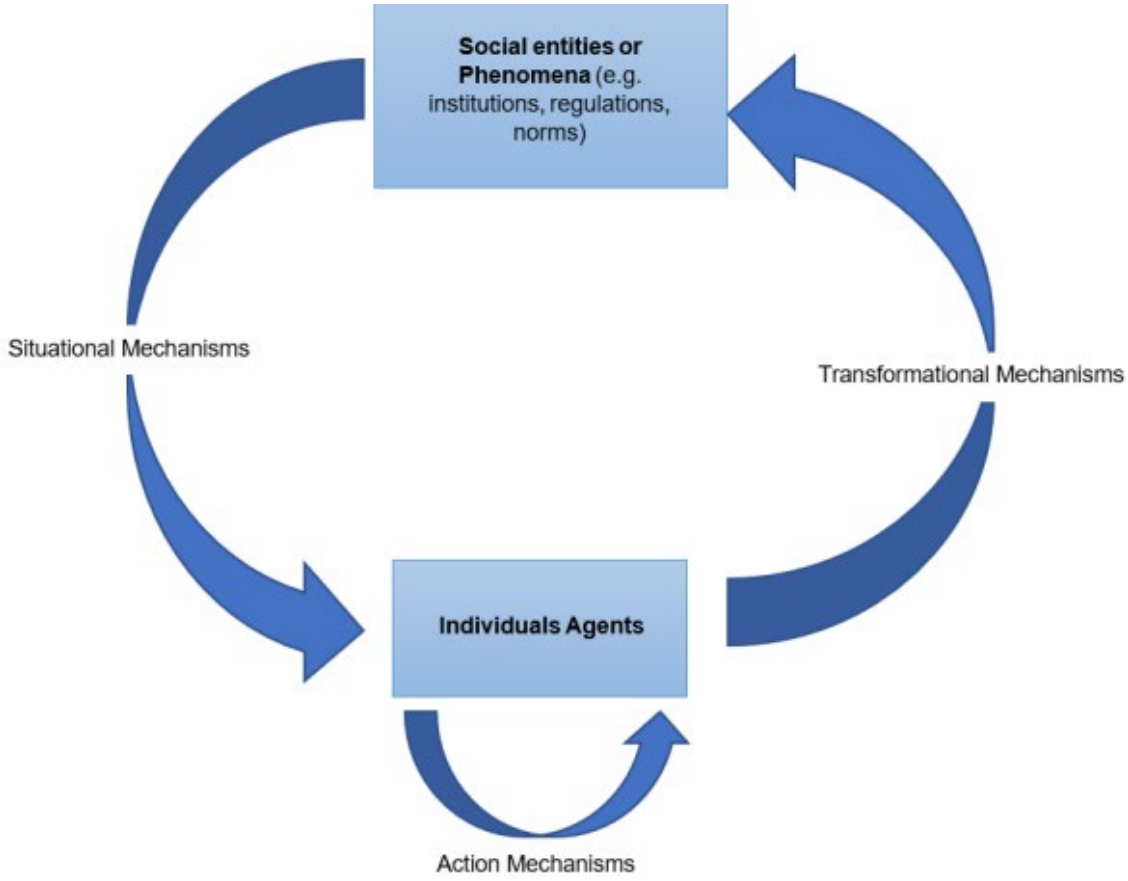
This chapter will apply an existing approach to translate two theories described in chapter 5 into conceptual models that can be used to design two separate ABMs to explain the AHP. This chapter will provide background covering the features of the mechanism-based social systems modelling (MBSSM) architecture, describe in detail two examples of conceptual models designed using the architecture and discuss the reasons for taking one of these conceptual models forward to the model implementation stage.

7.2 Background

The first stage of ABM development is to develop a conceptual model which translates a theory into mechanisms and entities which can be coded into an ABM. Abductive reasoning is required to formulate a conceptual model, specifically *redescription* which aims to abstract the interacting entities and mechanisms within the theory that are argued to generate observable phenomenon (160,217), for example the Alcohol Harm Paradox. Meta-models (defined in Chapter 6 section 6.4.3) are useful tools to extract entities and mechanisms described in a theory. As previously discussed, this PhD draws on the MBSSM architecture developed by Vu and colleagues (160).

The MBSSM architecture follows a general micro-macro scheme (see Figure 7.1). This architecture specifies two types of entity; macro-level entities which tend to be social entities or phenomenon (e.g., institutions or norms), and micro-level entities which can represent

Figure 7-1: *MBSSM Macro-Micro Meta-Model adapted from Vu et al., 2020.*



individuals or households (160). According to this architecture there are three types of mechanisms that can be extracted from a theory: situational, action, and transformational.

Situational mechanisms within the MBSSM refer to the influence of social entities on individual agents' attributes (160). Situational mechanisms may arise from the perceived collective behaviours of individual agents (e.g., the influence of social norms on behaviour), directly from the interaction between social entities and individual agents (e.g., the implementation of rules and regulations by institutions that are imposed on individuals) or where the social entities are the relationships and social contexts in which individual agents act (e.g., a social network connecting agents to other agents). This type of mechanism is useful for representing how the wider environment impacts on beliefs or values (160).

Action mechanisms are the processes by which individuals make decisions (either consciously or unconsciously) often based on their attributes, characteristics such as age, sex and

occupation, to then perform actions based on that decision (160). Implementing action mechanisms often involves drawing on an agent-decision making architecture which can be based on simple rules or informed by psychological theory; for example, the Belief, Desire, Intention framework (233) or Dual Process Theory (208).

Finally, transformational mechanisms or micro-macro mechanisms result from the collective actions of individuals which then impact on features of social entities in the model (160). Manzo (234) has helpfully distinguished transformational mechanisms into two general types: "*simple aggregation mechanisms*" and "*complex aggregation mechanisms*". Simple aggregation mechanisms are those which result from the actions of individual independent agents that are not connected to each other (160). However, within a macro-micro meta-model, simple aggregation mechanisms are rare given that there is often agent-agent or agent-environment interactions. The other type of transformational mechanism are complex aggregation mechanisms which can be distinguished further into two forms: direct complex aggregation ("*the aggregation of actions is based on acting agents whose states have been subject to interactions with other agents*") and indirect complex aggregation ("*the aggregation of actions is based on acting agents whose states have been subject to interactions with macro-level entities*") (160). The MBSSM provides the flexibility to encode both types or a combination of these complex aggregation mechanisms using sequencing; for both forms situational mechanisms would need to occur first in the model before transformational mechanisms do the aggregating (160).

The MBSSM architecture has been applied to translate theories to ABMs in the CASCADE (Calibrated Agent Simulations for Combined Analysis of Drinking Etiologies) project (208,235). Chapter 5 discussed several theories of health inequality that could be used to explain the AHP. It was beyond the scope of this PhD to construct conceptual models for each of these theories. Rather the purpose was to illustrate the process of taking a theory and using it to construct a conceptual model that could then inform the implementation of an ABM with the potential to explain the AHP. Therefore, two theories were selected from chapter 5; social

support theory and Fundamental Cause Theory (FCT). These theories were chosen for two main reasons; both theories specified a crucial role for the actions and interactions of individuals in generating health inequalities, which is well suited to the development of an ABM, and the components of both theories easily fit within the MBSSM architecture. The remainder of this chapter will apply the MBSSM architecture to develop two conceptual models based on social support theory and Fundamental Cause Theory (FCT).

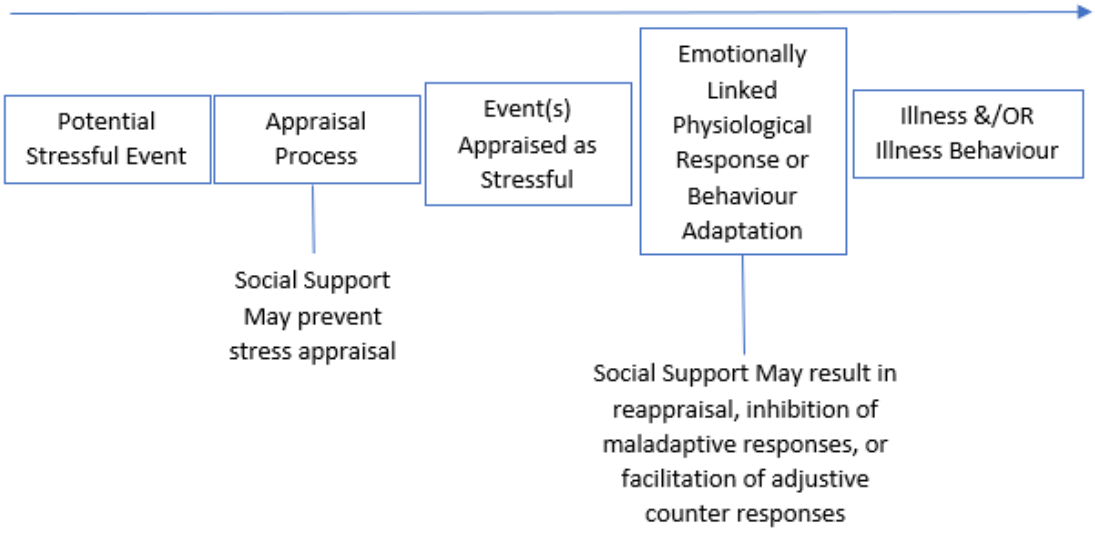
7.3 Social Support Model

As discussed in detail in chapter 5 the social determinants of health (SDH) are the social and economic factors that shape health at both the individual and population level (92,171). Psychosocial explanations for health inequalities are one category of explanations within the SDH that focus on the intersection between social and psychological experiences and how those experiences determine health. The role of social support is central to psychosocial explanations for health inequalities and it is possible that social support theory could explain the AHP.

There is a wealth of evidence to suggest that supportive behaviour has positive impacts for health (236–239). Cohen & Wills outline two processes through which social support is beneficial for health; the ‘buffering model’ and the ‘main-effect model’ (240). The ‘buffering model’ suggests that social support protects individuals from the negative biological impacts of stressful events. Research investigating the AHP suggests that alcohol harms are greater for those of a lower socioeconomic position because they experience a greater number of stressful life events, negative stereotyping, stigma and social isolation (22,37,144,146). In contrast, those from more affluent groups tend to have a beneficial network of social connections and therefore a greater social ‘buffer’ against stressful life events (2,98). The ‘main-effect model’ instead posits that social support has a positive and direct impact on health regardless of whether an individual experiences stress or not (240). These two models of social support do not necessarily compete, but rather represent two distinct processes through which social support impacts health.

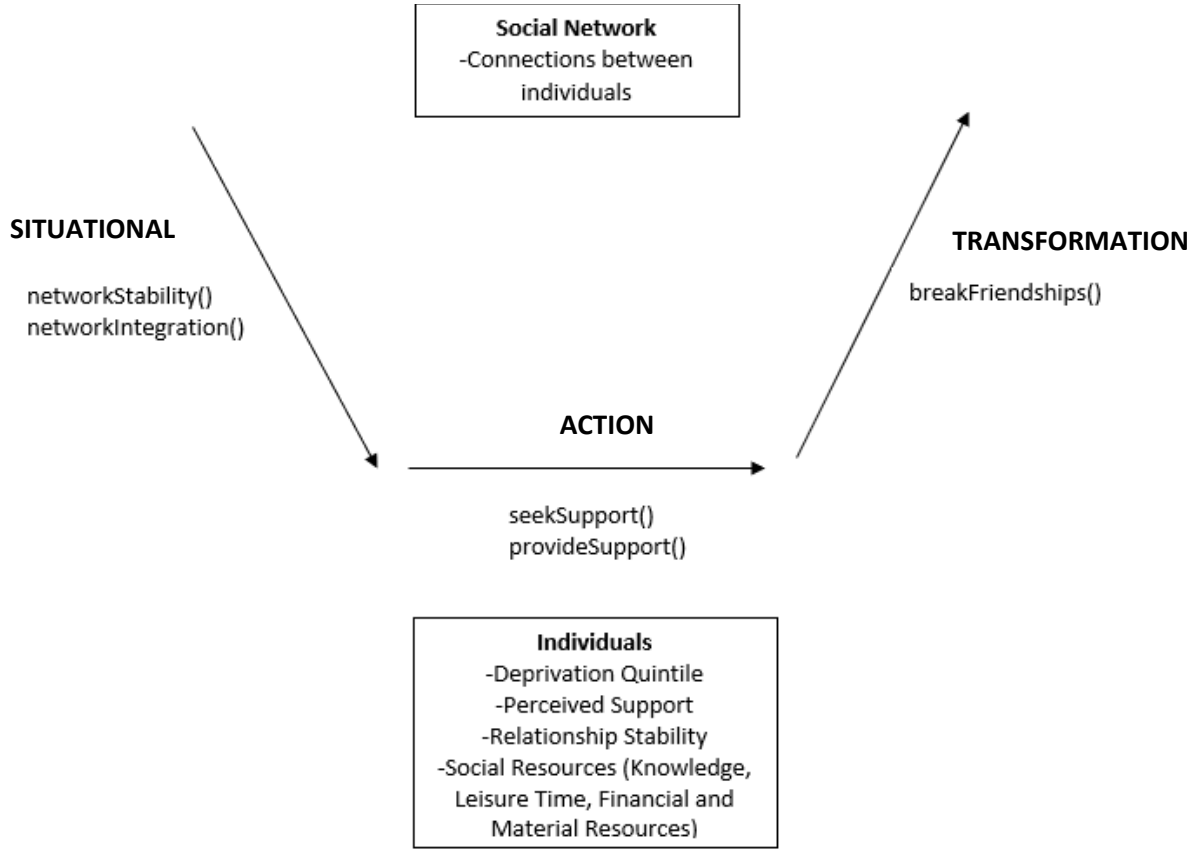
Cohen & Wills combine these two processes into one model of social support (Figure 7.2) (240). When individuals experience a potentially stressful event they first appraise that event, and in the presence of adequate social support, they do not perceive the event as stressful (240). However, if they do perceive the event as stressful they then experience an emotionally linked physiological response or attempt behavioural adaptation to the event. At this appraisal and response stage social support impacts the individual in three possible ways: 1) through a re-appraisal process, 2) by inhibiting maladaptive responses to stress and 3) by facilitating healthy adjustive responses to stress. Cohen & Wills detail four types of social support: 1) esteem support: the extent to which a person is supported by feeling esteemed or accepted despite difficulties or personal faults, 2) informational support: advice and help with defining, understanding and coping with stressful events, 3) social companionship support: spending time with others while engaging in recreational or leisure activities and 4) instrumental support: the provision of material or financial resources (240). In order to accurately construct an ABM of the influence of social support on health both processes, which can also be thought of as mechanisms, all four types of social support need to be encoded in the model.

Figure 7-2: *The model of social support and the buffering hypothesis adapted from Cohen & Wills, 1985* (240).



When applying the MBSSM architecture to develop an ABM, the first step is to define the entities required to operationalise the theory. For this theory it is clear that the micro-entities in the model are individuals, while the macro-entity is a social network connecting individuals. The situational mechanism in the model is the level of stability, integration and quality of relationships between agents within their social network. A greater degree of connectivity both in terms of actual and perceived social support of all four types would result in an appraisal that the event is not stressful. This situational mechanism broadly reflects the 'main-effect' model. However, in the absence of social support individuals would instead perceive the event as stressful, which would trigger one of two action mechanisms identified from this theory; the first to seek support, and the second to provide support. These actions are heavily influenced by the agents own attributes. For example, the decision to seek support would be a function of the agents' perceived support, financial resources and whether they possess coping skills to successfully adapt without seeking support. Similarly, an individual's decision to provide support would be based on the psychological and material resources the individual has available to assist their social connection. If support is provided to an individual this would 'buffer' the negative effect of the stressful event and they would either reappraise the event as not stressful or engage in adjustive coping as opposed to maladaptive coping. However, if they seek support and do not receive it this would result in maladaptive coping which would negatively impact their health. In the context of the alcohol harm paradox this would be the experience of alcohol harm. When individuals seek support and do not receive it, this would place strain on their relationship with their social connections and they may decide to break the friendship, an action mechanism. The collective changes being applied to the social network as the result of decision to break friendships would be the transformational mechanism and the social network structure would be updated. Figure 7.3 illustrates a simplified MBSSM conceptual model of social support.

Figure 7-3: *Simple Social Support MBSSM Conceptual Model.*



In Figure 7.3 agents are represented in the two boxes as individuals and the social network which is an agent in itself as it possesses attributes and can carry out methods. Individuals have the following attributes: deprivation quintile, perceived support, relationship stability, and social resources, while the social network contains connections between individuals. Methods are indicated next to the arrows as situational methods, action methods and transformational methods. The social network can assess the network stability and network integration of the existing network and communicate this to individuals. Individuals can then act to seek support or provide support from or to fellow individuals in their social network. Individuals can also break friendships.

7.4 Fundamental Cause Theory Model

Another theory described in chapter 5 that could be applied to understand the AHP is fundamental cause theory (FCT). FCT aims to shift the focus from individual-level causes of

health inequalities (e.g., alcohol consumption), to looking at the context – what puts people "at risk of risks" (96). Central to this theory are fundamental resources defined as power, money, knowledge, access to social connections and prestige. These resources determine whether individuals have the ability to adapt to the introduction of a new disease, risks or treatment (97). FCT proposes that advantaged groups have increased access to these flexible resources and therefore can deploy them to avoid risks, reduce the consequences of disease and uptake available treatment to improve their health. In contrast, these resources are not readily available to disadvantaged groups.

FCT does not appear in research investigating the AHP, however it has been used to understand inequalities in the development of lung cancer. One study used FCT to predict that as lung cancer becomes more preventable, due to knowledge of risks and treatment, those with greater access to fundamental resources disproportionately benefit and health inequalities for the disease increase (181). This is a particularly compelling explanation for health inequalities in preventable diseases given that for a disease lacking in major prevention or treatment innovation (e.g., pancreatic cancer) there is no difference in mortality rates between socioeconomic groups (181). Alcohol-related harms (e.g., liver disease) are also largely preventable therefore FCT could explain the large socioeconomic inequalities in these harms.

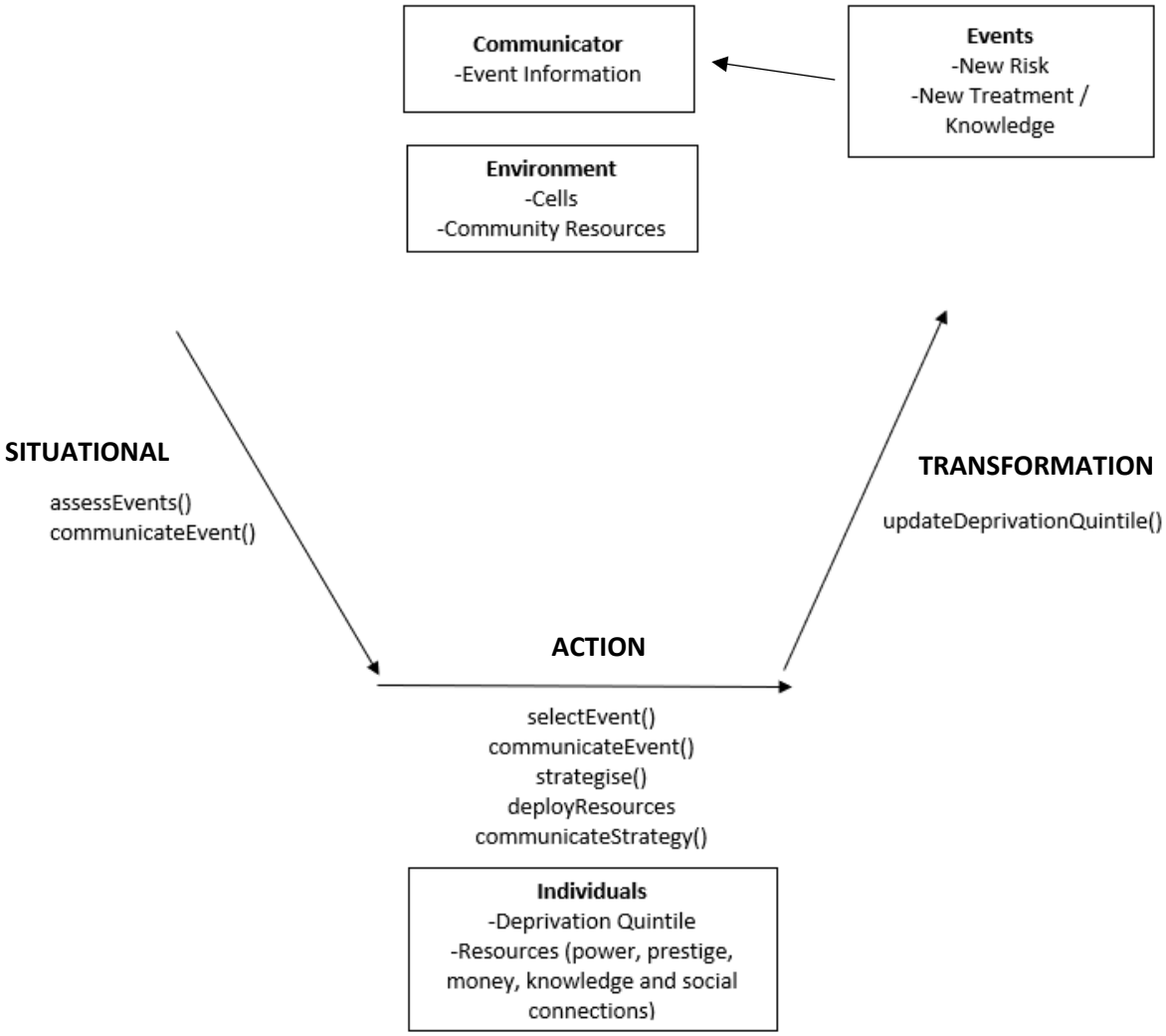
Alcohol harm is arguably unique compared to harms from smoking given that those of a higher SEP tend to consume more alcohol on average, whereas smoking is much more prevalent in those of a lower SEP (38). So rather than using FCT to explain a shift in health behaviour, as is done for smoking, FCT can instead be understand how fundamental resources could be used to moderate the relationship between alcohol consumption and alcohol-specific death. For example, while more affluent people may consume more alcohol on average, they consume their alcohol in safer neighbourhoods and settings, and have healthier drinking patterns (e.g., drinking with meals at a restaurant). These examples refer to moderation via alcohol consumption, however FCT could also be used to explain inequalities in alcohol harm

via pathways not directly related to drinking. For example, greater access to fundamental resources means that more affluent people can uptake treatments and visit healthcare services, choose to work in better conditions, and purchase better housing all of which may compound the effect of alcohol consumption on health. However, the model developed for this PhD did not distinguish these two causal pathways as the 'events' in the model remained abstract.

To translate FCT into an ABM using the MBSSM we first identified the entities present in this theory. We identified two macro entities; the environment, a physical space that represents the community's individuals reside in categorised by area-level deprivation quintile and the communicator - an entity that communicates and receives information from an exogenous source which is a broad abstraction of the source of innovations regarding risk, treatment and prevention. The macro-level agent which performed behaviours is the communicator entity. This agent broadly represents a societal institution (e.g., the mass media or health service) that communicates new information about the risks, treatment or knowledge of the causes and cures of alcohol-related disease to individuals (the situational mechanism). We identified individuals as micro-level entities. Individuals perform several action mechanisms. Given the complexity of FCT, to illustrate the behaviours of individual agents, we present a walk-through of two individual agents: "Agent A" and "Agent B". "Agent A" lives in the least deprived community and has a relatively large resource pool, while "Agent B" lives in the most deprived community and has few resources available to them. Both individuals receive information about a new brief intervention from the communicator entity. "Agent A" and "Agent B" remember all the information they have been informed of (new risk, treatment or prevention information), information that they have already adapted their behaviour to and information that they have not yet adapted their behaviour to. Both agents have the opportunity to select a piece of information from their memory (represented using a list variable) that they have not yet adapted to.

Both agents then have the opportunity to inform someone in their social network of the new event, in this example about the new brief intervention available. Both “Agent A” and “Agent B” then check whether anyone in their social network has provided them with a strategy on how to adapt their behaviour in response to this information. “Agent A” may have received strategic information from a connection in their network that a friend has successfully adapted their behaviour (e.g., they received the treatment while attending a particular healthcare service). This information provides “Agent A” with a strategy to adapt to the relevant event (e.g., attend that same healthcare service), while “Agent B” has not received any advice on how to adapt. Awareness of a strategy enhances the resources available to “Agent A” that are required to adapt to this information, making it easier for “Agent A” to adapt their behaviour. Both agents then draw on their available resource pool, a composition of power, prestige, money and knowledge, to adapt and change their behaviour. “Agent A” has sufficient resources to adapt their behaviour to this information and successfully takes on the new behaviour (e.g., they attend the healthcare service and receive the intervention) which reduces their risk of alcohol harm, they therefore deploy their resources to adapt. Successful adaptation always costs “Agent A” a proportion of their available resources (e.g., there are financial costs associated with attending treatment from transportation in the form of bus fares or petrol but also potentially in lost work hours). However, their knowledge resource increases because they have learned from their successful adaptation. Following successful adaptation “Agent A” can also communicate their adaptation strategy to another social connection. On the other hand, “Agent B” does not have sufficient available resources and cannot successfully adapt they therefore do not attend treatment. Individual agents also have the opportunity to move communities throughout the simulation, and as a result transform the environment they live in and the community resources available in that area which are an aggregation of all the resources of individuals living in that community. Individuals that move location update their deprivation quintile to the quintile associated with the new community they now reside in. Figure 7.4 illustrates a simplified MBSSM conceptual model of FCT.

Figure 7-4: *Simple FCT MBSSM Conceptual Model.*



In Figure 7.4 agents are represented in boxes as the communicator, individuals and to an extent the environment itself. The attributes belonging to these agents are listed in the boxes, the communicator contains event information, the environment contains cells and information on community resources, while individuals have the following attributes: deprivation quintile, power, prestige, money, knowledge and social connections. The methods are listed next to each arrow. The communicator agent can engage in two methods; assess whether an event has occurred and communicate that event to individuals. While the individuals have several methods including the selection of an event, communicating the event to their social connections, looking for a strategy to adapt to the

event, deploying their own resources and communicating a strategy to their social connections. Individual agents can also move location and therefore can update their deprivation quintile accordingly.

7.5 Theory Selection

Both conceptual models outlined above could be used to implement an ABM to attempt to explain the AHP. However, due to the complexity associated with generating an appropriate microsynthetic population, and implementing, calibrating and validating an ABM, it was only possible to focus on one theory in this PhD thesis. To select a theory, both the appropriateness of the theory to explain the AHP and whether there was data freely and readily available to parameterise the model in the Scottish setting were considered.

FCT is particularly well suited to explain the AHP as it shifts the focus from individual risk behaviours (e.g., alcohol consumption), however it does not preclude the role of these risk factors. Research exploring the causes of the AHP has found that alcohol consumption does contribute to socioeconomic inequalities in alcohol harm (41). FCT suggests that the base mechanism generating health inequalities is the use of fundamental resources to adapt to the introduction of new disease, risks or treatment (97). FCT situates alcohol consumption as a risk factor that can be managed by deploying fundamental resources, with a backdrop that the distribution of these fundamental resources is unequal; a reflection of the social stratification of society (97). This somewhat explains the paradoxical effects of the AHP, as even though those of a higher socioeconomic position drink more alcohol on average they too have greater access to fundamental resources and can deploy them to act and reduce their risk of harm. The role of alcohol consumption in a model pertaining to social support theory is less clear. Social support theory does clearly state that the experience of stressful events in the absence of social support results in maladaptive coping and worse health outcomes (240), which could include drinking alcohol to cope. However, the clearer link to harm in social support theory is physiological responses to stressful events that are potentially dampened in the presence of social support, and in which alcohol consumption plays no direct role.

Additionally, FCT has previously been used to understand socioeconomic health inequalities in lung cancer (181). Lung cancer is similar to many alcohol-related harms including liver disease, as it is also closely related to health behaviour but rather than alcohol consumption, the behaviour of interest is smoking. Contrastingly social support theory has prominently been used to understand differences in wellbeing (240). While it is possible that wellbeing has a role in generating the AHP, the link from the social support model to physical health outcomes such as alcohol-related mortality is less clear.

Critical to operationalising a model of FCT is sourcing data on variables which can represent the fundamental resources: power, money, knowledge, prestige and social connections. There are several available data sets on the UK Data Service that collect information on income, education, and occupational status that could be used to parameterise money, knowledge and prestige respectively. For example, the British Household Panel Survey collected detailed social and economic information from a large sample of UK households (241). To operationalise the social support model, we would require data to generate an accurate social network and variables to define the four types of social support described by Cohen and Wills: esteem support, information support, social companionship and instrumental support (240). There are very few studies that collect detailed data on social networks in the context of alcohol use and the majority of these focus on young adult populations in university settings in the US (242). Therefore, it was not possible to obtain the data required to set up a representative social network for an adult population in Scotland. Additionally, more nuanced data is required to parameterise different types of social support. It would be possible to use variables from the British Household Panel Survey, discussed above, which capture how much time is spent engaging in leisure activities to operationalise social companionship or variables capturing financial and material resources to operationalise instrumental support. However, it is difficult to justify that these variables accurately reflect the social support constructs defined by Cohen & Wills.

In summary, based on relevance and data availability we selected FCT as the candidate explanation to implement and test using ABM.

7.6 Summary

To summarise this chapter has illustrated how conceptual models can be constructed from theory in a format that facilitates the development of a computational agent-based model. Two detailed examples are provided which draw on social support theory and FCT. It was determined based on appropriateness and data availability that FCT is selected for model implementation and testing.

Chapter 8 Developing a static microsimulation model of the Scottish population

8.1 Chapter Overview

This chapter will describe the process used to generate a synthetic population representative of the Scottish population in 2001, which is the spatio-temporal setting for examining the AHP and testing FCT. In doing so the chapter will cover data processing, the method used to construct the synthetic population, and results from internal and external validation of the resulting population. It will also describe the process of assigning alcohol consumption to individuals in the synthetic population using health data not included in the British Household Panel survey, and subsequent data analyses used to create variables which represent the fundamental resources described in FCT (money, power, prestige and knowledge). R version 4.1.2 was used for the data cleaning and analysis procedures described throughout this chapter.

8.2 Background

To construct a data driven agent-based model requires a sample of heterogeneous agents based on the population of interest. The setting for the model is Scotland from 2001-2019, therefore, it was necessary to obtain a baseline population of the Scottish population in 2001 to enter the model in that year. Given that such a resource does not already exist, a microsimulation modelling approach outlined by Wu and colleagues (227) was used to generate a synthetic population. This approach estimates the characteristics of a population using a combination of attribute-rich individual-level data (e.g., survey data) and geographical (area-level) aggregate counts of individuals living in specific areas. The micro units are individuals which are simulated by assigning them attributes from other data sources (227).

The aim of this chapter was to implement a static microsimulation approach to produce a synthetic population of adult individuals (aged 16 and older) at the Data Zone scale (with

approximately 500-1000 individuals per data zone) for Scotland, in the year 2001. Data Zones are the key geographical unit used in Scotland to capture small area-level statistics (243).

8.3 Data cleaning

To generate a synthetic population two types of data are required: aggregate counts of individuals at the area-level and attribute rich individual-level data (227). For this microsimulation we used the 2001 Scottish Census which provided constraint tables containing aggregate counts for each Data Zone. These counts were broken down by age/sex, marital status, economic status, and highest educational qualification, for each of the 6,505 data zones in Scotland. For example, in Data Zone S01000001 there were 23 males between the ages of 16 and 24 years, and 371 people in paid employment. It is known how many people are in each of these categories, however the microsimulation model estimates who is in both categories. See tables 8.1, and 8.2 for extracts of example constraint tables used in this model.

Table 8-1: *Extracted sample of the sex/age constraint table.*

DataZone	Male 16-24	Male 25-34	Male 35-49	Male 65-74	Male 75+	…	Female 75+
S01000001	23	109	108	21	13		32
S01000002	9	141	90	3	0		2
S01000003	15	104	143	4	5		4

Table 8-2: *Extracted sample of the economic status constraint table.*

DataZone	In paid employment	Self-employed	Student	Retired	Other economically inactive	Unemployed
S01000001	371	72	37	72	80	22
S01000002	423	25	14	15	36	6
S01000003	467	29	24	18	49	14

To provide the non-geographical and attribute rich individual-level data, also termed ‘microdata’, we used the British Household Panel Survey (BHPS) 2001. Originally, we had decided to use the Scottish Household Survey (SHS) 2001, however while the resulting population was internally valid (R^2, SRMSE and SAE values were similar to those found when

using the BHPS – see Table 8.5), the population failed tests of external validity. The synthetic population constructed using the Scottish Household Survey did not accurately represent the deprivation level of Data Zone. For example, household annual income and area-level deprivation were weakly correlated (r = 0.37, p<0.001) when using the Scottish Household Survey compared to the correlations observed in previous research which tend to be around r = 0.8 (227). While it is to an extent unclear why the generated synthetic population failed tests of external validity, there could be two possible reasons for this. The first that there is a problem with the survey sample itself; mainly data in the Scottish Household Survey is only collected at the household level rather than the individual level and therefore data in this survey pertains to the individual that identifies themselves as head of the household. Alternatively, additional constraints may be required to generate a more accurate population using this survey data. However, concerns around the constraints used can be discounted as they were used to successfully generate a representative and externally valid population of Scotland when using the BHPS. As it was particularly important for this thesis to accurately represent area-level deprivation given that the aim is to understand socioeconomic inequalities in alcohol harm. Therefore, instead of using the Scottish Household Survey we opted to use the BHPS 2001.

The BHPS began in 1991 and follows the same representative sample of individuals until 2009, when it was then replaced with the Understanding Society Survey (241). The 2001 survey sample consisted of 15,519 individuals sampled from across the UK and collects information on a range of topics including demographic, social, health, economic and behavioural information.

To create a microsimulation model of representative of a particular population weights need to be derived to estimate how many times an individual from the survey data needs to be replicated to construct that representative population. To calculate the weights for each individual in the survey data, linking variables available in both the aggregate and individual level data are required (227). The BHPS includes sociodemographic information that overlaps

with the variables in the aggregate area-level counts obtained from the census. The sociodemographic information used in this model were personal identifier, sex, age, marital status, economic activity, and highest educational qualification. Ethnic group was not included in this model as it was missing for more than half of the cases in the BHPS. Missing cases for the other sociodemographic variables were excluded leaving 14,623 adult individuals in the microdata. Comparing the demographic data of the population including and excluding missing cases revealed that it was likely that the missing data was missing at random given negligible differences in demographics after removing missing cases (see Table 8.3). To match the constraint aggregate counts from the census data the sociodemographic variables in the microdata were formatted as shown in Table 8.4.

Table 8-3: *Demographic data for the BHPS survey population including and excluding missing cases.*

	All cases (n = 15,519)	**Excluding missing cases** (n = 14,623)
Age (Mean)	45.4	45.5
Sex		
Male	7140 (46%)	6650 (45.5%)
Female	8379 (54%)	7973 (54.5%)
Economic Activity		
In paid employment	7888 (50.8%)	7471 (51.0%)
Other economically inactive	1827 (11.8%)	1719 (11.8%)
Retired	3310 (21.3%)	3183 (21.8%)
Self employed	1012 (6.5%)	941 (6.4%)
Student	816 (5.3%)	773 (5.3%)
Unemployed	576 (3.7%)	536 (3.7%)
NA	90 (0.6%)	-
Marital Status		
Married	8257 (53.2%)	7827 (53.5%)
Divorced	1341 (8.6%)	1292 (8.8%)
Separated	316 (2.0%)	297 (2.0%)
Single	4328 (28.0%)	4005 (27.5%)
Widowed	1263 (8.1%)	1202 (8.2%)
NA	14 (0.1%)	-
Education		
Group 1	3433 (22.1%)	3413 (23.3%)
Group 2	1752 (11.3%)	1742 (11.9%)
Group 3	528 (3.4%)	525 (3.6%)
Group 4	5250 (33.8%)	5220 (35.7%)
No qualifications	3743 (24.1%)	3723 (25.5%)
NA	813 (5.2%)	-

Table 8-4: *Summary of variables in the British Household Panel Survey (BHPS) adult microdata.*

Variable	Description	Values and categories
UNIQID	ID number assigned to each individual in the dataset	e.g., 1, 2, 3, 4, etc.
JSex/Jage	Sex and age group	M_16_24 (Male, aged 16-24)
		M_25_34 (Male, aged 25-34)
		M_35_49 (Male, aged 35-49)
		M_50_64 (Male, aged 50-64)
		M_65_74 (Male, aged 65-74)
		M_75+ (Male, aged 75 and over)
		F_16_24 (Female, aged 16-24)
		F_25_34 (Female, aged 25-34)
		F_35_49 (Female, aged 35-49)
		F_50_64 (Female, aged 50-64)
		F_65_74 (Female, aged 65-74)
		F_75+ (Female, aged 75 and over)
Jjbstat	Economic status	In_paid_employment
		Self_employed
		Student
		Unemployed
		Retired
		Other_economically_inactive
jqfedhi	Highest educational qualification	No qualifications
		Group 1 (GCE O-levels or equiv)
		Group 2 (GCE A-levels or equiv)
		Group 3 (CSE Grade 2-5, Scot Grade 4-5)
		Group 4 (Degree level)
jmlstat	Marital status	Married
		Separated
		Divorced
		Widowed
		Single

8.4 The software and algorithm

To generate the microsimulation model, we used the Flexible Modelling Framework (FMF) software. This software was developed by the University of Leeds as a tool to build microsimulation models (244). Both the microdata taken from the survey and the constraint tables sourced from the census data are used as inputs. The FMF implements a simulated annealing approach to generate a microsynthetic population. Simulated annealing selects the optimal configuration of individuals from the microdata that best fit the population observed in the aggregate counts sourced from the census (227). This approach randomly selects

individuals from the microdata and considers whether they should be admitted to a Data Zone based on the goodness of fit to the constraint tables. This process is repeated and individuals are replaced in each Data Zone until the fit is optimized (227,245,246).

Upon completion of this process, a list of individual identifiers and the codes for the Data Zone each individual resides in is produced. Identifiers can then be used to join the individual-level attribute-rich data available from the full survey with each individual in the synthetic population. A total of 4,089,946 individuals were simulated.

8.5 Internal and External Validation

To ensure that the synthetic population was representative of the desired population, we carried out internal and external validation tests. Internal validation compares the simulated data to the datasets used as inputs in the simulation (227,247). Internal validity for each constraint variable was assessed by examining several commonly used fit statistics: R^2, Standardised Root Mean Square Error (SRMSE), and Standard Absolute Error (SAE). The goodness of fit statistics displayed in Table 8.5 demonstrate a good fit between the simulated and observed data. The R^2 for all constraint variables was above 0.9, and the error estimates were low.

Table 8-5: *Validation metrics for the comparison of simulated and actual counts in each constraint.*

Constraint	R^2	SRSME	SAE
Age/sex	1.0	0	0
Marital status	1.0	0	0
Economic status	0.970	0.199	0.067
Highest educational qualification	0.925	0.215	0.096

We also externally validated the microsimulation model by comparing simulated results to a dataset not used to generate the model. We compared simulated results at the aggregate Data Zone level with estimates from the Scottish Index of Multiple Deprivation (SIMD), using the data from 2004 as the closest year available. SIMD is a relative measure of area-level

deprivation which consists of seven domains: income, employment, health, education, geographic access to services, housing and crime (248). SIMD can be used to rank the 6,505 Data Zones in Scotland from the most deprived (rank 1) to the least deprived (rank 6,505). There is a well-established relationship between deprivation and personal or household income, where those from more deprived areas tend to have lower income (249). Therefore, the SIMD rank of each Data Zone should in theory strongly correlate with measures of income. As expected a higher rank was strongly positively correlated with monthly income ($r = 0.8313$, $p < 0.0001$), and annual income ($r = 0.8185$, $p < 0.0001$), and strongly negatively correlated with annual benefits receipt ($r = -0.7714$, $p < 0.0001$). This highlights that less deprived datazones in the microsimulation model on aggregate have higher incomes and less money received from benefits.

Both internal and external validation results indicate that the simulated synthetic population adequately captures the characteristics of the Scottish 2001 population for the included attributes.

8.6 Demographics of the Scottish Microsimulation

The descriptive demographic data by SIMD deprivation quintile for the Scottish static microsimulation model is displayed in Table 8.6. The average age, and the sex split were similar across all SIMD quintiles. There was a visible socioeconomic gradient in marital status. The most deprived quintile (quintile 1) had the lowest proportion of married individuals (36.4%) and the highest proportion of those that were divorced (10.2%), separated (5.4%), widowed (10.8%) or never married (37.2%). While the least deprived quintile (quintile 5) had the highest proportion of married individuals (60.2%) and the lowest proportion of those that were divorced (4.3%), separated (2.3%), widowed (10.8%) or never married (26.4%).

There was also a clear socioeconomic gradient in both economic activity and education. Only 37.2% of individuals in the most deprived quintile were employed, while 52.5% of individuals in the least deprived quintile were employed. The opposite trend was seen for levels of unemployment as almost three times the proportion of individuals in the most

deprived quintile were unemployed (11.9%) compared to the least deprived quintile (3.2%). In terms of education more than double the proportion of individuals in the most deprived quintile reported that they had obtained no qualifications (45.7%) compared to the least deprived quintile (20.3%). While the proportion of those obtaining a first degree or a higher degree was double in the least deprived quintile (8.4% and 2.1% respectively) compared to the proportion in the most deprived quintile (4.2% and 0.9% respectively). The microsimulation model therefore clearly demonstrates an expected socioeconomic gradient in economic activity and education associated with deprivation quintile.

Table 8-6: *Descriptive demographic data for the Scottish Microsimulation Model by SIMD deprivation quintile.*

	Quintile 1 (*n*=814,479)	**Quintile 2** (*n*=825,230)	**Quintile 3** (*n*=811,301)	**Quintile 4** (*n*=808,498)	**Quintile 5** (*n*=830,438)
Age (Mean, sd)	45.6 (19.2)	46.2 (19.3)	46.6 (18.95)	46.3 (18.4)	45.6 (18.13)
Sex					
Male	45.8%	46.8%	47.6%	48.0%	48.2%
Female	54.2%	53.2%	52.4%	52.0%	51.8%
Marital Status					
Married	36.4%	44.7%	51.3%	56.2%	60.2%
Divorced	10.2%	8.2%	6.8%	5.7%	4.3%
Separated	5.4%	4.0%	3.3%	2.9%	2.3%
Widowed	10.8%	10.3%	9.3%	8.1%	6.8%
Never Married	37.2%	32.8%	29.3%	27.1%	26.4%
Economic Activity					
Employed	37.2%	44.9%	47.4%	50.6%	52.5%
Self-employed	2.7%	4.5%	7.4%	8.5%	7.8%
Unemployed	11.9%	7.9%	5.9%	4.3%	3.2%
Training Scheme	0.2%	0.2%	0.2%	0.2%	0.1%
Maternity leave	0.2%	0.2%	0.3%	0.3%	0.3%
Long term sick	11.9%	7.4%	5.6%	4.4%	3.4%
Retired	15.8%	18.5%	19.1%	18.6%	18.2%
Family Carer	14.7%	10.1%	7.8%	6.5%	5.5%
Student	5.5%	6.3%	6.3%	6.6%	8.9%
Education					
No qualifications	45.7%	36.4%	31.2%	20.9%	20.3%
Apprenticeship	1.9%	2.2%	2.3%	2.0%	2.0%
Commercial QF	2.2%	2.5%	2.6%	2.4%	2.3%
CSE Grade 2-5, Scot Grade 4-5	4.9%	5.9%	6.4%	7.8%	7.6%

GCE O Levels or Equiv	20.0%	20.0%	19.3%	16.7%	16.3%
GCE A Levels	10.4%	13.1%	14.6%	18.8%	18.3%
Nursing QF	0.7%	0.9%	1.0%	1.6%	1.5%
Teaching QF	0.9%	1.3%	1.7%	2.7%	2.6%
Other Higher QF	8.4%	11.0%	13.2%	19.0%	18.5%
First Degree	4.2%	5.5%	6.3%	8.8%	8.5%
Higher Degree	0.9%	1.2%	1.4%	2.1%	2.1%

**Note Quintile 1 is the most deprived and Quintile 5 is the least deprived.*

8.7 Integrating alcohol consumption from the GHS and SHeS

The BHPS used to generate the synthetic population does not collect information on health behaviour. As discussed in previous chapters the Alcohol Harm Paradox is the consistent finding that those of lower socioeconomic position are a greater risk of alcohol harm despite consuming the same or less alcohol than those of a higher socioeconomic position (16,41). Evidence suggests that alcohol consumption does play a partial role in this relationship (41), therefore to create an accurate representation of the alcohol harm paradox a measure of alcohol consumption is required. To provide each individual with an estimated value for alcohol consumption we used the General Household Survey (GHS) for years 2001-2006, and the Scottish Health Survey (SHeS) for the years 2008-2019. We opted to use average units of alcohol consumed per week as the measure of alcohol consumption, as it was consistently measured across survey years (excluding the GHS in 2003 and 2004).

8.7.1 Estimating baseline alcohol consumption

Following removal of cases with missing data on economic status the GHS 2001 contained a sample of 16,443 adult individuals. Of this sample 8.9% did not have a recorded value for average units of alcohol consumed per week. Therefore, we used multiple imputation to estimate a value for average units per week for missing cases. We implemented the multiple imputation procedure using the MICE package in R version 4.1.2. We used age, sex (male/female), marital status (single, married, divorced, separated, windowed), economic status (employed, unemployed, student, economically inactive) and equivalisation scale

(measure used to adjust household income to account for family size and structure) to estimate missing values for alcohol consumption.

Following this we used propensity score matching to match individuals from the GHS to individuals in the BHPS to estimate average units of alcohol consumed per week. Propensity score matching was carried out in R version 4.1.2 using the MatchIt package. The first step was to format both datasets to ensure each of the demographic variables were coded in the same way across datasets. This involved recoding the BHPS economic activity variable to match the economic status variable in the GHS. 'Family care', 'LT sick, disabled', and 'Retired' were all recoded to 'economically inactive', and 'Gvt trng scheme', 'maternity leave', and 'self-employed' were recoded to 'employed'. A new variable 'S' (to indicate which survey each individual belonged to) was then created and those in the GHS assigned the value of 0 and those in the BHPS assigned a value of 1. The dependent variables used for matching were age, sex, marital status and economic status. We attempted to also use highest educational qualification as a matching variable, however the coding between the GHS and BHPS was drastically different and therefore recoding was not possible. A binomial logistic regression was used to calculate propensity scores PS for each individual was as follows:

$$PS = Prob(S = 1)|L: age, sex, maritalStatus, economicStatus$$

Equation 8-1

Where $Prob(S = 1)$, is the probability that the individual belongs to the BHPS given the demographic constraints age, sex, marital status and economic status. This essentially generates a propensity score for each individual in both surveys that represents the probability that individual would be recruited by the BHPS based on the demographic variables used in the formula. The propensity score can also be thought of as how closely an individual in one survey matches an individual in the other survey based on the demographic their age, sex, marital status and economic status. We used the nearest neighbour method with a 1:1 ratio; each individual in the BHPS was matched with one individual from the GHS. Individuals in the BHPS were then assigned a value for average units of alcohol consumed per week as taken

from their match in the GHS. The *R* code for the propensity score calculation and matching procedure can be found in Appendix B.

8.7.2 Updating alcohol consumption from 2002-2019

As alcohol consumption can change over the life course (250,251), it was necessary to estimate any changes in drinking for each year of the simulation. Individual changes in alcohol consumption over time was the only dynamic process that did not occur within the ABM simulation, as the ABM itself did not contain mechanisms that aim to explain alcohol consumption. Instead a value for average units per week for every year of the simulation was estimated prior to agents entering the model. To do this we sourced data from the GHS for the years 2002-2006 and the SHeS for the years 2008-2019. The decision to use two datasets to estimate alcohol consumption was based on data availability. Ideally, we would have used the SHeS for each year of the simulation, however the SHeS is only available in 1998, 2003 and annually from 2008. Therefore, for consistency we used the GHS for the first six years of the simulation and the SHeS for the remaining years. There was no survey data available for 2007 – instead we merged the GHS 2006 and SHeS 2008, to estimate a dataset for 2007. For each year of survey data, we used the same multiple imputation method outlined in section 9.6.1 to estimate average weekly units of alcohol consumed for missing cases.

There was no measure of average weekly units of alcohol consumed in the GHS for years 2003 and 2004. To calculate average weekly units, we created a mapping algorithm using the 2005 and 2006 data, and applied this to the 2003 and 2004 data. A similar method has been used to estimate average consumption measures from diary data (252). To create the algorithm, we identified five variables that were present in all four datasets; age, whether they had drunk in the last 7 days (yes/no), how much they normally drink (Hardly at all, A little, A moderate amount, Quite a lot, Heavily), the number of weekly drinking days and the total units on the heaviest drinking day in the previous week. We created two mapping algorithms; one for males and one for females due to gender differences in alcohol consumption (253). We

also excluded abstainers, which were individuals in the datasets that reported that they don't drink and as a result were given a value 0 for average units per week.

A linear regression model was then used to predict the logarithm of units per week for the remaining drinkers, as the sample was not normally distributed. The results for males are displayed in Table 8.7, and Table 8.8 for females. The following model was used to estimate average units consumed per week in the 2003 and 2004 GHS data for men and women separately:

$$Log(unitsPerWeek) = \beta 1\ x\ ageGroup + \beta 2\ x\ drinkLast7 + \beta 3\ x\ drinkAmt + \beta 4\ x\ numDrinkDays + \beta 5\ x\ totUnitsHeaviestDrinkDay + \varepsilon$$

Equation 8-2

Table 8-7: Fitted regression model for average units consumed per week for males.

Model Parameter	Weekly alcohol consumption (units)			
	B (SE)		t	P>t
Intercept	0.365	0.037	9.837	<0.001
Age group: 16-24	-	-	-	-
Age group: 25-34	-0.134	0.034	-3.980	<0.001
Age group: 35-44	-0.228	0.032	-7.069	<0.001
Age group: 45-54	-0.241	0.033	-7.287	<0.001
Age group: 55-64	-0.280	0.033	-8.459	<0.001
Age group: 65-74	-0.386	0.035	-10.900	<0.001
Age group: 75+	-0.496	0.039	-12.717	<0.001
Drank in the last 7 days: Yes	-	-	-	-
Drank in the last 7 days: No	-0.547	0.028	-19.638	<0.001
Drink amount: Hardly at all	-	-	-	-
Drink amount: A little	1.179	0.026	45.389	<0.001
Drink amount: A moderate amount	1.871	0.028	68.029	<0.001
Drink amount: Quite a lot	2.274	0.039	57.966	<0.001
Drink amount: Heavily	2.542	0.086	29.571	<0.001
Number of weekly drinking days	0.152	0.005	33.312	<0.001
Total units on heaviest drinking day in the previous week	0.040	0.002	23.822	<0.001

Model details	
Number of observations	12,866
Residual SE	0.915
R^2	0.651
Adjusted R^2	0.650

Table 8-8: *Fitted regression model for average units consumed per week for females.*

Model Parameter	Weekly alcohol consumption (units)			
	B (SE)		t	P>t
Intercept	0.119	0.040	3.121	0.00181
Age group: 16-24	-	-	-	-
Age group: 25-34	-0.292	0.035	-8.267	<0.001
Age group: 35-44	-0.461	0.034	-13.538	<0.001
Age group: 45-54	-0.571	0.035	-16.183	<0.001
Age group: 55-64	-0.670	0.036	-18.536	<0.001
Age group: 65-74	-0.785	0.040	-19.829	<0.001
Age group: 75+	-0.962	0.044	-22.083	<0.001
Drank in the last 7 days: Yes	-	-	-	-
Drank in the last 7 days: No	-0.560	0.028	-20.306	<0.001
Drink amount: Hardly at all	-	-	-	-
Drink amount: A little	1.165	0.024	48.129	<0.001
Drink amount: A moderate amount	1.803	0.028	64.192	<0.001
Drink amount: Quite a lot	2.164	0.050	42.858	<0.001
Drink amount: Heavily	2.392	0.171	14.015	<0.001
Number of weekly drinking days	0.202	0.006	35.897	<0.001
Total units on heaviest drinking day in the previous week	0.051	0.003	15.957	<0.001

Model details	
Number of observations	13,615
Residual SE	1.035
R^2	0.633
Adjusted R^2	0.633

To assign each individual in the microsimulation a value for average units of alcohol consumed per week for each year after 2001 we used an imputation method based on matching individuals in the microsimulation and survey data by age, sex, and economic status. This procedure matched individuals in the microsimulation with individuals in the survey data from years 2002-2019. However, given that there were over 4 million individuals in the microsimulation and far less individuals in the survey data (approximately 15,000 respondents per year) it was not possible to assign each individual a new estimated units per week. Instead, a representative sample of 1,250 individuals obtained from the microsimulation was used and matched to individuals in the survey data.

We did not attempt to model life histories of drinking for each individual, the drinking of an individual at time t was independent of their drinking at t-1. Drinking history was not a predictor of future drinking as all alcohol consumption data used in the microsimulation was inputted

from another data source, as the survey data used to generate the microsimulation did not collect information about alcohol consumption. Therefore, the simplest way to input alcohol consumption data into the microsimulation was by matching individuals based on demographic information with individuals in another survey (the GHS and SHeS). Using the alcohol consumption inputted from the previous survey year to adjust the new value for alcohol consumption in the following year could heavily bias subsequent alcohol consumption and as a result may not be reflective of patterns of alcohol consumption at the population level from 2001-2019.

A very small number of individuals in the microsimulation did not match to any individual in the survey data based on age, sex and economic status groups (*n*=1 in 2002, *n*=2 in 2003-2011 and *n*=36 in 2012-2019). For the individuals with missing data in 2002 and 2003-2011 we used the value for alcohol consumption assigned to them from the survey data for the previous year, with the assumption that their drinking was relatively stable over time (253). For those with missing data from 2012-2019 we calculated an average units per week for the previous years we did have data to estimate drinking in future years.

Given noisy estimates of alcohol consumption (see Figures 8.1 and 8.2), we applied a 3-year window to each individual's alcohol consumption (excluding baseline). Therefore, alcohol consumption only changes in the model at 3-year time points: 2002, 2005, 2008, 2011, 2014 and 2017. The average is calculated from that year and the two years following (e.g., 2002 consumption is an average of 2002-2004 drinking). This windowing approach greatly reduced the noise observed in the simulated data (see Figures 8.3 and 8.4). The simulated estimates of alcohol consumption also accurately reflect differences in alcohol consumption by sex; males drink more than females (254) and deprivation quintile; those in the most deprived quintile (quintile 1) drink the same or less alcohol on average compared to the least deprived quintile (quintile 5) (16,41). It should be noted that Figure 8.3 and Figure 8.4 are not presented on a linear x-axis scale as the data points used to generate the figures were from 2001, 2002, 2005, 2008, 2011, 2014 and 2017.

Figure 8-1: *Simulated Average Units of Alcohol Consumed per week by sex.*

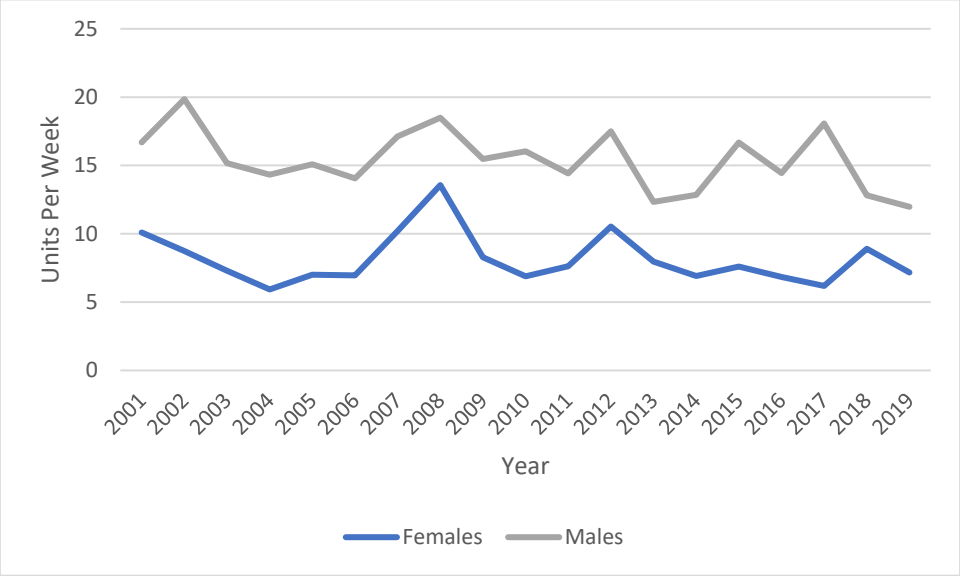


Figure 8-2: *Simulated Average Units of Alcohol Consumed per week by SIMD Quintile.*

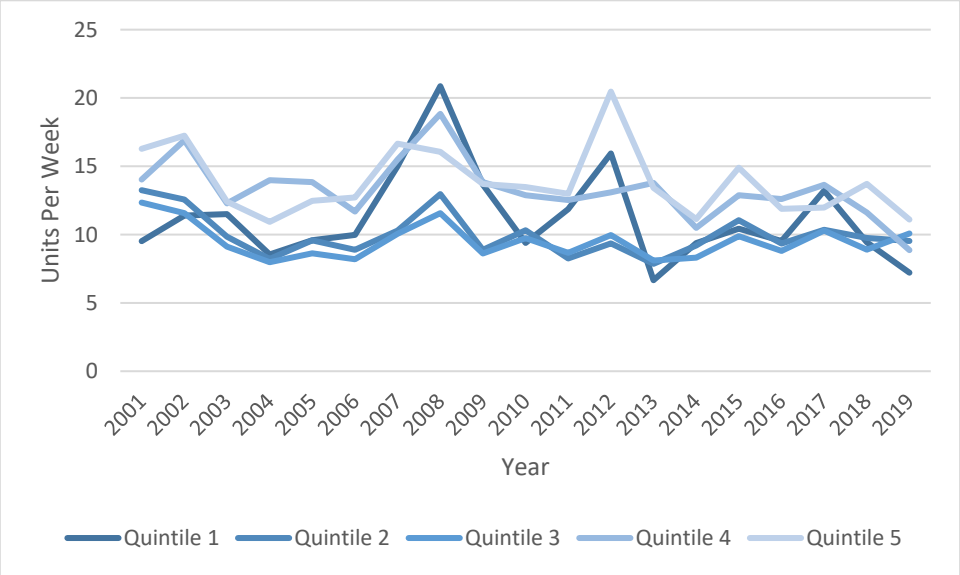


Figure 8-3: *Windowed Simulated Average Units of Alcohol Consumed per week by sex.*

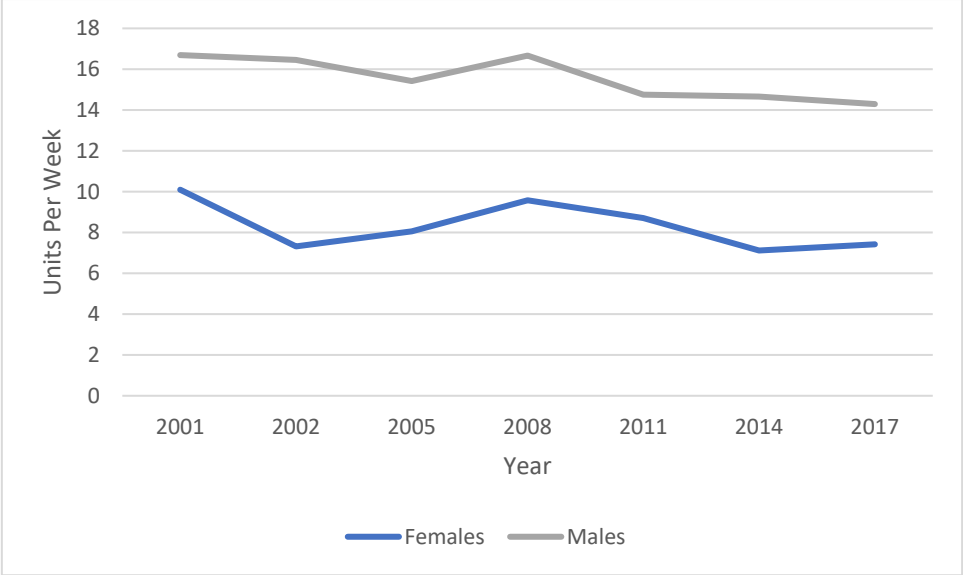
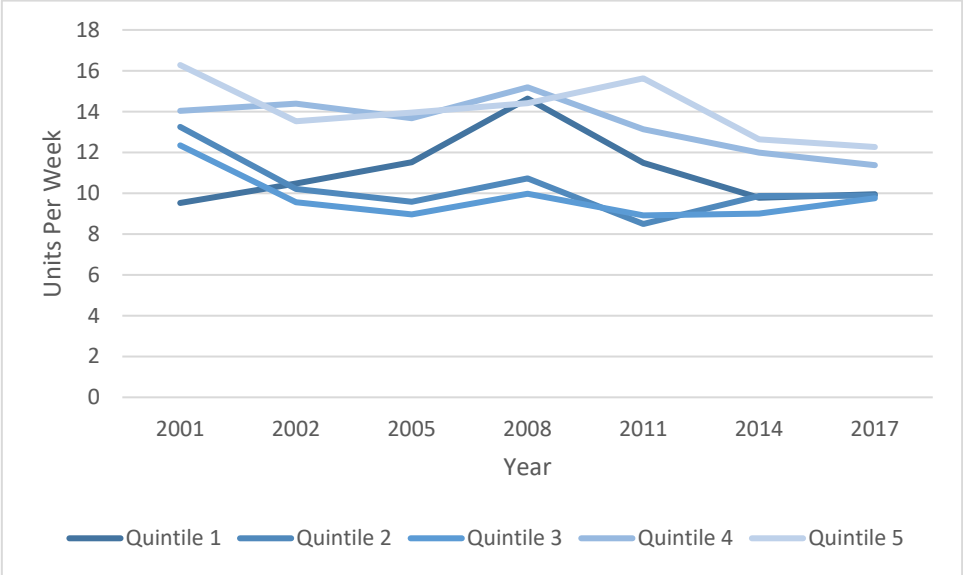


Figure 8-4: *Windowed Simulated Average Units of Alcohol Consumed per week by SIMD Quintile.*



8.7 Creating Fundamental resources described in Fundamental Cause Theory

The BHPS collects information on a range of topics including demographic, social, health, economic and behavioural questions. The particular variables that make this survey suitable to support operationalization of Fundamental Cause Theory were equivalized net household

income, equivalized real household annual income, level of qualification, social class grade, whether respondents had managerial experience, and questions that reflected the ability to achieve your goals.

Table 8.9 presents the data for these variables for each SIMD quintile from the microsimulation model. Similar to the demographic data shown in Table 8.6 there were clear social gradients for each of socioeconomic variables recorded in the BHPS. In terms of social class grade, a greater proportion of those living the least deprived quintile held professional (4.8%) or managerial & technical occupations (26.2%) compared to those living in the most deprived quintile (3.7% and 21.0% respectively). While a greater proportion of those living in the most deprived quintile held unskilled occupations (10.4%) compared to those living in the least deprived (7.4%). Similarly, a greater proportion of those living in the least deprived quintile held manager or supervisor positions (cumulatively 31.5%) compared to those living in the most deprived quintile (22.3%). As expected both annual and weekly average household income was higher in the least deprived quintile (£25,112.24, £299.73), while the households in the most deprived quintile had the lowest average income (£21,088.54, £248.97). A greater proportion of those in the least deprived quintile were able to pay for holidays (75.3%), buy new clothes (93.6%), owned 3 or more cars (7.7%), were living comfortably (29.1%), did not experience any problems overcoming difficulties (33.5%) and felt more capable of making decisions (12.6%) compared to those in all other quintiles.

Table 8-9: *Descriptive data of the socioeconomic variables recorded in the BHPS split by SIMD quintile.*

	Quintile 1 (*n*=814,479)	**Quintile 2** (*n*=825,230)	**Quintile 3** (*n*=811,301)	**Quintile 4** (*n*=808,498)	**Quintile 5** (*n*=830,438)
Social Class Grade					
Professional	3.7%	3.9%	4.1%	4.4%	4.8%
Managerial & technical	21.0%	22.2%	23.7%	24.9%	26.2%
Skilled non-manual	30.8%	30.2%	29.3%	28.7%	28.4%
Skilled manual	19.2%	19.5%	19.6%	19.6%	19.2%
Partly skilled	15.3%	14.9%	14.6%	14.3%	14.0%
Unskilled	10.1%	9.3%	8.6%	8.0%	7.4%

Managerial Duties					
Manager	10.0%	12.0%	13.5%	14.8%	16.1%
Foreman / supervisor	12.3%	13.7%	14.5%	15.0%	15.4%
Not manager / supervisor	77.6%	74.3%	72.0%	70.1%	68.5%
Equivalised Net Household Income (Mean, sd)	£248.97 (160.79)	£268.14 (170.99)	£279.63 (177.34)	£290.13 (182.68)	£299.73 (188.94)
Equivalised Real Household Annual Income (Mean, sd)	£21,089.54 (14186.28)	£22,612.47 (15195.56)	£23,518.42 (15838.40)	£24,384.89 (16493.33)	£25,112.24 (17071.12)
Ability to pay for a holiday					
Yes	61.1%	67.3%	70.6%	73.1%	75.3%
No	38.9%	32.7%	29.4%	26.9%	24.7%
Ability to pay for new clothes					
Yes	90.1%	91.7%	92.6%	93.1%	93.6%
No	9.9%	8.3%	7.4%	6.9%	6.4%
Private car use					
3+	5.4%	6.2%	6.8%	7.3%	7.7%
Two	31.9%	22.3%	25.1%	27.3%	29.3%
One	43.8%	44.7%	44.7%	44.7%	44.6%
None	31.9%	26.7%	23.4%	20.7%	18.4%
Financial Situation					
Living comfortably	21.1%	24.3%	26.3%	27.8%	29.1%
Doing alright	33.5%	35.3%	35.8%	36.4%	36.9%
Just about getting by	32.6%	30.1%	28.7%	27.6%	26.5%
Finding it quite difficult	8.4%	7.0%	6.4%	6.0%	5.6%
Finding it very difficult	4.5%	3.3%	2.7%	2.3%	2.0%
Problems overcoming difficulties					
Not at all	30.5%	32.0%	32.6%	33.0%	33.5%
No more than usual	52.9%	52.7%	52.8%	52.7%	52.7%
Rather more	13.2%	12.5%	12.1%	12.0%	11.8%
Much more	3.4%	2.8%	2.5%	2.3%	2.0%
Capable of making decisions					
More so than usual	11.8%	12.1%	12.1%	12.1%	12.6%

Same as usual	76.1%	77.0%	77.6%	78.0%	77.9%
Less so than usual	10.1%	9.3%	8.8%	8.5%	8.3%
Much less capable	2.0%	1.7%	1.5%	1.4%	1.2%

**Note Quintile 1 is the most deprived and Quintile 5 is the least deprived.*

Note that equivalised net household income represents weekly income after deductions for income tax and national insurance contributions, while equivalised real household income is the annual income after adjusting for inflation. Both measures of income are equivalised and therefore account for the household's size and composition. In terms of the wording of the levels under the variable 'Capable of making decisions', it is not clear the BHPS what is meant by the term 'usual'. However, it can be assumed to mean compared to how they typically might feel when making decisions compared to how they feel more recently at the time of data collection.

We opted to clean and construct these variables which represent fundamental resources so they could be easily implemented in an ABM. Highest educational qualification was used to represent the fundamental resource knowledge. We transformed this ordinal variable (1 = no qualifications, to 5 = group 4; degree level education) into a continuous variable bounded from 0 to 1 whereby 1 = 0.2, 2 = 0.4 etc. To construct three variables to represent power, prestige and money we carried out exploratory factor analysis (EFA) on a set of relevant variables available in the BHPS 2001 (see Table 8.10). Prior to conducting the EFA we hypothesised that the equivalised net household income and equivalized real household annual income variables would likely represent the FCT construct money, and that occupation would represent prestige given that different occupations are viewed as more or less prestigious as occupation is an indicator of social status (255). Additionally, whether individuals experience problems overcoming difficulties or are capable of making decision was also expected to be potential proxies for power, in terms of power as empowerment (256). However, whether the other included socioeconomic variables would represent prestige, power or money was less clear and therefore EFA was conducted.

Table 8-10: *Factor Loadings for Exploratory Factor Analysis used to create latent variables for fundamental resources.*

	F1: Prestige	F2: Money	F3: Power 1	F4: Power 2
Social Class Grade	0.82			
Occupation	0.94			
Managerial Duties	0.4			
Equivalised Net Household Income		0.96		
Equivalised Real Household Annual Income		0.81		
Ability to pay for a holiday			0.69	
Ability to buy new clothes			0.32	
Private car use			0.35	
Financial Situation			0.41	
Problems overcoming difficulties				0.88
Capable of making decisions				0.37

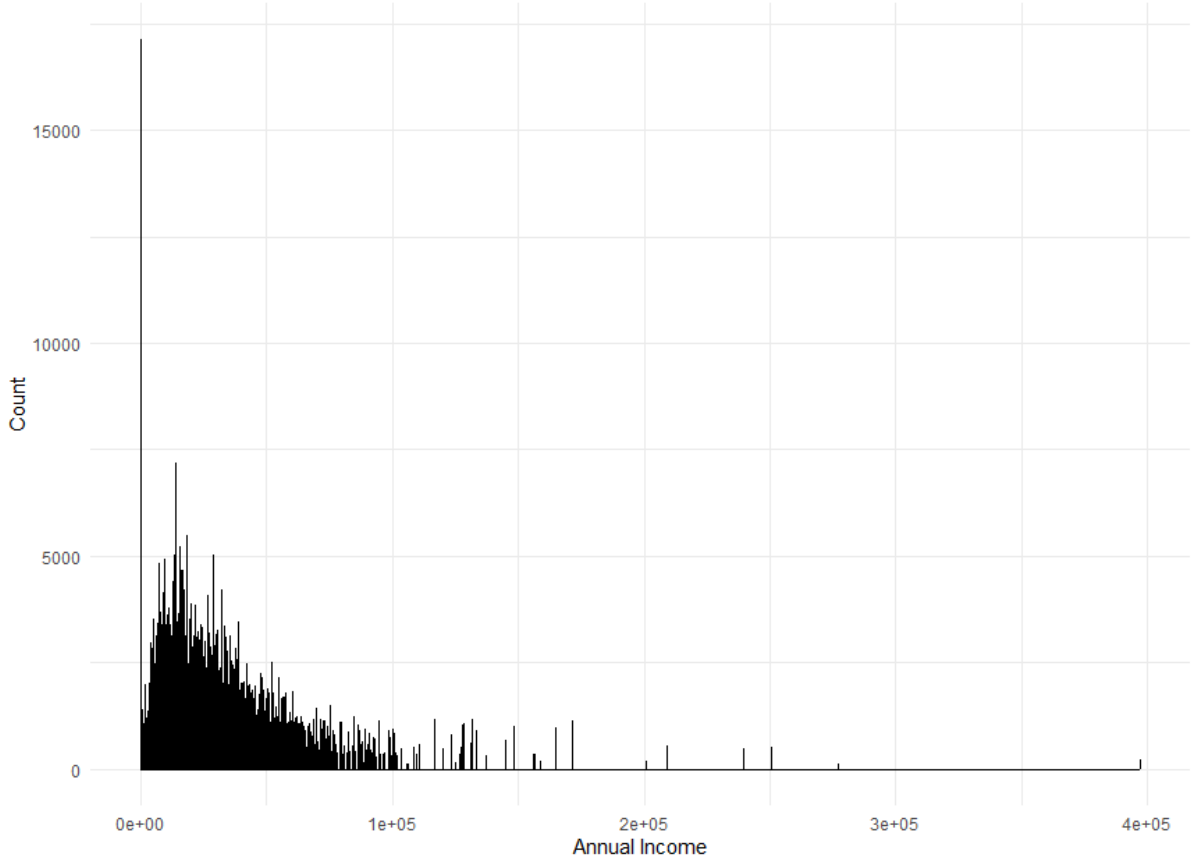
Df (24), X^2 = 71567.28, p <0.001.

**Note:* factors were produced as 1-4 and names were attached which matched each fundamental resource.*

The EFA model provided a four-factor solution, which was selected based on an eigenvalue threshold of 0.3. Factor 1 consisted of a measure of social class, occupation and whether the individual had managerial duties recently. We interpreted this factor to represent the fundamental resource prestige and therefore labelled it in Table 8.10 accordingly. In FCT prestige is associated with an individual's social standing in the eyes of others (96), and certain occupations are seen to be more prestigious (255). Factor 2 consisted of the variables equivalized net household income and equivalized real household annual income and therefore represents monetary resource. Factor 3 consisted of variables associated with being financially able to achieve your goals (e.g., whether you can afford to pay for holidays when you want to take them) and therefore we interpreted this factor to be a sub-component of the resource power. Factor 4 consisted of variables which represented whether you are psychologically capable of achieving your goals (e.g., do you feel capable of making your own decisions), and therefore we interpreted this factor to represent another sub-component of the resource power. Each latent variable was created by simply summing the component variables (e.g., Prestige was calculated by summing the variables Social Class Grade, Occupation and Managerial Duties) and then the resulting aggregated latent variable was normalised to values between 0 and 1 for ease of use in the ABM. The power latent variable was created by

aggregating both variables in Factor 3 and 4, before normalizing on a scale from 0 and 1. As a result all variables used to construct the latent factors were given equal weighting. Factor variables were transformed into ordinal variables for example for the variable managerial duties Not a manager or supervisor was coded as 1, Foreman or Supervisor was coded as 2 and Manager was coded as 3. Therefore, the higher the score for these variables the more resources they possessed in the respective latent variable. The factor variables transformed were: Social Class Grade, Managerial Duties, Ability to pay for a holiday, Ability to buy new clothes, Private car use, Financial Situation, Problems overcoming difficulties and Capable of making decisions. While there were outliers in the monetary variables (see Figure 8.5), the impact of normalising this variable to values between 0 and 1 was not a concern. In the ABM it is important to capture the difference in available resources between individual agents and those with an annual household income of above £200,000 will have far greater monetary resource.

Figure 8-5: *Household Annual Income in the Scottish Microsimulation Model.*



8.8 Strengths and Limitations of the Microsimulation

A key strength of the microsimulation model described in this chapter is that it was internally and externally validated and therefore we can be confident the model is representative of the population of Scotland in 2001. Particularly by observing a strong relationship between deprivation and income, and the socioeconomically patterned demographic data for economic activity and education indicates that the microsimulation model has captured differences between deprivation quintiles which was essential to explore socioeconomic inequalities in alcohol harm in the ABM described in chapter 9. Another strength of using microsimulation modelling to construct a representative population of Scotland in 2001 was that the method allows the synthesis of data from a range of data sources. Given that a data set in the Scottish context does not currently exist which captures both alcohol consumption and variables that could be used to represent fundamental resources described in FCT this was essential to construct a population that could be implemented in an ABM that uses FCT to explain to AHP.

The variables used to represent fundamental resources where the best available socioeconomic variables from the data sets on the UK data service for the year 2001. The British Household Panel Survey not only collected objective measures of socioeconomic position (e.g., annual household income, and occupation), but also more subjective measures such as how people viewed their financial situation and whether they felt like they were capable of making decisions. The variables identified are likely good proxies for the resources: knowledge, prestige and money. However, a limitation of the microsimulation model is its ability to represent the fundamental resource power.

In a recent paper McCartney and colleagues (256) discuss the use of FCT to explain health inequalities with a particular focus on how to conceptualise power. The paper starts by identifying that there are two contemporary schools of thought as to how power should be defined, the first is power as domination (power over) and the second is power as empowerment (power to or with) (256). It is clear that the operationalisation of power in the

microsimulation captures the latter definition of power. The variables which measure individual's ability to pay for holidays, ability to pay for new clothes, their subjective opinion on their financial situation and how many privately-owned cars they can use all indicate a measure of the power to achieve their goals, specifically a financial capability. While the variables which measure whether an individual experiences problems' overcoming difficulties and feels capable of making their own decisions reflects the psychological power to achieve their goals. However, as identified by McCartney and colleagues operationalizing power is more complex than just capturing an individual's power to achieve their goals. Rather they situate power as the key fundamental cause and outline several additional sources of power including economic, knowledge, culture and belief, collective organisations, the state and positional power (e.g., hierarchies and networks) (256). While to an extent economic and knowledge sources of power are captured by the other fundamental resources' money and knowledge, the current microsimulation model lacks variables that can capture other sources of power outlined in the recent paper by McCartney and colleagues. This suggests that the fundamental resource power may not be adequately captured by the current microsimulation model and therefore is a critical limitation of the model which may impact the ABM results described in chapter 9.

8.9 Summary

To summarise, this chapter has presented the process of generating a synthetic population of Scotland in 2001. This population was required to populate the ABM which will be presented in the next chapter. Results from this microsimulation suggest that the population accurately captures the Scottish population in 2001 based on both internal and external tests of validation. Given that no single data source had information on both alcohol consumption and variables to reflect resources described in FCT, we also estimated average units of alcohol consumed per week by matching a representative sample of the microsimulation to individuals in the GHS and SHeS. The estimations of alcohol consumption generally reflect expected trends in alcohol consumption split by both sex and deprivation quintile. The best available

socioeconomic variables were used to capture the resources described in FCT. While knowledge, prestige and money were relatively easy to capture using the available data, it was only possible to represent power as power to achieve goals which is a potential limitation of the microsimulation model.

Chapter 9 Using Agent-based Modelling to understand the causes of the Alcohol Harm Paradox: A Scottish case study investigating Fundamental Cause Theory.

9.1 Chapter Overview

This chapter presents a paper which describes an ABM developed to test whether FCT can explain inequalities in alcohol-specific mortality in Scotland. The chapter first provides background on the AHP and FCT, and situates agent-based modelling as a method that can test mechanisms specified in theory. The chapter then describes in detail the process of model development and implementation. Results comparing the model outputs to the target data and demonstrating the functionality of the model are also presented. The chapter is concluded by a discussion of ways in which model implementation could be improved and suggestions for future research to address these implementation issues or to look to other explanations to attempt to explain the AHP.

9.2 Abstract

Introduction: The Alcohol Harm Paradox is the consistent finding that disadvantaged people suffer from alcohol-related harms disproportionate to their drinking. Health inequality theories offer a new perspective to understand the underlying causal mechanisms. This paper presents an agent-based model simulating Fundamental Cause Theory which posits that the unequal distribution of fundamental resources (power, money, prestige, knowledge and social connections) drives health inequality. We explore whether this model can reproduce historical patterns of inequality in alcohol-related mortality in Scotland.

Methods: Individuals in the model were from a Scottish population microsimulation informed by the British Household Panel Survey 2001 which contains socioeconomic variables used to represent power, money, prestige and knowledge. Individuals were initialized in a neighbourhood based on SIMD quintile with a social network determined by demographic characteristics, spatial proximity and drinking status. Communication institutions (e.g. the media) communicate risk, prevention or treatment information to individuals. Based on a resource threshold, individuals can deploy fundamental resources to adapt to this information. The number of failures versus successes to adapt was used to adjust the baseline risk of alcohol-related mortality from alcohol consumption. Time series data from the model was compared to observed alcohol-specific mortality data.

Results: The model could not reproduce the socioeconomic gradient in alcohol-specific deaths in Scotland from 2001-2014. The calibration procedure did not identify a parameter set that could produce model outputs that closely match the target data. The model underestimated deaths for the most deprived quintile (SIMD quintile 1) and overestimated deaths for the least deprived quintiles (SIMD quintiles 4 and 5). Despite this finding, the model functioned as intended – the most deprived quintile experienced a greater number of failures to adapt and fewer successes, while the least deprived quintile experienced a far greater number of successes and fewer failures.

Conclusions: This case study presented an example of how an agent-based model could be applied to capture complex mechanisms which may generate the AHP. The model could not reproduce trends in alcohol specific deaths by SIMD quintile in Scotland between 2001 and 2014. One possibility is that FCT cannot sufficiently explain inequalities in alcohol specific deaths. Alternatively, there were limitations with model implementation, particularly that fundamental resources may not be adequately represented and lack of a model burn in period, that could explain why the model could not reproduce the paradox.

Key words: Alcohol, Health Inequality, Agent-based Modelling

9.3 Introduction

Despite reporting the same or lower average levels of alcohol consumption, disadvantaged groups suffer higher rates of alcohol-related hospital admissions and deaths compared with advantaged groups (16,41). This consistent finding termed the 'Alcohol Harm Paradox' (AHP) is evidenced in several countries including Scotland (16), Australia (17), the Netherlands (19) and Finland (9). A recent review identified that existing research has focused on understanding the contribution of risk behaviours (e.g., binge drinking, smoking) to this phenomenon (164). However, evidence suggests that these risk behaviours can only partially explain the AHP. Despite this, other potential explanations, particularly those associated with the lived experience of disadvantage or societal-level factors, have been mainly theoretical.

The AHP is clearly an issue of health inequality. Alcohol-related harms contribute significantly to inequalities in life expectancy between socioeconomic groups (49). However, research has yet to apply what is known about the causes of health inequalities to this phenomenon. Recently, there has been an increased interest in the field to shift the focus away from individual-level behaviour with calls to explore contextual factors (e.g., the characteristics of drinking environments) (110) and to explicitly draw on theories of health inequality including fundamental cause theory (FCT) (257).

FCT shifts the focus from individual-level risk behaviours as the cause of health inequalities to a focus on the context; what puts people "at risk of risks" (96). Central to this theory are the role of fundamental resources defined as money, knowledge, power, prestige and social connections (96,97). FCT suggests that the distribution of fundamental resources based on the social stratification of society impacts whether individuals can adapt to the introduction of new disease, risks, prevention information or treatments (97). More advantaged individuals have increased access to fundamental flexible resources and as a result deploy them to avoid risks, reduce the consequences of disease and uptake available treatment. On the other hand, these resources are not readily available to disadvantaged individuals.

Previous research has used FCT to understand inequalities in lung cancer. A comparative case study predicted that as lung cancer becomes more preventable, due to new information connecting smoking to the disease, more advantaged individuals with greater access to resources disproportionately benefit, thus increasing health inequalities (181). Contrastingly, for pancreatic cancer, a disease that lacks major prevention or treatment information, there is no mortality advantage associated with socioeconomic group (181). Alcohol-related harms (e.g., liver disease) are largely preventable, therefore FCT could explain differences in alcohol-related harm between socioeconomic groups.

The relationships present in FCT are complex, dynamic in nature and exist on multiple societal levels (e.g., individual, and structural levels). In FCT new risk, prevention or treatment information is discoverable by individuals through institutions (e.g., mass media) or word of mouth, and upon discovery of this information individuals can deploy the resources available to them to act. These relationships are difficult to capture with the traditional risk factor approach to epidemiology (13). However, a mechanism-based approach which represents "entities" and the "activities" entities engage in to bring about a particular outcome (53) would be able to capture these complex relationships.

Computer simulations methods are a good candidate to test mechanisms and have become increasingly attractive in public health. A review of simulation models in the context of health inequality found that these methods have the potential to enhance our understanding of socioeconomic inequalities in health (191). In particular agent-based models (ABMs) can flexibly model the multilevel, reciprocal and indirect effects of socioeconomic inequalities (191). Comprised of agents (e.g., individuals) and their interactions with each other and the environment (192), ABMs can test mechanisms specified in theory (54). An additional benefit of ABMs is that they enable evidence to be synthesized from multiple sources to address research questions and inform decision making (195).

This study aims to address two research gaps on the causes of the AHP: 1) by explicitly drawing on health inequality theory, specifically FCT, to explain inequalities in alcohol harm

and 2) to adopt a novel methodological approach, ABM, to capture the complex mechanisms described in FCT. To our knowledge this is the first study to use ABM methodology to understand the AHP, and the first to explicitly apply health inequality theory.

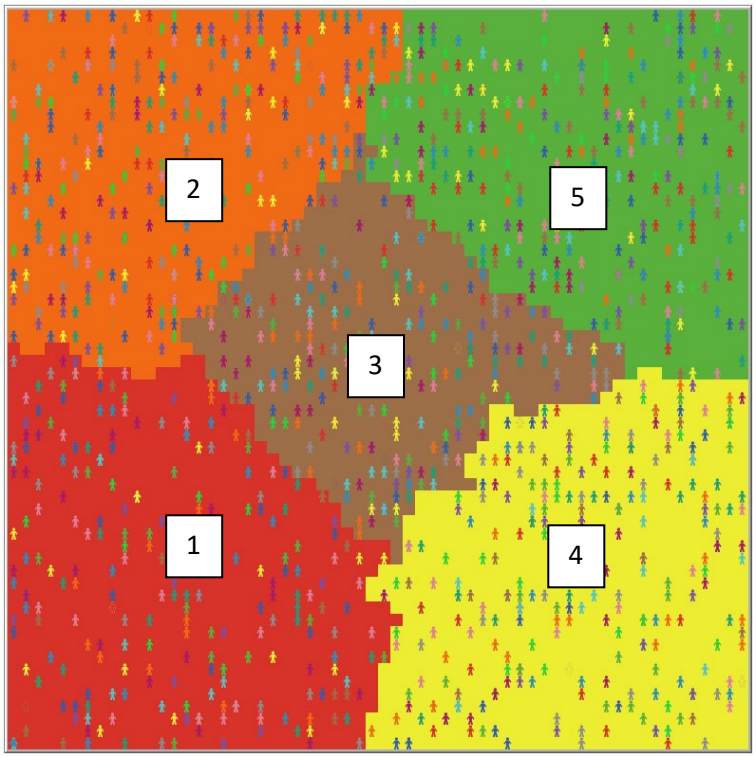
9.4 Method

We developed an agent-based model (ABM) which simulates the mechanisms described in Fundamental Cause Theory. We chose Scotland as the setting for this model as the AHP is evident in this context (16) and data was freely available via the UK Data Service and National Records for Scotland. A description and rationale of each modelled component has been written according to the Overview, Design concepts and Details (ODD) protocol for consistent and logical reporting of individual and agent-based models (258) (Appendix C).

9.4.1 Model Initialization

The physical environment was represented using a 60x60-cell grid, split into five communities, to represent the most deprived to least deprived quintiles according to the Scottish Index of Multiple Deprivation (SIMD) shown in Figure 9.1. SIMD is a relative measure of area-level deprivation which consists of seven domains: income, employment, health, education, geographic access to services, housing and crime (248). SIMD splits Scotland into 6,505 Data Zones and based on the included domains ranks these Data Zones from most to least deprived. In this model we represent the quintiles of this rank measure in physical space where quintile 1 is the most deprived Data Zones and 5 is least deprived Data Zones (Figure 9.1).

Figure 9-1: *Visual Representation of the Model Environment.*



The model was populated with adults initialized from a static microsimulation of the Scottish population in 2001 (more details of the microsimulation model can be found in chapter 8). The microsimulation uses data from the Scottish Census (259) and the British Household Panel Survey (260) and is comprised of a population aged 16 to 95, and updates each year over time to add new individuals aged 16. Individuals were entered in the community that matched their own deprivation quintile sourced from the microsimulation model. Individuals were initialized with age, sex, education group, drinking status, average units of alcohol consumed per week, and variables which represented fundamental resources: power, prestige, money and knowledge. Latent variables were constructed for power, prestige and money using exploratory factor analysis, before normalizing each latent variable on a scale of 0-1 (see Chapter 8). The variable knowledge was represented using the highest educational qualification which was also normalized on a scale of 0-1.

Individuals were connected to other individuals in a social network. Connections were created probabilistically based on similarities in age, sex, education group, deprivation quintile and drinking status. This method for generating social networks has been used to successfully

generate realistic social networks in several existing ABMs on the topic of alcohol (211,212). Given that there are no existing studies which map out the social networks of adults in Scotland using empirical data it was necessary to use this method of social network generation to simulate a realistic social network for this population. The method used for social network generation was also relatively simple to implement and therefore did not require a lot of computation power or time to generate which was particularly important given the time constraints of the PhD thesis. To create the network, individuals were randomly assigned a number of target connections, with an average of 3 connections per individual. Social networks were generated at baseline, and individuals could not create new connections or break connections over the course of the simulation. Connections were only broken in the instance of death and formed by new individuals that migrated into the model.

9.4.2 Conceptual Design

To translate the mechanisms described in FCT we used the Mechanism-based Social Systems Modelling (MBSSM) framework (160). The MBSSM framework details two types of entity; macro-entities (which are social entities or phenomena such as institutions or regulations) and micro-entities (such as individuals or households, that can interact with each other and the environment). The MBSSM framework also outlines three types of mechanism: situational mechanisms (the impact of the macro-level on micro-entities), action mechanisms (actions performed by micro-entities as a result of their internal states), and transformational mechanisms (the collective impact of micro-entities on societal structures at the macro-level).

A simplified MBSSM diagram of FCT is shown in Figure 9.2. From the theory we identified two macro entities; the *environment* individuals live in and the *communicator* - an abstract entity that communicates and receives information from an exogenous source. We identified individuals as micro-level entities.

9.4.3 Communicator Entity and Behaviours

The macro-level agent which performed behaviours in this simulation was the communicator entity. This agent represents a societal institution (e.g., the mass media) that communicates

new information about the risks, treatment or knowledge of the causes and cures of alcohol-related disease to individuals in the simulation. Previous work has shown that television, radio and other media channels are used to deliver messages to reduce consumption and related harms (261). At each time step (1 week), the communicator assessed whether a new event had occurred of any type based on the probability of an event. The communicator then communicates this new information to a proportion of individuals in the simulation.

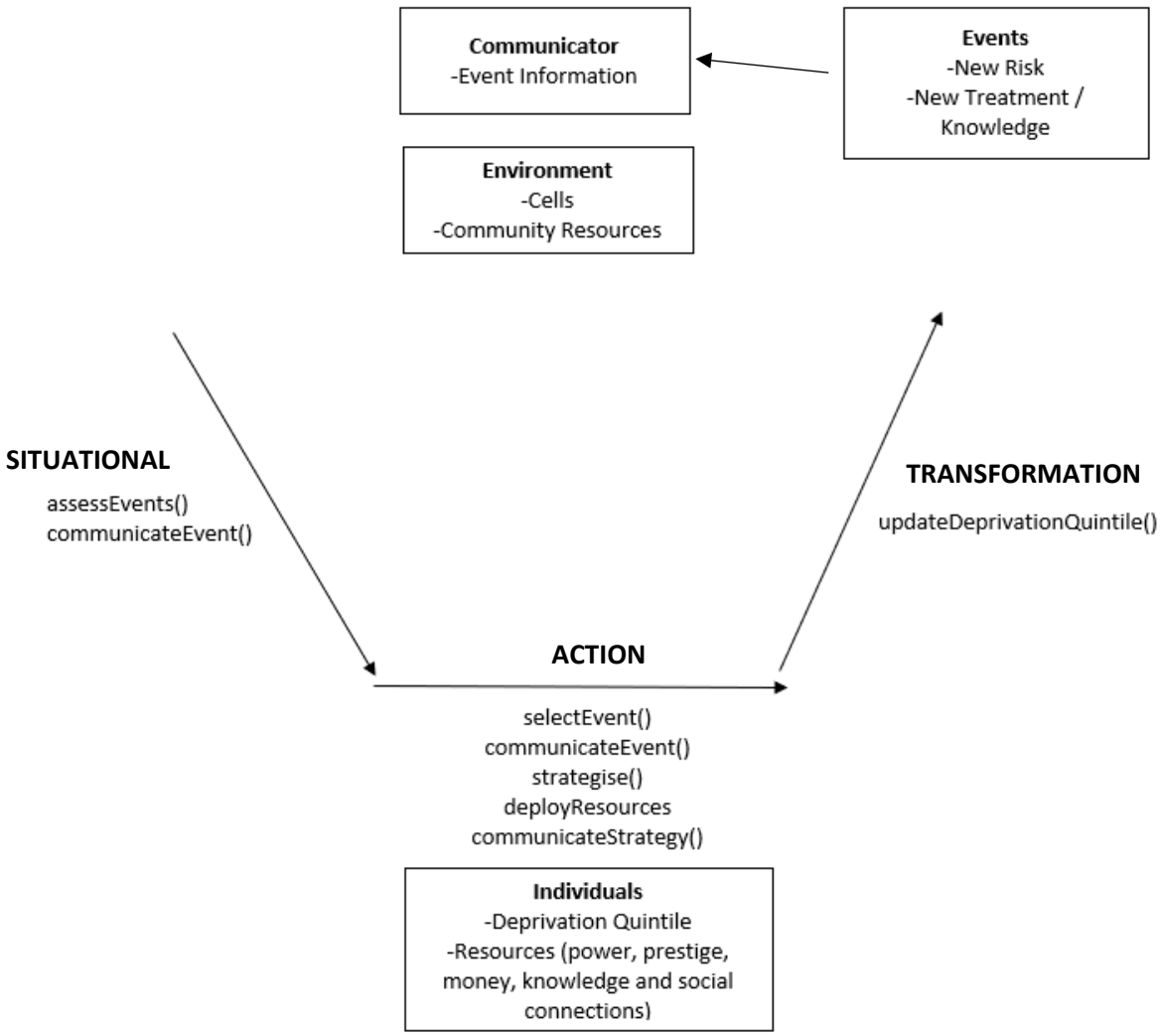
9.4.4 Individual Behaviour

At each time step individuals perform several behaviours. To illustrate these, we present a walk-through of two individual agents we call “Agent A” and “Agent B”. “Agent A” lives in the least deprived community and has a relatively large resource pool, while “Agent B” lives in the most deprived community and has few resources available to them. Both individuals receive information about alcohol harm from the communicator entity. “Agent A” and “Agent B” remember all the information they have been informed of (event list), information that they have already adapted their behaviour to (resolved events) and information that they have not adapted their behaviour to (unresolved events). Each tick[4] both agents can select a piece of information from their unresolved event list and attempt to adapt their behaviour. In the context of alcohol specific mortality these events represent public education campaigns, (e.g., the ‘Don’t let too much alcohol spoil a good night out’ campaign introduced in Scotland in 2003 (262)), or new available treatments, (e.g., the widespread introduction of alcohol brief interventions in primary care settings from 2008 onwards (263)). While it is simpler to think of the events that occur in the model as events that would moderate the relationship between alcohol consumption and alcohol specific death directly via changing consumption it is also possible to think of these events as moderating the effects of alcohol consumption by influencing harm via alcohol consumption or directly. For example, an event could be the opening of a new bar/restaurant that more affluent individuals would have the resources to be able to visit and therefore consume alcohol in a safer environment and with a meal which

[4] A tick is a time step and in the model each tick represents one week.

would reduce their risk of harm while more disadvantaged individual would not be able to access this environment given their resources. Equally, an event could be the availability of new housing in an advantaged area which more affluent individuals could choose to buy and improve their living conditions therefore improving their health directly, whereas more disadvantaged individuals with fewer resources would not be able to purchase new housing and move areas in the absence of the required resources. As events in this model are abstract they can represent any of the three types of event: events which change consumption, moderate consumption and directly impact harm.

Figure 9-2: *Fundamental Cause Theory within the MBSSM framework. Note that entities and their attributes appear in boxes and the situational, action and transformation mechanisms are indicated with ().*



In Figure 9.2 agents are represented in boxes as the communicator, individuals and to an extent the environment itself. The attributes belonging to these agents are listed in the boxes, the communicator contains event information, the environment contains cells and information on community resources, while individuals have the following attributes: deprivation quintile, power, prestige, money, knowledge and social connections. The methods are listed next to each arrow. The communicator agent can engage in two methods; assess whether an event has occurred and communicate that event to individuals. While the individuals have several methods including the selection of an event, communicating the event to their social connections, looking for a strategy to adapt to the event, deploying their own resources and communicating a strategy to their social connections. Individual agents can also move location and therefore can update their deprivation quintile accordingly.

To give an arbitrary example, both agents may select to adapt to information from the communicator that a new brief intervention is now available. Both agents then have the opportunity to relay this information to someone in their social network, based on the probability that they will communicate. Both “Agent A” and “Agent B” then check whether anyone in their social network has given them any tips about how to adapt their behaviour in line with this information. “Agent A” may have received information from a connection that a friend has adapted by seeking this treatment from a particular healthcare service. This information provides “Agent A” with a strategy to adapt to this information, while “Agent B” has not received any tips on how to adapt. Awareness of a strategy enhances the resources available to “Agent A” that are required to adapt to this information. Both agents then draw on their available resource pool, a summation of power, prestige, money and knowledge, to adapt and change their behaviour. “Agent A” meets the resource threshold for adapting to this information and successfully takes on the new behaviour and receives the new brief intervention. Successful adaptation costs “Agent A” a proportion of their available resources

but their knowledge resource increases. On the other hand, “Agent B” does not have adequate available resources and cannot successfully adapt their current behaviour, this unsuccessful adaptation is recorded and their resource pool remains the same. Successful adaptations will be termed successes and unsuccessful adaptations termed failures for the remainder of the paper.

Following a success “Agent A” can communicate the strategy they used to adapt to a friend in their social network, again this is based on the probability they will communicate.

It should be noted that intergenerational advantage nor history is not modelled in this version of the ABM. All resources were pooled to create the resource pool with equal weighting given that previous work has not investigated whether some fundamental resources are more important for adaptation than others.

9.4.5 Deprivation Swap

To capture the relative change in deprivation quintile over time throughout the simulation we implemented a pre-existing deprivation swap model (264). Annually, each individual has the opportunity to attempt to swap deprivation quintile and, if possible, commit to the swap. The existing model simulates the movement of individuals and therefore the change in SIMD quintile. However, as SIMD is a relative measure of deprivation these movements are required to be swap events, one individual must replace another in a different quintile. Based on the existing model young individuals aged 16-30 and in less deprived quintiles have the chance to move to more deprived areas, for example to continue education. While older adults ages 30-45, and in deprived areas can move to a less deprived area, for example to settle down and start a family. In the model individuals first assess whether they can attempt to move deprivation quintile based on their age and their current deprivation quintile. Individuals can attempt to swap to any other deprivation quintile, however this is controlled by probabilities for both attempting to swap and committing to a swap. Full details of this sub-model are found in Appendix C.

9.4.6 Alcohol Specific Mortality

The risk of alcohol-specific death was calculated annually. To calculate the absolute risk of death from alcohol consumption we adapted the method described by Meier and colleagues' (265). We assumed that the threshold for absolute risk of harm was 14 units per week for both males and females in line with current UK drinking guidelines (266). We used the following absolute risk function based on consumption (for which a slope is defined) and the threshold:

$$AR(c) = 0, \ \ if \ c < T$$

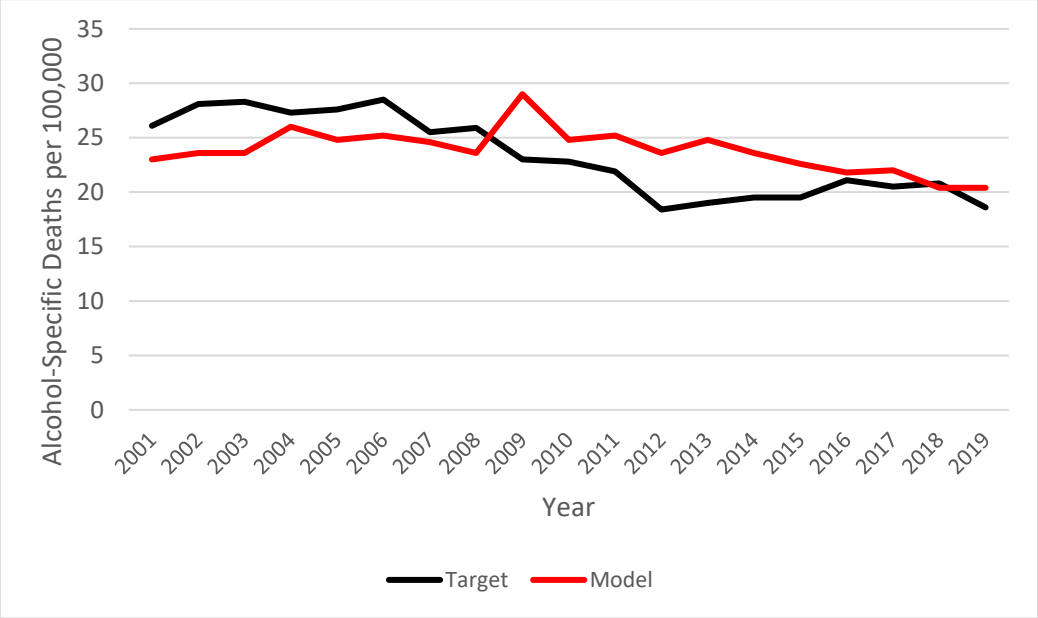
$$AR(c) = \ \beta(c - T), otherwise$$

**Where AR = absolute risk, c = alcohol consumption (units per week), T = threshold and* β *= slope parameter.*

Equation 9-1

Given biological differences between males and females that result in differences in vulnerability to the effects of alcohol (267,268), we defined two separate slope parameters – one for males and one for females. The absolute risk function was calibrated to population-level alcohol specific deaths in Scotland from 2001-2019 to obtain β slope parameters (see Appendix D for more details). Figure 9.3 shows the fit to the to the observed alcohol-specific deaths in Scotland from 2001-2019 from the best fitting model setting. The model slightly underestimates deaths from 2001-2008 and slightly overestimates deaths from 2009-2017. This coincides with the switch in data source used to estimate alcohol consumption and a spike in alcohol consumption observed in the data in 2008 (as shown in chapter 8, Figures 8.3 and 8.4). However, as this was the best model setting identified from 15,000 simulations and given time and resource constraints the resulting β beta slope parameter values were used in the ABM model calibration.

Figure 9-3: *The best calibrated model compared to the observed rate of alcohol specific deaths for the whole Scottish population per 100,000 from 2001-2019.*



To calculate the adjusted risk of alcohol specific death for each simulated individual the following equations were used:

$$RiskMod = \left(\frac{\sum failures}{\sum successes + failures}\right) + 0.5$$

Equation 9-2

$$RiskMod\beta = RiskMod^{\beta Mod}$$

Equation 9-3

$$AdjRisk = (AR\ x\ RiskMod\beta)$$

Equation 9-4

In equation 9.2 a risk modifier is first calculated by taking the number of failures and dividing by the sum of all successes and failures each individual has experienced in the simulation. This equation essentially creates a multiplier that is proportionate to the number of successes

versus failures for each individual in the simulation. For example, someone that experiences 0 successes and 10 failures would have a RiskMod of 1.5 (the multiplier is >1 and therefore risk of alcohol specific death increases) whereas someone that experiences 10 successes and 0 failures would have a RiskMod of 0.5 (the multiplier is <1 and therefore the risk of alcohol specific death decreases). To identify accurate estimates for adjusted risk, the risk modifier is exponentiated using βMod. By exponentiating the RiskMod value by βMod (a calibrated parameter) allows the model to generate different estimates for RiskModβ. A βMod value of 2 results in a much smaller range of values used to multiple absolute risk, while a βMod of 10 results in a much wider range of values. For example, someone who has experienced 10 failures and 0 failures will have their absolute risk adjusted by 2.25 when the βMod is 2, whereas they will have their absolute risk adjusted by 57.67 when the βMod is 10. The absolute risk previous calculated in equation 9.1 is then adjusted by multiplying it by the calculated RiskModβ to produce an adjusted risk (AdjRisk). A greater number of successes results in a reduced risk of alcohol-specific death, while a greater number of failures resulted in an increased risk. To illustrate how absolute risk is adjusted, we present a walk-through of two individual agents we call “Agent A” and “Agent B”. Agent A as before lives in the least deprived community and has successfully adapted to 9 events and failed to adapt to 1 event, while their absolute risk of alcohol specific death based on their alcohol consumption is 0.0270. Taking a βMod value of 5 (the mid-point of possible parameterisation values) the risk of alcohol specific death for Agent A following the equations above would be adjusted by a $RiskMod\beta$ of 0.0778 and therefore their risk of alcohol specific death would be reduced from 0.0270 to 0.0021. Agent B lives in the most deprived community and has only successfully adapted to 1 event and failed to adapt to 9 events, Agent B also has an absolute risk of alcohol specific death based on their alcohol consumption of 0.0270. However, based on a βMod value of 5, the risk of alcohol specific death for Agent B would be adjusted by a $RiskMod\beta$ of 5.3782 and therefore their risk of alcohol specific death would increase from 0.0270 to 0.1450.

9.4.7 Aging, mortality and annual transitions

Annually, individuals age 1 year and died from causes unrelated to alcohol based on the probabilities split by age and sex taken from Scottish Lifetables for the years 2001-2020 (269). For simplification an average probability across the time period for each age and sex group was used. Additionally, individuals aged 16-30 years could gain educational qualifications and as a result move up an education group and improve their knowledge. Educational transitions were based on the proportion of individuals over the age of 30 belonging to each education group in the 2001 baseline microsimulation.

Alcohol consumption, measured in average units per week was also updated annually. These values were parameterized from the General Household Survey for the years 2002-2006 (270), and from the Scottish Health Survey for the years 2008-2019 (271) based on data availability. Each individual in the microsimulation was given a value for average units per week for each year of the survey data by matching them to individuals from the surveys based on age group, sex and economic activity.

9.4.8 Implementation

This model was implemented in NetLogo version 6.2.0 (272). The model is run forward in time for 15 years for calibration (2001-2014), with 5 years of target data to be preserved for validation (2015-2019). Every model tick represents one simulated week. Every year, the risk of alcohol specific death is calculated for each individual as a function of their absolute risk based on their alcohol consumption adjusted by the success and failure outcomes from the FCT mechanisms. Summary statistics capturing the count of deaths split by SIMD quintile were recorded annually. Given the simulation time for each model run (up to approximately 3 minutes per run using a 32 core AMD Ryzen Threadripper 3970X processor), models were calibrated using 1,250 individuals sampled from the representative population of Scotland. All model results report the best calibrated settings for a random sample of 100,000 Scottish individuals.

9.4.9 Model functionality

To demonstrate the model's functionality the best fitting model setting was used to conduct subsequent model runs. Data pertaining to each individual's alcohol consumption, their successes and failures with regard to behavioural adaptation and the adjusted risk of alcohol-specific death was recorded.

9.4.10 Model Calibration

The model was calibrated using Latin hypercube sampling, which samples from the prior distributions of the unobserved parameters in the model (see Table 9.1). Implausibility values were calculated to identify parameter settings which match the outputs of the model with the observed alcohol specific death data (targets). Rejection sampling was also used to calculate approximate posterior distributions which describe the probability that each model parameter will take a particular value given the observed target data.

Table 9-1: *A description of unobserved parameters in the model.*

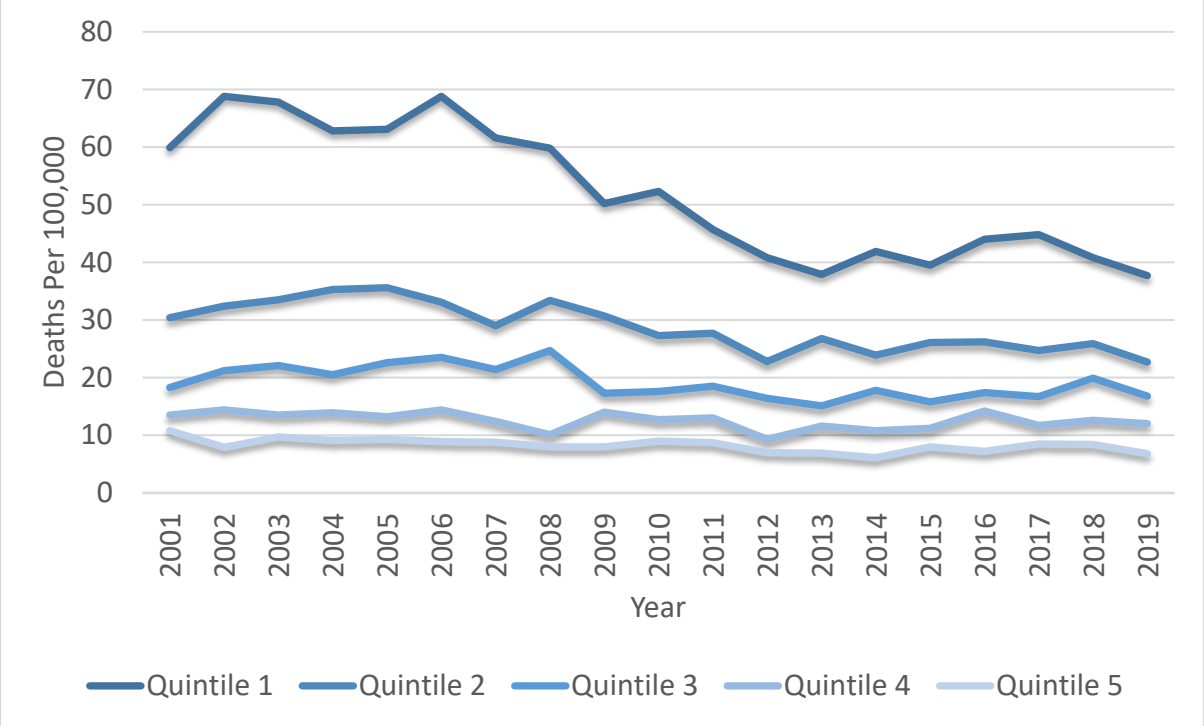
Model Parameter	Prior Distribution	Description
probabilityEvent	Uniform (0.01, 0.9)	The likelihood that an event (an instance of information about a new treatment, new prevention or risk information) in any given tick of the simulation.
%agentsKnowEvent	Uniform (0.1, 0.9)	The proportion of individual agents that are informed of the new event by the communicator entity.
adaptation Threshold	Uniform (0.1, 3.0)	The threshold that must be met by the individual agents' total resource pool to successfully adapt their behaviour to an event.
probabilityCommunicate	Uniform (0.1, 0.9)	The likelihood that individual agents will communicate information about an event or strategy to a social connection.
strategyMultiplier	Uniform (1.0, 3.0)	The number used to enhance the number of available resources to an individual agent, for the tick that it is applied.
knowledgeGain	Uniform (0.1, 0.9)	A value added to the individual agents existing knowledge in the instance where they successfully adapt to an event and is only applied if their knowledge has not already reached the maximum value of 1.
resourceDepletion	Uniform (0.1, 3.0)	The number subtracted from the total resource pool of individual agents when they successfully deploy resources to adapt their behaviour to an event.

βMod	Uniform (0.1, 10)	The value used to stabilise the risk modifier which is used to calculate the adjusted risk of alcohol-specific death based on whether individual agents have succeeded or failed to adapt their behaviour to an event.

9.4.11 Targets

Targets were provided by the National Records for Scotland for the years 2001-2019 and were age-standardised alcohol specific deaths split by SIMD quintile. Alcohol specific deaths were defined as "*deaths which are known to be direct consequences of alcohol misuse, meaning they are wholly attributable to alcohol misuse*" (273). An example of a wholly attributable cause is alcoholic liver disease. For each year there were five calibration targets given that targets were split by SIMD quintile (see Figure 9.4).

Figure 9-4: *Age-standardised alcohol specific deaths rates per 100,000 people, by IMD Quintile in Scotland from 2001-2019.*

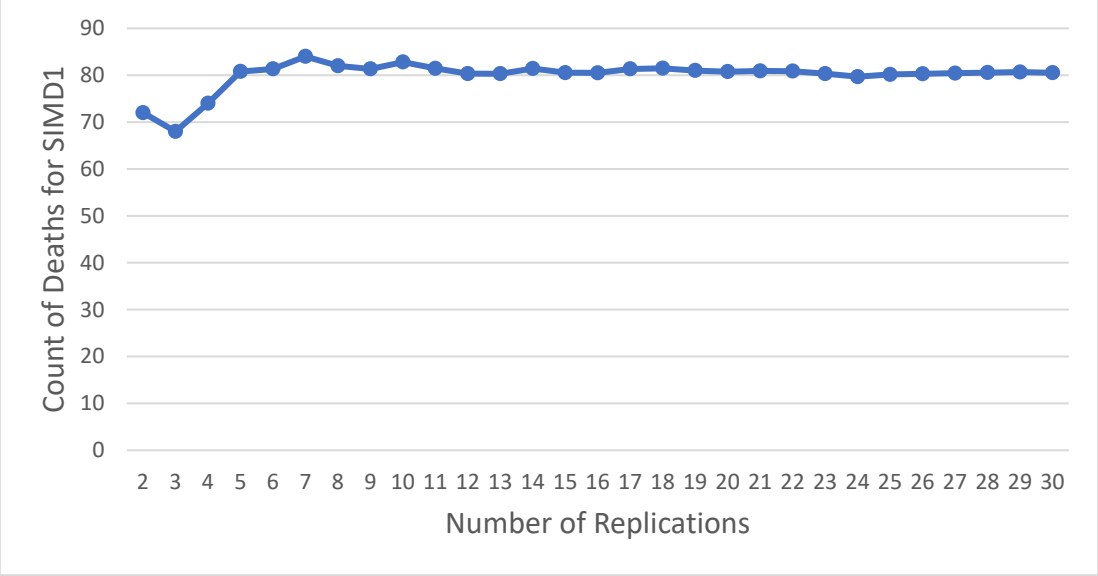


9.4.12 Procedure

The model representing the mechanisms described in FCT contained a total of 8 unobserved parameters (see Table 9.1). A Latin hypercube design was used to sample 10,000 parameter

settings from joint prior distributions using the nlrx R package (229). The number of model replications required was estimated by running one sample 100 times and plotting the stability of results by incrementally calculating the mean (the average model output from runs 1 and 2, the average model output from runs 1-3, the average model output from runs 1-4 and so on). Each sample was run five times to ensure stability in the model results (see Figure 9.5).

Figure 9-5: *Stability of Model Outputs across Replications; showing count of deaths for SIMD1 as an illustrative example.*



Implausibility values were calculated for each parameter setting using equation 9.5, which describes the difference between the model outputs and the target data (230). The implausibility value captures the difference between the simulated output ym^* and target data yt divided by the standard error of the model se_m and the standard error of the empirical target data se_t, for all targets k. The parameter setting that produces the lowest implausibility value represents the model setting that most closely fit the target data.

$$I_{[k]=} \frac{(ym[k]-yt\,[k])^2}{(se_m[k]^2)+(se_t[k]^2)}$$

Equation 9-5

Implausibility I is a metric which captures the distance from the model outputs to the target data by calculating the difference between the simulated output ym and target data yt

divided by the standard error of the model se_m and the standard error of the empirical target data se_t. Then the overall implausibility was taken to be the average implausibility over the k targets (see equation 6.2).

$$\frac{1}{K}\sum_{k=1}^{K} I[k]$$

Equation 9-6

Rejection sampling was also used to calculate approximate posterior distributions using the abc package in R (231). For this analysis a tolerance threshold of 0.01 was selected to estimate posterior distributions using the best fitting 1% of model simulations, equivalent to using the best 100 samples from 10,000 simulations.

9.5 Results

9.5.1 Testing Model Functionality

To gain a better understanding of the model results the best model setting was used to run additional simulations of the model to obtain data on alcohol consumption, successes and failures to adapt for each SIMD quintile. The average quantity of fundamental resources available to an individual in each SIMD quintile at baseline was also recorded. The values identified as the producing results closest to the target data for each parameter were as follows: probability event (0.33), % agents know event (0.51), adaptation threshold (0.35), probability communicate (0.53), strategy multiplier (1.04), knowledge gain (0.87), resource depletion (1.67) and beta modifier (9.19). This essentially means that for the model to produce estimates of alcohol-specific death which most closely matched the target data there was a 33% chance that an event would occur each week, 51% of people would then be informed of that event by the communicator entity, and there was a 53% chance that an individual would share information with another individual in their social network. To be able to adapt to the event individuals required a minimum of 0.35 in their resource pool and every time they adapted this cost them 1.67 of their total resources. When individuals successfully adapted

they gain 0.87 in knowledge, almost guaranteeing that every individual that had successfully adapted would have the maximum knowledge available in their resource pool, which was a value of 1. Knowing of a strategy to adapt did not increase the resource pool by much given the identified multiplier was 1.04, and finally the beta modifier was high (9.19) indicating a steep increase in risk given any increase in the number of failures experienced. Parameters were set to these values and the model was run five times from 2001-2014. All results were averaged across the five replications.

At baseline on average an individual in SIMD quintile 1 had 1.17 resources available (min = 0.2, max = 3.0), in SIMD quintile 2, 1.22 (min = 0.23, max = 2.98), in SIMD quintile 3, 1.28 (min = 0.24, max = 3.0), in SIMD quintile 4, 1.32 (min = 0.2, max = 2.87) and in SIMD quintile 5, 1.31 (min = 0.18, max = 2.93). Figure 9.6 shows the average units of alcohol consumed per week for each SIMD quintile. It is clear from these results that across the simulation period the trends in alcohol consumption are consistent with the AHP. The most deprived quintile (SIMD 1) had consistently lower levels of alcohol consumption compared to the least deprived quintile (SIMD 5), which had the highest levels of alcohol consumption.

Figure 9-6: *Average units per week of alcohol consumed across the best simulation split by SIMD quintile.*

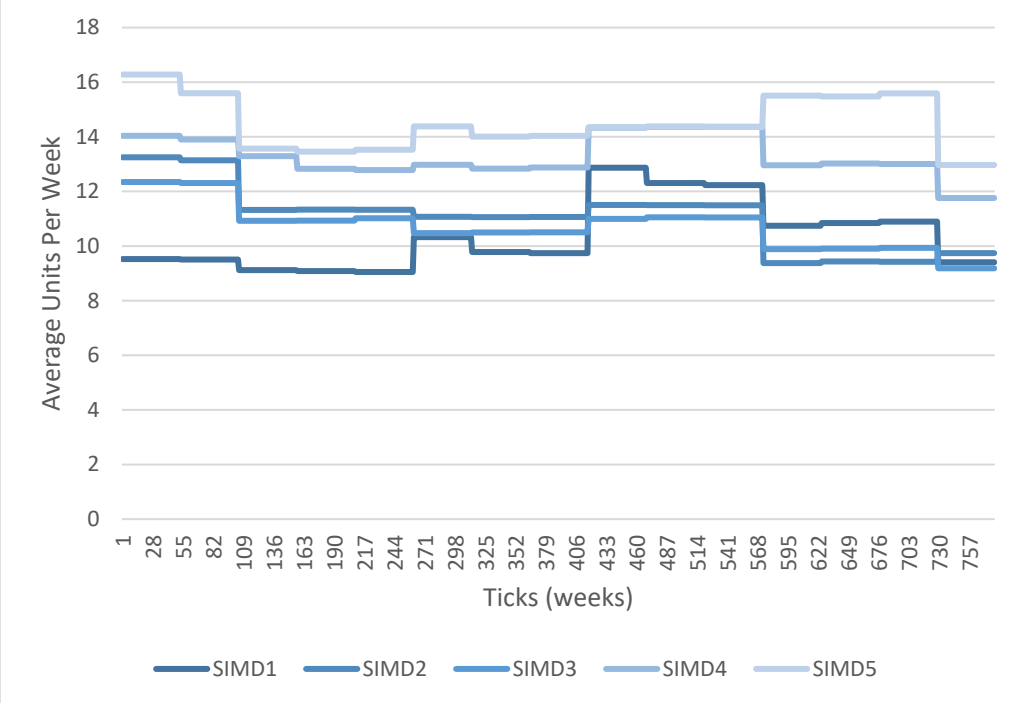
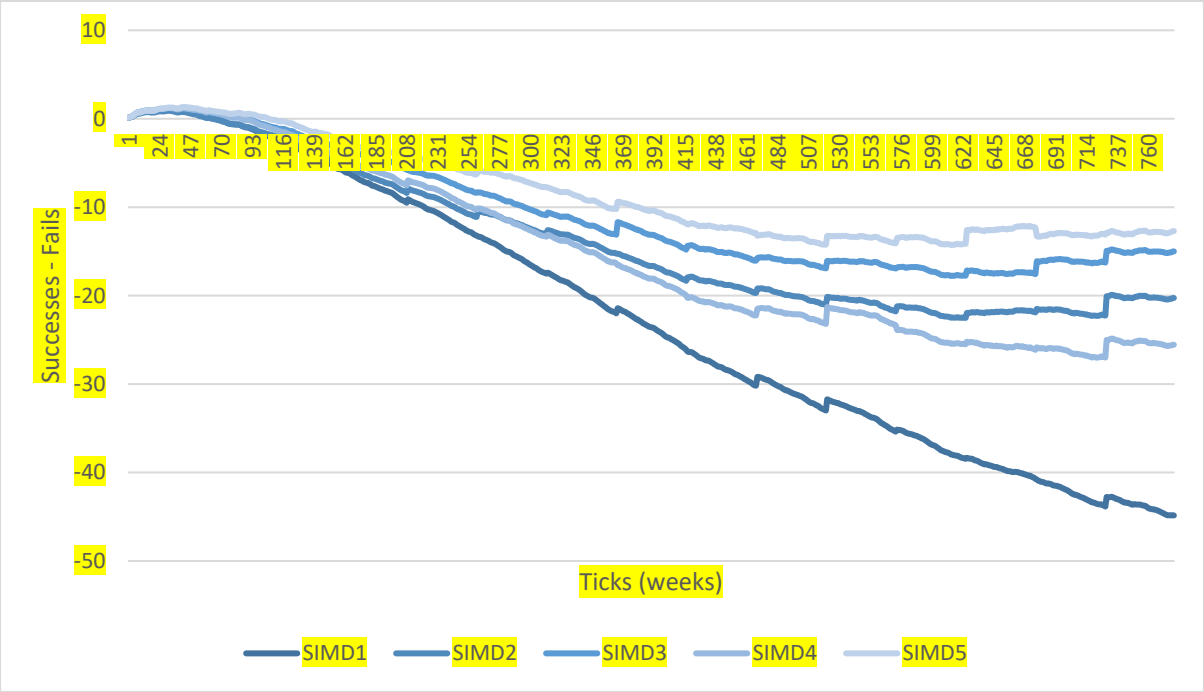


Figure 9.7 presents the average number of successes minus the average number of failures for each SIMD quintile. At the beginning of the simulation the difference in the number of successes and failures between SIMD quintiles is negligible. This is expected given that the individual agents enter the simulation with no previous history of successes and failures. However, as the simulation progresses the difference between each SIMD quintile steadily increases, with the least deprived quintile experiencing the fewest failures and the least deprived quintile experiencing the greatest number of failures. The difference in failures largely follows a socioeconomic gradient, however SIMD quintile 4 experiences more failures than the more deprived quintiles 2 and 3.

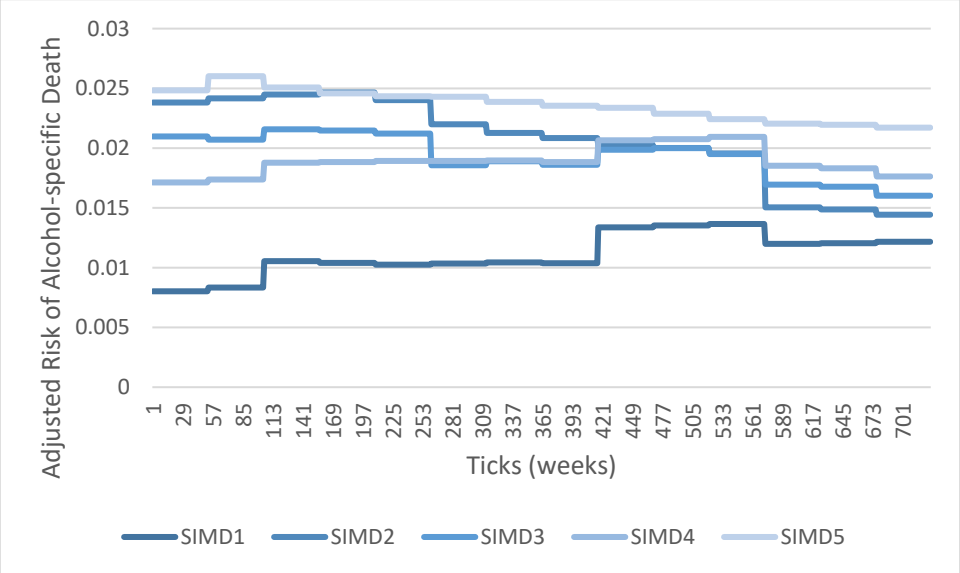
Figure 9-7: *Average successes minus failures across the best simulation split by SIMD quintile.*



These results testing the model's functionality suggest that the model is functioning as intended. The interim behavioural results fit preconceived notions that the most deprived quintile drink less alcohol on average, and given that they have fewer fundamental resources experience a far greater number of failures to their behaviour adapt to new risk or prevention information and new treatment.

The average adjusted risk split by SIMD quintile over the course of the simulation is displayed in Figure 9.8. The results are as expected, at the beginning of the simulation the risk of alcohol-specific death closely follows the observed trends in alcohol consumption displayed in Figure 9.6. Individuals in the least deprived quintile have the greatest risk of alcohol specific death, while those in the most deprived quintile have the lowest risk of alcohol specific death. However, as the simulation progresses and the most deprived quintile experience a greater number of failures to adapt their behaviour their risk steadily increases, while the risk for the least deprived quintile declines slightly.

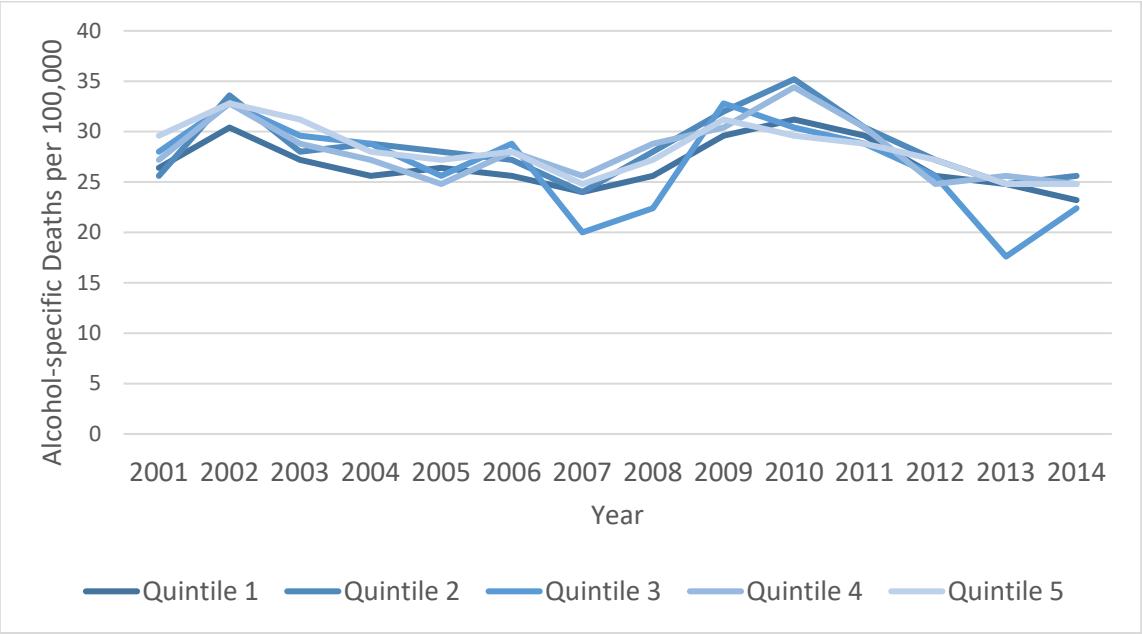
Figure 9-8: *The average adjusted risk of alcohol-specific death split by SIMD quintile.*



9.5.1 Model Calibration: Socioeconomic Gradient

The best fitting model setting to targets for all SIMD quintiles from 2001-2014 yielded an implausibility value of 9.29, which suggests the model output is a poor fit to the target data. The results of this model setting did not produce a socioeconomic gradient in alcohol specific deaths observed in the target data (see Figure 9.9).

Figure 9-9: *The best calibrated model results for all SIMD quintiles from 2001-2014.*



Figures 9.10 to 9.14 present the results from this best fitting model setting compared to the observed alcohol-specific deaths for each SIMD quintile in Scotland from 2001-2014. Note that model validation was not conducted given lack of model fit.

Figure 9-10: *The best calibrated model compared to the observed rate of alcohol specific deaths for the most deprived quintile (SIMD 1).*

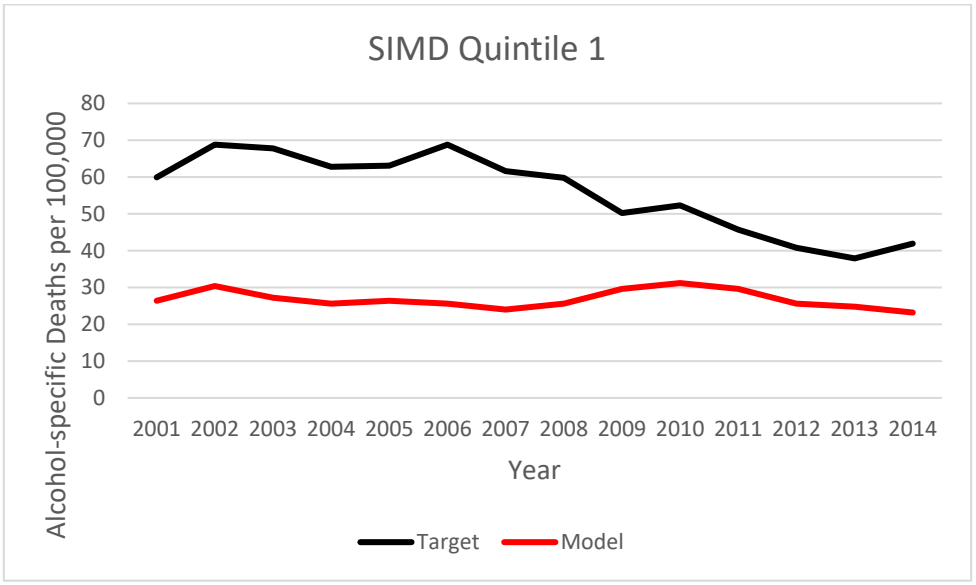


Figure 9-11: *The best calibrated model compared to the observed rate of alcohol specific deaths for SIMD Quintile 2.*

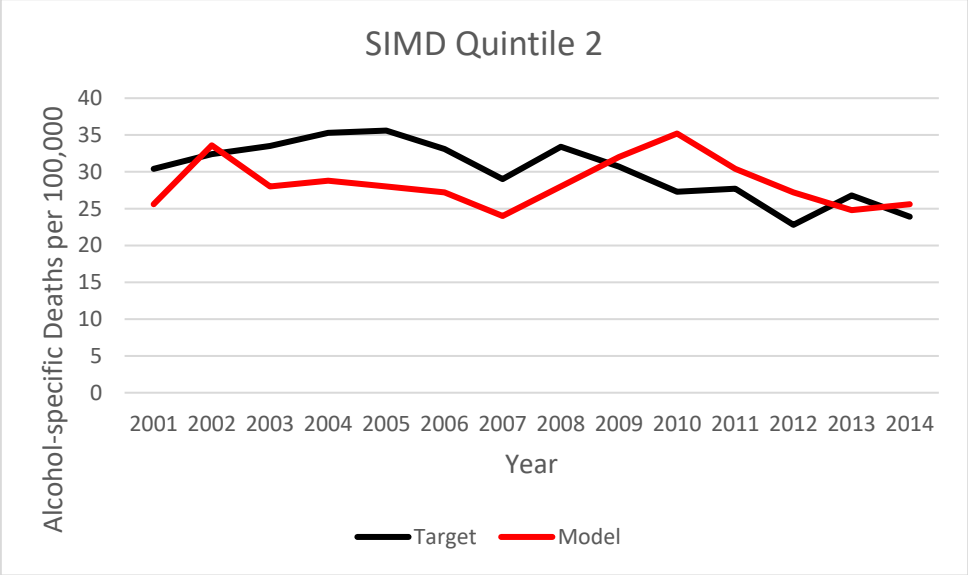


Figure 9-12: *The best calibrated model compared to the observed rate of alcohol specific deaths for SIMD Quintile 3.*

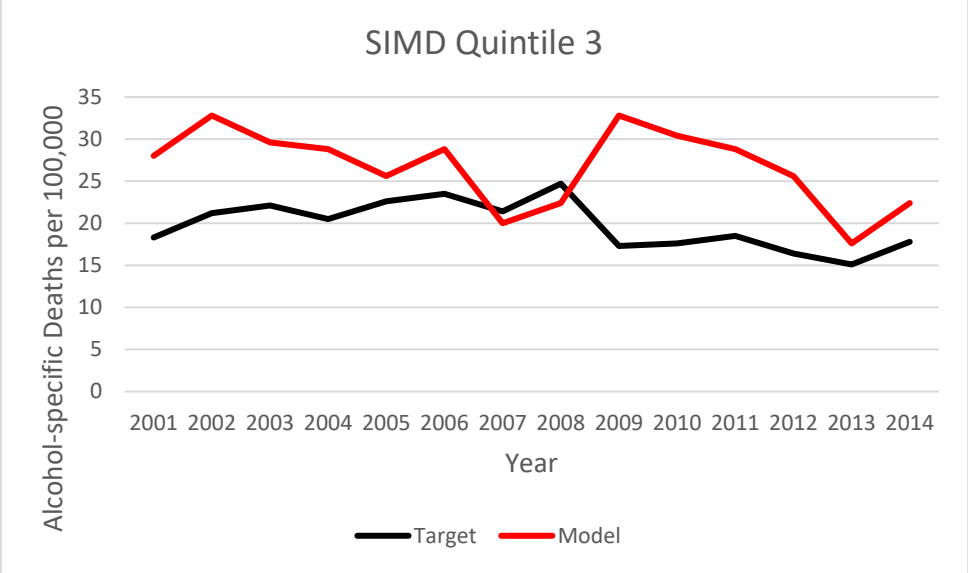


Figure 9-13: *The best calibrated model compared to the observed rate of alcohol specific deaths for SIMD Quintile 4.*

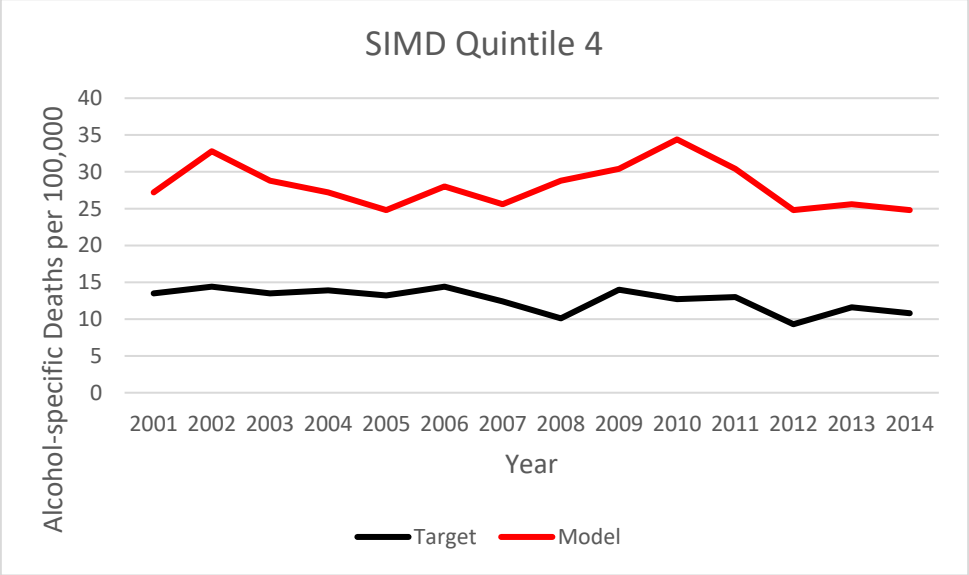
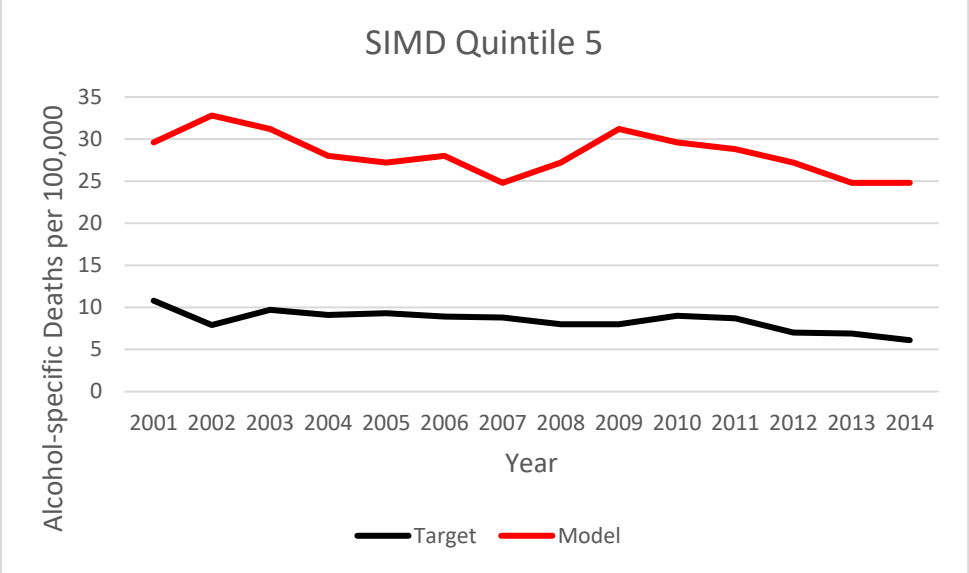


Figure 9-14: *The best calibrated model compared to the observed rate of alcohol specific death for the least deprived quintile (SIMD 5).*

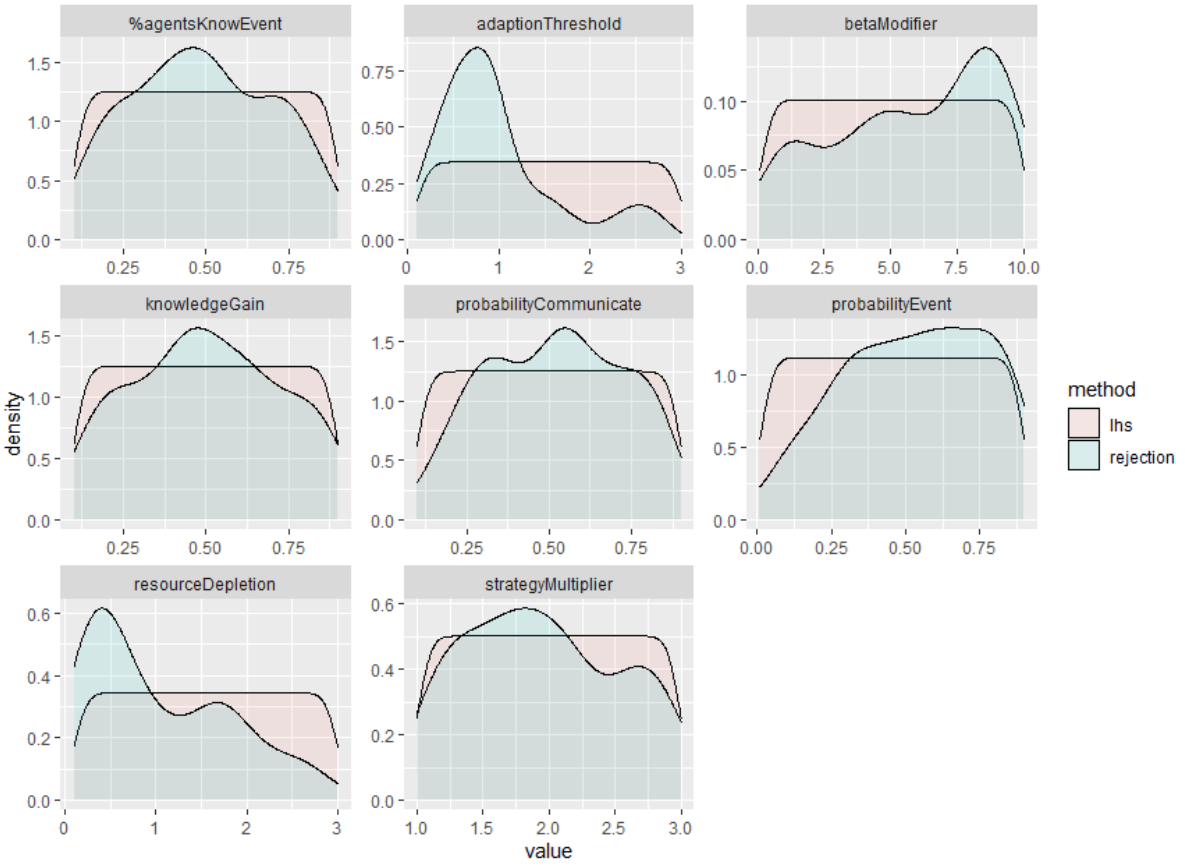


It is clear from the results that the model is not a good fit for the socioeconomic gradient in alcohol-specific deaths observed in the real-world data. The model underestimates the rate of alcohol specific deaths for the most deprived quintile (SIMD 1) and overestimates the rate of alcohol specific deaths for the least deprived quintiles (SIMD 4 and 5). It is a slightly better fit to alcohol specific deaths for quintiles 2 and 3. However, the model underestimates deaths for individuals in SIMD quintile 2 for the first 10 years and overestimates deaths for the remaining

5 years. The model also consistently overestimates deaths for individuals in SIMD quintile 3, except for years 2007 and 2008.

Figure 9.15 shows the posterior distributions estimated using rejection sampling and compares them to the prior distributions from the Latin hypercube sampling method. Posterior densities represent the frequency that each parameter takes a particular value from the set of simulations estimated to be the closest fit to observed target data by the rejection procedure. In this case the best 100 (or 1%) of model simulations. Therefore, these distributions indicate the particular parameter values that result in model outputs closest to the target data.

Figure 9-15: *Rejection sampling estimates of posterior distributions using a tolerance threshold of 1%.*



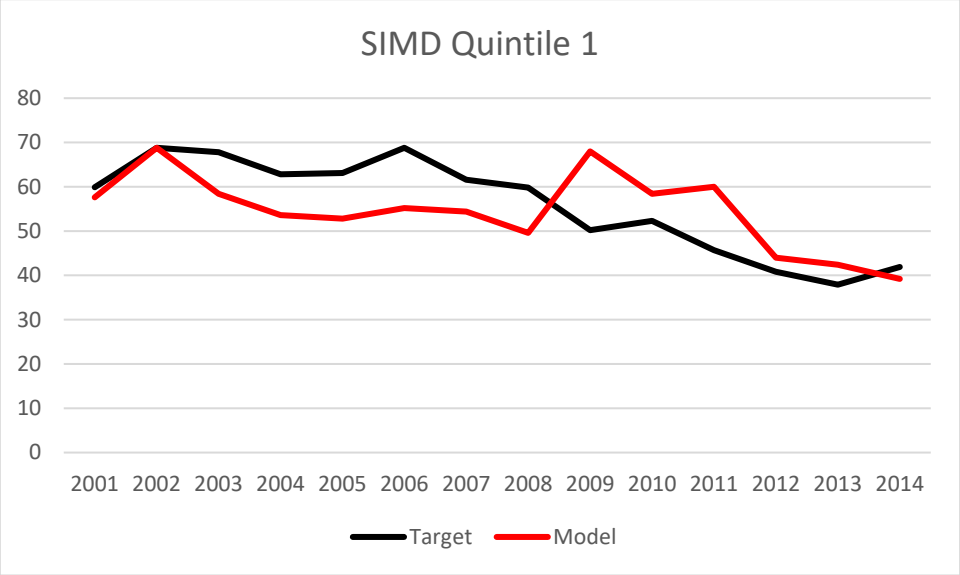
The posterior distributions for each parameter were distinctly different from the prior distributions used in the Latin hypercube sampling. For the parameters %agentsKnowEvent, knowledgeGain and probabilityCommunicate the model outputs more closely matched the targets when these values were around the midpoint. For both the adaptationThreshold and

the resourceDepletion parameters the values at the lower end of the range were more likely to produce results closer to the observed target data. Specifically, when the adaptationThreshold was <1 and the resourceDepletion value was <0.5. The betaModifier and probabilityEvent parameters were more likely to produce results closer to the targets when values were at the upper end of the range of possible values. The posterior distribution for the betaModifier was more precise with values above 7.5 producing more accurate results, while the posterior distribution for probabilityEvent indicated that values 0.25 and above were more likely to yield results closer to the targets. Finally, lower values and values around the midpoint for the strategyModifier were more likely to result in model outputs closer to the targets, while higher values, specifically those >2 were less likely to yield results close to the target data.

9.5.3 Exploratory Model Calibration: SIMD Quintile 1 and 5 independently

To test whether there were parameter settings which could produce model outputs which closely fit with the targets for SIMD quintile 1 and quintile 5, subsequent analysis was conducted to identify the lowest implausibility values for each quintile independently. Of the 10,000 samples, the best fitting model setting for a calibration only to the most deprived quintile (SIMD 1) target data yielded an implausibility value of 1.50, which is not a particularly good fit to the target data. While, the best fitting model setting for a calibration only to the targets for SIMD quintile 5 yielded an implausibility value of 0.25. This much lower implausibility value suggests a model which is a good fit to the observed target data. Figure 9.16 shows the results from this best fitting model setting compared to the observed alcohol-specific deaths for SIMD quintile 1 in Scotland from 2001-2014.

***Figure 9-16**: The best calibrated model compared to the observed rate of alcohol specific deaths for the most deprived quintile (SIMD 1) – only calibrating to SIMD 1 targets.*



The results suggest that it is possible for the model to produce results more similar to the target data when calibrating to only SIMD quintile 1 targets. However, while the model can explain deaths in 2001 and 2002 well, it underestimates the number of deaths from 2003-2008 and overestimates deaths from 2009-2013. This may be the result of error inherent in the absolute risk function which when calibrated to alcohol-specific deaths for the whole population slightly underestimates deaths from 2001-2008 and slightly overestimates deaths from 2009-2017 (see Figure 9.2).

Figure 9.17 shows the results from this best fitting model setting compared to the observed alcohol-specific deaths for SIMD quintile 5 in Scotland from 2001-2014. The model results suggest a good fit to the target data, with deaths only slightly overestimated in years 2006 and 2009 and slightly underestimated in years 2007, 2008 and 2011.

Figure 9-17: *The best calibrated model compared to the observed rate of alcohol specific deaths for the least deprived quintile (SIMD 5) – only calibrating to SIMD 5 targets.*

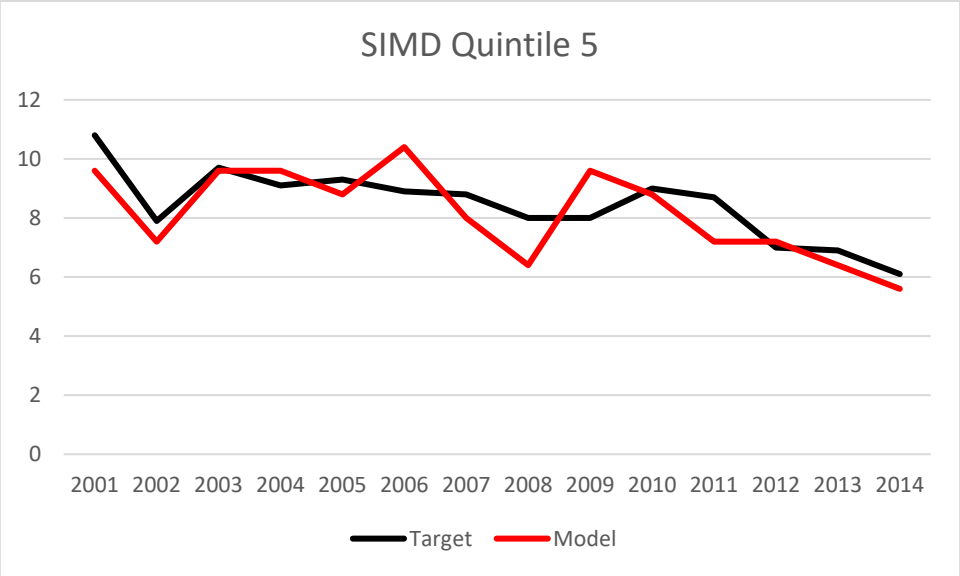


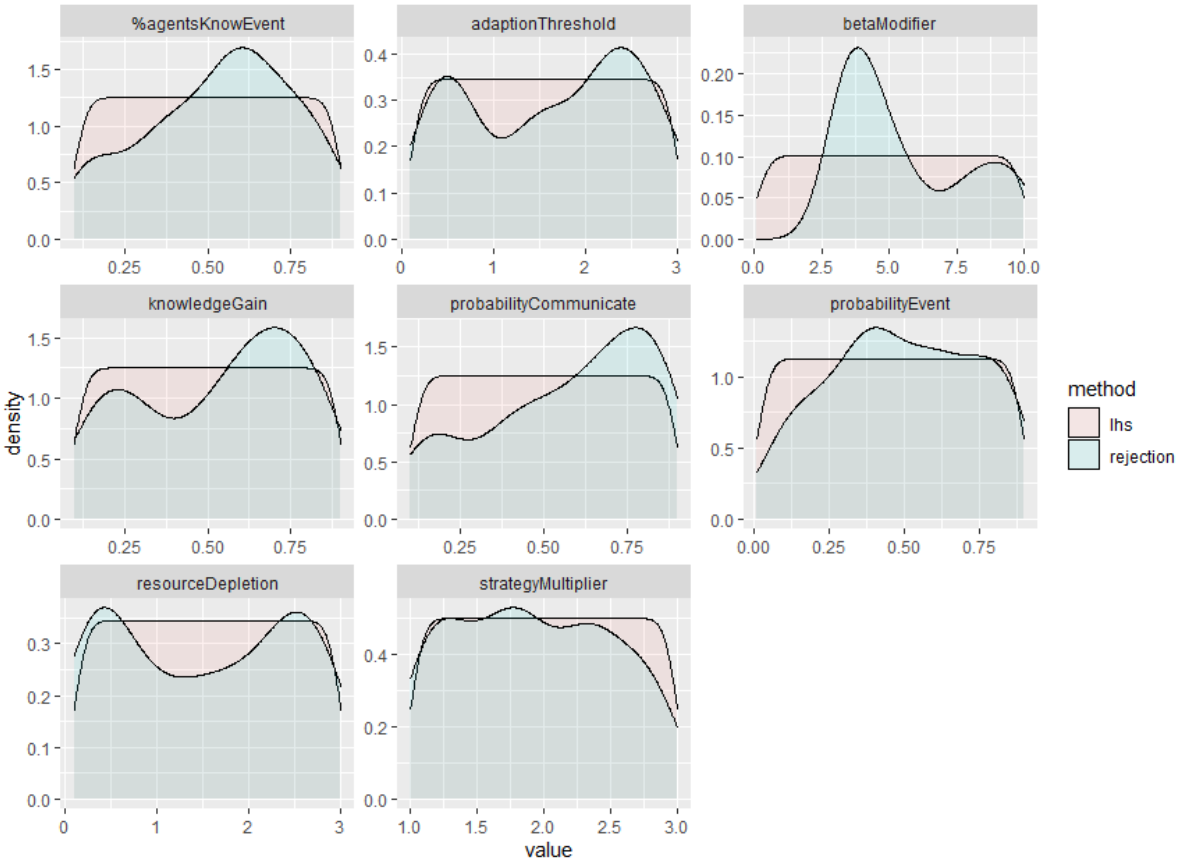
Table 9.2 presents the parameter values for each calibrated parameter when the model was either calibrated to targets for SIMD quintile 1 only or SIMD quintile 5 only. As shown in the table there was substantial differences in the estimated parameter values for all parameters other than knowledgeGain.

Table 9-2: *Parameter values for the best fitting models when calibrating to SIMD quintile 1 targets only and SIMD quintile 5 targets only.*

Parameter	SIMD Quintile 1 Targets Only	SIMD Quintile 5 Targets Only
probabilityEvent	0.6973	0.1020
%agentsKnowEvent	0.4484	0.7203
adaptationThreshold	0.2204	0.5372
probabilityCommunicate	0.8633	0.3638
strategyMultiplier	1.9322	2.8454
knowledgeGain	0.6714	0.6194
resourceDepletion	2.7558	0.4953
betaModifier	9.5349	4.8115

To identify which parameter values are most likely to produce outputs closest to the target data rejection sampling was implemented. As before the tolerance threshold was set to 0.01. Figure 9.18 shows the posterior distributions estimated from the best fitting 100 model settings when calibration to only SIMD quintile 1 targets.

Figure 9-18: *Rejection sampling estimates of posterior distributions using a tolerance threshold of 1% - when only calibrating to SIMD quintile 1 targets.*



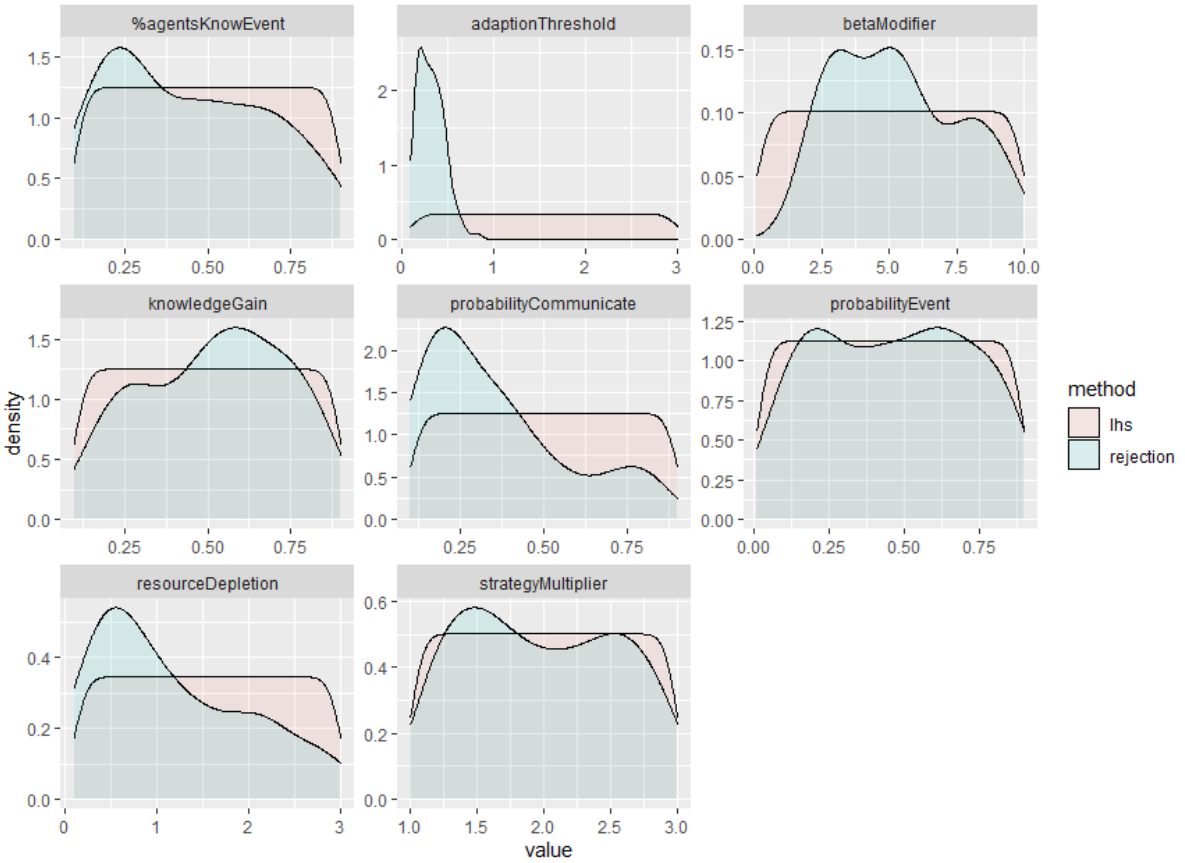
The posterior distributions for almost all parameters were distinctly different from the posterior distributions estimated using targets for all SIMD quintiles, and all but one was distinctly different from those used in the Latin hypercube sampling. For %agentsKnowEvent, knowledgeGain and probabilityCommunicate values higher than the midpoint were more likely to yield results closer to the target data, whereas when the model was calibrated to all targets values around the midpoint where more likely to produce more accurate results (see Figure 9.15). The posterior distributions for adaptationThreshold and resourceDepletion were bimodal. The lower end and the upper end of the range of values were most likely to result in outputs closer to the target data. Again, these distributions greatly differed from the posteriors produced when calibrating to all targets, values were more likely to be at the lower end of the range (see Figure 9.15). The posterior distribution for the strategyMultiplier did not greatly differ from the prior distribution, however values below 2.5 were more likely to result in model

outputs closer to the target data. This was similar to the posterior distribution produced for this parameter from the calibration to all targets, however the distribution from the calibration to all targets was more precise (see Figure 9.15). The posterior distribution for the probabilityEvent parameter again was slightly different posterior distribution estimated from the calibration to all targets. Value around the midpoint for this parameter tended to produce results closer to the targets. Finally, the posterior distribution for the betaModifier parameter highlighted that values at the lower end, particularly between 2.5 and 5.0, resulted in outputs closer to the target data. This was vastly different from the posteriors distribution produced for this parameter when calibrating to all targets which showed that values at the very upper end of the range, between 7.5 and 10.0 resulted in outputs closet to the targets.

Rejection sampling was used to identify the parameter values which are most likely to lead to model outputs closest to the targets. As before the tolerance threshold was set to 0.01. Figure 9.19 shows the posterior distributions estimated from the best fitting 100 model settings when calibrating only to SIMD quintile 5 targets. The posterior distributions displayed in Figure 9.17 were all distinct from the prior distributions used in the Latin hypercube sampling, and for some parameters differed from both the posteriors estimated when calibrating to all targets (see Figure 9.15) and when calibrating to only the SIMD quintile 1 (see Figure 9.18).

When the %agentsKnowEvent and the probabilityCommunicate parameters had values at the lower end of the value range model results were closer to the targets. This was distinctly different from the results calibrated to all targets, which demonstrated that values around the midpoint are more likely to produce closer outputs (see Figure 9.15), and the results from calibrating to SIMD quintile 1 targets only, values above the midpoint and higher were more likely to produce closer results (see Figure 9.18). For the knowledgeGain parameter values between 0.5 and 0.75 were more likely to produce model outputs closer to the targets, this was similar but less pronounced than the distribution produced when calibrating only to SIMD quintile 1 targets.

Figure 9-19: *Rejection sampling estimates of posterior distributions using a tolerance threshold of 1% - when only calibrating to SIMD quintile 5 targets.*



The posterior distributions for the adaptationThreshold and resourceDepletion parameters were left-skewed, meaning that when the parameters took lower values they were more likely to produce results closer to the targets, this was similar to the distributions produced when calibrating to all targets (see Figure 9.15), however the distribution was more precise when calibrating to SIMD quintile 5 targets only. The strategy multiplier when set to values at the lower end of the value range was more likely to produce results closer to targets, particularly when values were around 1.5. The probabilityEvent parameter was slightly bimodal, with values below and above the midpoint being more likely to produce results closer to the targets. This differed from the posterior distributions estimated from calibrating to all targets, which showed that higher values were more likely to produce closer results, and the posterior distributions estimated from calibrating to only SIMD quintile 1 targets, which showed values below the midpoint were more likely to produce closer results. Finally, the betaModifier

posterior distribution calibrated to SIMD quintile 5 only was the least precise distribution when compared to those estimated when calibrating to all targets (see Figure 9.15) and calibrating to SIMD quintile 1 targets only (see Figure 9.18). Values between 2.5 and 6.0 were more likely to produce model outputs close to the targets.

9.6 Discussion

9.6.1 Overview of Model Results

This paper presents the first agent-based model to simulate the main mechanism described in FCT and link this mechanism to alcohol-specific deaths for each deprivation quintile. It is clear from the model calibration results that the model cannot explain the socioeconomic gradient in alcohol specific deaths in Scotland from 2001-2014. The implausibility value obtained from a calibration to all target data for every SIMD quintile suggests that even the "best" model setting is a poor fit to the targets. The model when calibrated to all targets underestimates deaths for the most deprived quintile (SIMD quintile 1) and over estimates deaths for the least deprived quintiles (SIMD quintiles 4 and 5). These results suggest that there is not one best model setting that can account for alcohol specific deaths for every SIMD quintile.

This is despite the finding that the model functions as intended. Individuals in the most deprived quintile experience more failures to adapt and less successes, while individuals in the least deprived quintile experience more successes and fewer failures. Therefore, the simulation is accurately reflecting the main mechanism specified in FCT, that given the unequal distribution of fundamental resources more disadvantaged individuals do not have access to these resources and as a result cannot deploy them to adapt to risk, prevention and treatment information (97). Meanwhile, more advantaged individuals have increased access to flexible fundamental resources and can deploy them to successfully adapt their behaviour. In addition to successes and failures, alcohol consumption also determines alcohol specific death in this model. The trends in alcohol consumption by SIMD quintile were consistent with the AHP. The least deprived quintile had the highest average units per week consumed

throughout the simulation while those in the most deprived quintile on average had consistently lower alcohol consumption.

While the model could not simulate patterns of alcohol specific deaths consistent with the AHP, when calibrating to the most deprived quintile and least deprived quintile targets independently it was possible for the model to reproduce trends similar to the target data. Critically to produce these results there were several FCT parameters that differed greatly depending on which set of targets were used in the calibration procedure. Parameters which notably differed included the adaptation threshold, resource depletion value, % of individual agents that know of the event, and probability that individual agents will communicate.

When calibrating to all targets an adaptation threshold of less than 1 was more likely to produce results closer to the target data. An even lower value, of below 0.5, was more likely to accurately simulate deaths for the least deprived quintile. However, when calibrating to targets for the most deprived quintile an adaptation threshold either below 1 or above 2 was more likely to simulate alcohol specific deaths closest to the targets. The resource depletion parameter displayed a similar pattern to that observed for the adaptation threshold. These findings are likely to be the result of model implementation given that with a lower adaptation threshold and resource depletion value the least deprived quintile (and likely all quintiles to some extent) will be able to easily and quickly successfully adapt to new risk, prevention and treatment information and thus reduce their risk of alcohol specific death. While a higher adaptation threshold and higher resource depletion threshold would make it more difficult for the most deprived quintile (and again all quintiles to an extent) to adapt which will result in more failures and as a result increase their risk of alcohol specific death.

Another parameter that greatly varied depending on the chosen targets was the % of individual agents that know of the event. For a calibration to all targets a value around the midpoint yielded results closer to the target data. However, calibrating to the most deprived and least deprived quintiles independently revealed a large disparity in the value most likely to reproduce the observed data. To produce results closer to the targets for the most deprived

quintile a large proportion of individual agents (between 50% and 75%) must be informed of the event from the communicator. However, when a smaller proportion of individual agents (less than 25%) are informed of the event from the communicator the model is more likely to produce results closer to the targets for the least deprived quintile. This trend was also observed for the parameter probability communicate. Again, this is likely to be the result of model implementation. The individuals in the most deprived quintile would experience more failures if they are more likely to be informed of the event in the first place, either from the communicator or a social connection.

The model could not explain the socioeconomic gradient in alcohol specific deaths in Scotland from 2001-2014. These findings could indicate that FCT alone cannot explain the AHP. Alcohol specific deaths adjusted by successes and failures resulted in a pattern whereby all quintiles experienced similar rates of death. Therefore, the model did increase the risk of death for the most deprived quintile and reduce the risk of death for the least deprived quintile, as the least deprived quintile was shown to consume more alcohol on average yet die at the same rate. There are many explanations suggested for the AHP which could compound the risk of harm to the most deprived over and above the successes and failures they experience when adapting to new risk, prevention and treatment information (164). For example, it has been proposed that more deprived groups experience more stereotypes and stigma which leads to social rejection and exclusion, which leads to fewer social resources increasing their vulnerability to harm (21,22,131,132). It has also been purported that experiencing more psychological stressors (16,36,37,41), living in deprived areas with a lack of treatment facilities and greater neighbourhood disorder (5,22,108), and a disadvantaged start to life, including the experience of negative prenatal factors and childhood household dysfunction (9,23,144) compound the risk of harm for those of a low SEP. Therefore, the main mechanism described in FCT may not be sufficient to explain inequalities in alcohol specific deaths.

However, it is more likely that the model did not produce a socioeconomic gradient in alcohol specific deaths due to issues with model implementation. Findings suggest that the variables

used to operationalise fundamental resources may not have adequately reflected real-world resources available to each SIMD quintile. As highlighted in the results at baseline the resources available to an individual in the most deprived quintile compared to the least deprived quintile only differed by 0.14 on average. Given that we know from tests of model functionality that the most deprived quintile did experience more failures and less successes while the least deprived quintile experienced the reverse as expected, the fact the model did not produce a socioeconomic gradient in alcohol specific deaths could be due the limited difference in fundamental resources between the quintiles. As a result, individuals in the most deprived quintile experienced fewer failures than they would have if their resource pool was lower and individuals in the least deprived quintile experienced fewer successes than if their resource pool was higher. Therefore, the risk of alcohol specific death was not sufficiently adjusted, and alcohol consumption remained the stronger determinant of death.

It is particularly difficult to compare the model results to previous research given that it is the first ABM that attempts to test an explanation for the AHP and that there are very few empirical demonstrations of FCT. Previous empirical work testing FCT has demonstrated that SES was strongly correlated to deaths which were preventable (274). The previous empirical work only infers the role of resources in the generation of health inequalities, but does not directly test the theory. A subsequent study used a dynamic design to examine differences in cholesterol levels pre- and post the introduction of a new cholesterol lowering technology (275). This study found clear evidence of FCT in that after the introduction of statins the social gradient in cholesterol levels was reversed, prior- more wealthy individuals had higher levels of cholesterol, however post- this relationship was reversed illustrating that the wealthy were more successful at adapting to the introduction of this new treatment. However, to our knowledge there are no existing empirical studies which explicitly operationalise the resources described in FCT.

A recent qualitative study attempted to unpick the ambiguity of the connection between fundamental resources and health using health insurance denials for genetic testing as a case

study (276). Combining Bourdieu's work on capital and symbolic power and FCT, the paper identified three examples of the mechanisms through which fundamental resources influence health. The first was that social learning motivated an individual to deploy financial resources towards a health promoting behaviour (276). This is to an extent was represented in the current ABM as upon successful adaptation individual agents communicate strategies to peers in their social network which facilitates the behavioural adaptation of their peers themselves. The author also identified perceived self-efficacy as detrimental to a patient's power in a clinical setting ultimately leading to better advocacy by physicians (276). Finally, the paper suggests that agency, particularly the agency to "*pursue multiple avenues and cultivate the capacity to persuade and influence other actors*" facilitates consistent health promoting behaviour particularly when faced with opposing positions (276). Self-efficacy and agency were not explicitly operationalised in the current version of the ABM. To construct the fundamental resource power, variables that indicated the extent to which individuals had the power to financially and psychologically achieve your goals were used in the ABM. However, the power is undoubtedly more complex and would include constructs such as self-efficacy and agency, alongside other sources of power such as culture and belief, collective organisations and positional power (256,276). Therefore, the operationalisation of power in this model may not have been sufficient to capture this complex construct.

9.6.2 Limitations

The process of translating a theory into an ABM is a challenging task. To encode certain aspects of FCT required interpretation of the theory and assumptions. FCT at its core is concerned with the availability and use of fundamental resources to adapt to new risk, prevention and treatment information (96,97). However, descriptions of the theory lack precise specifications of how and in which situations these resources are deployed. We chose to use an adaptation threshold and provide equal weighting to the fundamental resources of money, power, prestige and knowledge. In terms of social connections instead of imposing a measure of quantity or quality, we generated a social network through which individuals could

communicate the event information or strategies to help members of their social network adapt to this information. This is just one possible interpretation of the theory. It is therefore important when using theory to inform the design and development of ABMs that decisions made during the implementation are transparent.

Another limitation of this study is that the calibrated beta slope values for the absolute risk function produced results that slight underestimate deaths from 2001-2008 and slightly overestimates deaths from 2009-2017. This error was therefore carried over into the ABM model calibration and could have impacted the model results. An extensive calibration procedure (sampling 15,000 parameters) was used to attempt to identify beta slope parameters which best explained alcohol specific deaths at the population level. However, this error was unavoidable and most likely resulted from the spike in alcohol consumption in the most deprived quintile from 2006-2010 (see Figure 6.4 in chapter 6) which made it impossible to estimate just two consistent beta slope parameters which could perfectly reproduce trends in alcohol specific deaths across the entire time period.

An additional limitation of the modelling work is that the individual agents enter the simulation with no history of successes or failures to adapt. However, in reality individuals will have experienced successes and failures prior to 2001 given that efforts to treat, prevent and inform the public of risks from alcohol began long before 2001. The tests of model functionality regarding successes and failures clearly shows the ‘burn in’ of inequality in FCT. However, the target data used for model calibration is from a time when the AHP is well established and is instead interested in the mechanisms that sustain the AHP as opposed to the mechanisms that generate it in the first place. This suggests that reasonable stability in the mechanisms encoded from FCT is required before applying this as an initial condition for modelling the AHP during this time period. A possible solution to this limitation is to introduce a burn in period in the model; this means simulating the model for several ticks prior to the starting timepoint in 2001 to identify stability in the inequality generated from FCT. A burn in period was not introduced in this version of the model due to time restrictions, and difficulties estimating how

long the burn in period should be and obtaining a synthetic population for years prior to 2001 given the lack of data.

9.6.3 Future Directions

Prior to taking this modelling work forward in future research, several implementation issues require consideration. As discussed above it was not possible to identify two beta slope parameters that could perfectly reproduce trends in alcohol specific death at the population level for Scotland from 2001-2019. It is possible that this was due to the alcohol consumption data used to assign each individual agent an average units per week. This data was not contained within the survey data used to generate the micro-synthetic population, and matching methods were used to assign alcohol consumption to similar individuals in the British Household Panel Survey from the General Household Survey and Scottish Health Surveys. Future research should attempt to identify alternative data sets, potentially in alternative settings, which contain both the variables required to operationalise fundamental resources and alcohol consumption in order to ensure that alcohol consumption is accurately estimated. Additionally, future work attempting to improve the functionality of this model should test the impact of introducing various lengths of burn in period to the model.

Currently this model represents the core mechanism present in FCT purported to generate health inequalities. However, more recent descriptions of FCT detail a role of the environment in the form of available community resources (277). In future iterations of the ABM presented in this paper an aggregate measure of community resources for each deprivation quintile could also be used to adjust the risk of alcohol-specific death.

Additionally, individuals in this ABM make the decisions using simple rules based on probabilities and thresholds. Researchers in the wider field of ABM have advocated for the use of more complex agent decision making frameworks incorporating psychological mechanisms such as Belief, Desire and Intentions (BDI) (209) and the Theory of Planned Behaviour (208,278). Therefore, subsequent versions of the model could attempt to replace the simple rule decision making framework with a more complex framework. The use of a

framework based on psychological theory would incorporate greater individual heterogeneity and could be used to capture instances where individuals have the information and resources available to adapt but choose not to.

Alternatively, the reason this model cannot reproduce the AHP could be that FCT is not sufficient to explain inequalities in alcohol specific deaths. As previously discussed there are numerous explanations for the AHP that remain untested (164). Agent-based models remain a useful tool to understand the mechanisms that may generate and sustain the AHP, and could be used to explore explanations the highlight a particular role for individual action and interaction such as the experience of stereotypes and stigma. Other potential explanations such as the cumulative impact of disadvantage across the life course may require exploration using alternative methods. However, researchers in the field should continue to test explanations for the AHP that shift the focus from individual level risk behaviour in order to identify ways to intervene to reduce inequalities in alcohol harm.

9.7 Conclusion

To conclude, this model is an example of how agent-based models can be applied to capture complex mechanisms which may generate inequalities in alcohol specific deaths. This particular model could not reproduce the socioeconomic gradient observed in alcohol specific deaths for Scotland between 2001 and 2014. It is possible that this FCT is not sufficient to explain the AHP or alternatively that there were issues with model implementation, particularly the findings point to the fact that fundamental resources may not be adequately represented and a burn in period is required. Future research should further explore the role of FCT generating and sustaining the AHP, including improving the implementation of the current model, the addition of a secondary mechanism which capture the role of community resources and implementing alternative agent decision making frameworks. Alternatively, there are still many other potential explanations for the AHP that remain untested and require further empirical investigation.

Chapter 10 Discussion

10.1 Chapter Overview

This PhD aimed to advance the understanding of the causal mechanisms generating and sustaining the AHP. This chapter summarises the main findings from this PhD thesis. It then discusses the contribution of the thesis, methodological reflections, suggestions for future research and implications for policy and practice. The chapter ends with concluding remarks.

10.2 Main Findings

10.2.1 Systematic Review

The first study of the thesis found that there were 16 themes within six domains (Individual, Lifestyle, Contextual, Disadvantage, Upstream and Artefactual) that were used in the existing literature to explain the AHP. Risk behaviours were the most prevalent explanations and dominated the empirical work. There were also many other, mainly hypothetical explanations for the AHP including individual-level mechanisms (e.g., biological or psychological), contextual (e.g., place-based factors), the lived experience of disadvantage, and upstream structural factors (e.g., the economy and politics). The suggestion of many wide-ranging possible explanations partly reflects an awareness that the AHP is complex and researchers do not view the causes in isolation. However, recurring explanations such as social support and access to health care have been neglected while researchers frequently return to testing the impact of risk behaviours. The reason for a focus on health risk behaviours could be due to a lack of structured theory used to explicate explanations or a lack of methods to carry out more complex analyses. Based on the evidence from the systematic review the key implication is that tackling drinking alone will not reduce inequalities in alcohol harm, while there is some evidence that improving multiple health behaviours may attenuate the risk of alcohol harm, it is important to look outside the scope of health behaviour to mitigate the inequality produced by the paradox.

10.2.2 Theory Paper

The second paper in this thesis found that research investigating the AHP does not explicitly use theory. Hypothetical explanations for the AHP do touch on mechanisms described in theories of health inequality, however this tends to be ad-hoc and lacks clear theoretical structure. Theories such as the social determinants of health, fundamental cause theory, political economy of health and the eco-social model could be used to provide structure to empirical work investigating the causes of the AHP. One study indicated that the use of health inequality theory is promising given they found that cumulative behaviours *across the life course* could explain a greater proportion of harm experienced by lower SEP groups (153). Explicitly drawing on health inequality theory would shift the current thinking away from health behaviour in isolation to the wider context of complex interacting mechanisms between individuals and their environment. However, as the causal processes described in these theories are complex and they therefore require different methodological perspectives, specifically drawing on methods from complexity science may be necessary to understand these processes.

10.2.3 Modelling Work

The conceptual modelling work described in chapter 7 of this thesis found that it was possible to represent FCT as a computer using the MBSSM framework to draw out the mechanisms and entities described in the theory. However, an agent-based model representing the main mechanism described in FCT could not explain the socioeconomic gradient observed in alcohol specific deaths in Scotland from 2001-2014. The mechanism in the model captured the unequal distribution of fundamental resources, that results in more affluent individuals having greater access to these fundamental resources which they can then deploy to adapt their behaviour to risk and prevention information, and uptake new treatment, while more disadvantaged individuals do not have access to these resources (97). There are two possible explanations as to why the model could not reproduce trends associated with the AHP: 1) due

to issues with model implementation and 2) that the main mechanism described in FCT on its own is not sufficient to explain the AHP.

10.3 Contribution of the thesis

The PhD contributes to the alcohol policy and epidemiology literature. Two papers (Chapters 4 and 5) summarise what is currently known about the causes of the AHP and suggest future directions for research in the endeavor to understand the mechanisms that generate and sustain the AHP. Chapter 5 in particular sets out explicitly how each health inequality theory discussed could be used in research by drawing on research designs implemented in social epidemiology which attempt to understand the causes of health inequality more generally. For example, the use of new measurements that capture social capital (178) or cross-national comparisons (186), methods which have yet to be applied in the context of the AHP.

A static microsimulation model representative of the population of Scotland in 2001 was also constructed and validated for the purposes of this PhD. The British Household Panel Survey and Scottish census were used to generate the microsimulation model. The British Household Panel Survey collects data on a wide range of topics including education, health and usage of health services, labour market behaviour, socioeconomic values and income (279), and therefore the microsimulation model could be used to investigate a wide range of research questions. The microsimulation model can be obtained for use in future simulation studies by members of the research team and by collaborators on request however future users of the microsimulation model are required to be registered with the UK Data Service.

The final contribution of this thesis was to serve as a case study of how a mechanism-based approach can be applied to investigate a complex public health problem. A six-step approach was taken to guide the stream of research conducted during this PhD. This approach was adapted from a previous approach for critical realist data analysis (220), to fit the context of using an ABM to test the explanatory value of a theory. The first step in the approach was to identify and describe the phenomenon of interest, in this case the AHP (Chapters 1 and 4). The second was to identify theories that could be used to explain the phenomenon (Chapter

5). The alteration to the second step was partially informed by the findings of the systematic review in Chapter 4, that there were many competing fragmented explanations for the AHP, and also reflected the PhDs focus on testing the ability of health inequality theory to explain the AHP. Therefore, in the context of this phenomenon it was important to start with composite theories that could be used to inform the modelling process. This step replaced the identification of key components of the phenomenon of interest, however in the context of other public health problems it may be more suitable to attempt to identify components as opposed to theories if the evidence base points to a particular explanation. The remaining steps were specific to the development of an ABM (Chapters 7-9), and included the translation or redescription of a theory in a conceptual model, the identification of target data and data used to generate a microsimulation of the population of interest, and retroduction; identifying the mechanisms necessary for producing and/or sustaining the AHP using a computer model which is one of the methods of retroduction. Carrying out these steps demonstrated how the MBSSM architecture can be used to translate theory into conceptual models to inform ABM development and how parameter calibration can explore whether a model can reproduce trends associated with the phenomenon of interest. Given that the model could not reproduce the socioeconomic gradient in alcohol specific deaths observed in Scotland from 2001-2014 it was not possible to carry out the final step: model validation. The novel methodological approach taken in this PhD is described and explained in detail and could be applied to other topics in the field of public health. To ensure the dissemination of this methodological approach the research team plan to write a methods paper that outlines the six-step process and uses the work from this thesis as an illustrative example.

10.4 Methodological reflections

This was the first study to explicitly apply health inequality theory and ABM methodology to understand the AHP. Critically this thesis demonstrates how a mechanism-based approach can be applied to bring together theory and complex systems methodologies such as ABM to attempt to understand a complex public health problem.

10.4.1 Strengths

The thesis presents the first detailed use of a mechanism-based approach to develop an ABM to understand the causes of the AHP. Most of the research investigating the AHP has focused on the impact individual level risk behaviour. However, there is an increasing appetite in the field to explore other factors associated with socioeconomic circumstances, for example access to healthcare, material resources and contextual factors (e.g., characteristics of the drinking environment) (42,110). A mechanism-based approach offers the opportunity to investigate these types of explanations. Mechanisms consist of entities, their properties, and the activities entities engage in, either collectively or independently, to bring about a particular outcome (53). Entities can be individuals, structural entities (e.g., the government) and the environment, and a mechanism-based approach can be used to capture the complex actions and interactions of entities to examine the impacts. Instead of testing statistical regularities between variables a mechanism-based approach offers an alternative perspective by detailing how regularities such as the AHP are brought about (53). That is not to say that a mechanism-based approach should replace traditional observational epidemiological research in public health. However, a combination of both methods would allow us to identify why observed trends come to be, and as a result the levers that can be used to provide solutions to complex public health problems which is particularly important for policy decision making.

Another strength of this PhD is that it develops an ABM which is particularly well-suited to quantitatively test mechanisms. ABM allowed us to explicitly model and therefore test the main mechanism described in FCT by simulating individuals as they react to new risk, prevention and treatment information. Specifically using ABM made it possible to capture not only individuals, their fundamental resources and under which conditions they deployed their resources to adapt their behaviour but also how they interacted with other individuals via a social network. The ABM also explicitly models features of a complex system, for example it was possible to represent a feedback loop of successful adaptations increasing individual level knowledge and greater knowledge increasing the chance of successful adaptation. The model

was constructed in a modular fashion (see Appendix E) which makes it particularly flexible and easy to adapt to new information including the possibility of introducing a secondary mechanism that simulates the role of community resources which has been emphasised in more recent descriptions of FCT (277).

Furthermore, the ABM constructed in this thesis attempted to explain inequalities in alcohol specific death, rather than differences in health behaviour between socioeconomic groups. Existing ABMs exploring socioeconomic inequalities in health more generally tend to focus on understanding health behaviour as opposed to linking behaviour to harm. For example, previous models have examined the impact of urban segregation on inequalities in diet (196), interventions to reduce tobacco sales for low income populations (280), and the differences in Urban Green Spaces visiting between socioeconomic groups (281). Of those studies that have used ABMs to examine physical health outcomes such as the incidence of severe neonatal morbidity and deaths (282), health status (283), and the prevalence of depression (284,285), the majority did not calibrate the model outputs to target data and those that did used behavioural targets as opposed to health-related targets (e.g., the prevalence of bus use) (284).

Finally, the thesis made use of readily available secondary data throughout the project and synthesised data from across disparate data sets which made it possible to follow the entire six-step process of a mechanism-based approach in 3 years. This included the development and validation of a static microsimulation model which was required to develop an empirical data driven ABM. On the other hand, because secondary data was used the project had to make use of the variables already available in existing datasets available from the UK Data Service. Two main issues arose from this, that there was no alcohol consumption data collected in the BHPS and there was limited choice regarding which proxies could be used to represent the fundamental resources described in FCT.

10.4.2 Limitations

The first limitation was that it was not possible to identify and obtain one survey that contained all of the variables necessary to operationalize the microsimulation and ABM. The British Household Panel Survey was selected to construct the static microsimulation model of the population of Scotland as it collected detailed information of socioeconomic position, including measures of income and education which could be used to operationalise fundamental resources in the model. However, the British Household Panel Survey does not collect information on health behaviour, and specifically relevant to this model it did not contain any information about alcohol consumption. Therefore, alcohol consumption was estimated for each individual from the General Household Survey, by matching individuals in each survey based on age, sex, marital status and economic status. This increases the likelihood of error in the estimation of alcohol consumption, and if possible obtaining a data set with both data on socioeconomic position and alcohol consumption would have been preferable to construct the micro-synthetic population.

An additional data related limitation was that UK data rather than Scottish specific data was used to generate the microsimulation model. While Scotland is similar to the other countries in the UK, Scotland has a unique relationship with alcohol consumption and alcohol harm. For example, in 2019 alcohol sales were 9% greater in Scotland compared to England & Wales (286), and the rate of alcohol specific death in Scotland was 18.6 per 100,000 compared to 10.9 per 100,000 in England and 11.8 per 100,000 in Wales (287). Therefore, as the General Household Survey was used to estimate alcohol consumption for years 2001-2006, given this was data from a UK wide survey the alcohol consumption in the population for those years may not be accurate. However, it was not possible to estimate alcohol consumption from Scotland only data as the Scottish Health Survey was only carried out for the years 1998 and 2003, before becoming an annual survey in 2008. Additionally, the Scottish sample within the General Household Survey was too small, for example in 2001 the Scottish sample made up 8.4% of the total sample, and therefore the subsample could not be used.

In terms of the ABM there were two technical limitations that are discussed in Chapter 9. The first is that it was not possible to calibrate the absolute risk function perfectly to the rate of alcohol specific deaths from 2001-2019. This was an issue because the two beta slope parameters, one for males and one for females, were estimated and fixed based on the best model setting identified from the calibration procedure. Therefore, the use of these fixed slope parameters in the ABM introduce error inherent to the model. To attempt to identify the best model setting 15,000 samples were taken for two parameters, however the best model setting still produced an implausibility value of approximately 0.90. It is clear from the results in Chapter 9, Figure 9.3 that alcohol specific deaths are slightly underestimated from the year 2001-2008 and slightly overestimated from 2009-2019. This directly corresponds to the time frame for each survey that was used to estimate alcohol consumption. From 2001-2006, the General Household Survey was used to estimate alcohol consumption, however from 2008-2019 the Scottish Health Survey was used to estimate alcohol consumption. Therefore, the reason it was not possible to identify better fitting parameter settings is likely due to the alcohol consumption data used in the microsimulation model.

Another technical limitation of the ABM was that a burn in period was not implemented in the model. The individual agents entered the model with no history of successes or failures to adapt to risk, prevention or treatment information. This does not accurately reflect reality as individuals will have been exposed to this type of information in some cases for decades before 2001. Given time constraints it was not possible to explore and test the impact of differing lengths of burn in period on the results as to run 10,000 samples 5 times took approximately four days to run on 50 cores. It would be difficult to initialize a ratio of successes to failures in the absence of data on socioeconomic variables of the individuals in the model from years prior to 2001.

Another limitation of the ABM work was that only one possible interpretation of FCT was explored while other credible interpretations of the theory may have yielded different outcomes. One paper explored the impact of implementing the theory of planned behaviour in

an ABM in four different ways (altering the architecture, the factors impacting agent decision making, the representation of these factors and the data used) and found quantitative and qualitative differences in the simulated outcomes based on changes to these four modelling domains (288). To address this the assumptions, model structure and data used to construct the ABM where made explicit both in chapter 9 and Appendix C.

An additional limitation of this model is that it was not calibrated to an intermediary behavioural target before connecting the behaviour outcomes to risk of alcohol specific death. The behavioural outcomes from the FCT mechanism were instead used to adapt the absolute risk of death based on alcohol consumption, and the targets for this model were alcohol specific deaths split by SIMD quintile. However, the majority of existing ABMs tend to use behavioural targets as opposed to mortality targets in model calibration. For example, in the alcohol field ABMs have been used to understand patterns of alcohol consumption, specifically the prevalence, frequency and quantity of alcohol consumed (208). However, as this model did not aim to understand the causes of alcohol consumption and behavioural outcomes are specified in FCT beyond successes and failures to adapt to risk, prevention and treatment information, it was not possible to obtain behavioural targets to calibrate this model. While there may be data on accessing treatment and other successful behavioural adaptation, it is not feasible to obtain targets relating to failures to adapt.

10.5 Future Directions

The first future direction arising from the limitation section of this thesis is to identify or commission a survey that contains all the variables of interest; both the socioeconomic variables required to represent fundamental resources and data on individual level alcohol consumption. To do so may require identifying data sources outside of the Scottish setting and as a result testing the model in a different context. For example, there is a readily available microsynthetic population of the United States, CASCADEPOP, that includes information on individual's employment, education, income and alcohol use (289). Some additional work would be required to identify target data for alcohol specific deaths split by deprivation and

whether the data sources used to generate CASCADEPOP contains variables that could be used to represent the fundamental resource power. However, testing the model in the US setting could address the current data limitations associated with testing the model in the Scottish setting. Testing the model in an alternative setting would also resolve the issue of calibrating the absolute risk function to alcohol specific deaths at the population level, specifically if alcohol consumption is estimated from the same data source.

Additionally, future research attempting to improve the existing model should explore the impact of introducing a burn in period to the model to more accurately identify the mechanisms of morphostasis (the mechanisms that sustain the AHP) as opposed to morphogenesis (the mechanisms that generate the AHP). An additional model improvement that could be explored in future research would be the introduction the role of resources at the community level which have been purported to facilitate or prohibit behavioural adaptation in more recent descriptions of FCT (277).

Finally, another improvement that could be made to this model in future research would be to identify an intermediary behavioural target to calibrate to that can represent the behavioural outcomes associated with FCT. Given that ABMs capture the actions and interactions of agents (193), they are potentially better suited to explore the causes of behavioural phenomenon. This is somewhat reflected in the existing literature given that the majority of ABMs in the field of public health focus on behaviours such as alcohol consumption (208,235), smoking (280), diet (196) and physical activity (290). That is not to say that it's not possible to use ABMs to estimate health outcomes (284,285), however the link from behaviour to health may require greater specificity. One possible way to achieve this is to introduce interim behavioural targets and calibrate the model parameter to that data prior to linking the behavioural outcomes to physical health outcomes. It is particularly difficult to define and obtain data for the behavioural outcomes associated with FCT. However, a survey could be developed to collect the required behavioural data which asked questions such as how often did you access treatment, did you ever intend to access treatment but did not actually attend

etc. The development of such a survey would require years of research, testing and data collection.

Finally, the work conducted during this PhD identified many potential explanations for the AHP but did not identify a cause. Future research should also explore alternative explanations for the AHP, which are identified in Chapters 4 and 5. Specifically, Chapter 5 details several potential pathways for future research seeking to understand the causes of the AHP using health inequality theory. ABM as a method is particularly well suited to understand how the actions and interactions of individuals with each other and the environment may result in observable phenomenon (160,193). Therefore, it is appropriate to use ABM to explore explanations for the AHP that explicitly detail the role of individual action and interaction. For example, the social support model suggests that different types of social support are used to protect individuals from the impacts of stressful life events (240). However, other potential explanations for the AHP are not as well suited to ABM methodologies. For example, explanations associated with the social and political context may be more appropriately explored using cross-national comparison studies to understand how the differences in policies between countries impact on inequalities in alcohol harm both within and between countries (186). Regardless of the methodological approach, future work should endeavour to uncover the underlying causes of the paradox, without the knowledge of the causes of this phenomenon we cannot seek to intervene to reduce inequalities in alcohol harm.

10.6 Research and policy implications

The use of a mechanism-based approach for quantitative simulation in the context of complex public health problems allows researchers and policy makers to understand why these problems come to be. In the process of identifying the mechanisms that generate observable phenomenon such as inequalities in alcohol harm, or inequalities in health generally, it is possible to identify the levers that could be used address public health problems.

ABMs are particularly well suited to test the impact of policy interventions and inform policy decisions (216). Unfortunately, it was not possible to use the model constructed for this thesis

to test policy interventions as the model could not reproduce the socioeconomic gradient in alcohol specific deaths. This confirms the difficulties of using computation modelling to inform policy decisions. Specifically, that timescales are often short (291) and it takes a substantial period of time to implement and test an ABM which in the end was unable to reproduce the trends necessary to simulate the impact of policy intervention.

However, if the model had been able to reproduce trends in alcohol-specific deaths in Scotland then it could have been used to examine the effects of potential interventions or policies on inequalities in alcohol harm. For example, given that this model simulated the mechanisms described in FCT it would have been possible to test the effects of resource redistribution on alcohol-specific deaths. The model could have been used to simulate a policy which introduced greater resource taxes for the most affluent individuals and then distributed this taxed resource to the most disadvantaged in the model. Hypothetically this would have changed the ratio of successes and failures these individuals experienced in the simulation, and as a result their risk of alcohol-specific death. Equally it would have been possible to test the impact of reducing the resource threshold or increasing the proportion of individuals informed of the event from the communicatory entity. While reducing the resource threshold and increasing the proportion of individuals informed of an event are attempts to reduce inequalities, they would also make it easier for more advantaged individuals to adapt and therefore may have the unintended consequence of increasing inequalities in alcohol-specific death. Using the model for policy and intervention simulation is particularly important for identifying the policy levers that will reduce (or increase) inequalities in alcohol-specific deaths.

There are several examples of how ABMs can be used to simulate the impact of policy interventions in the context of alcohol. For example, one study that assessed the impact of alcohol taxation on rates of violent victimisation using an ABM which simulated the effects of alcohol price elasticities, alcohol consumption and beverage preferences, found that taxation policies reduced inequalities in alcohol violence (211). Another ABM study examined the impact of closing alcohol outlets in areas with the highest levels of violence and found that

such an intervention did not change alcohol consumption or alcohol related problems in those areas (212). A final example investigated the impact of restricting firearm purchases based on alcohol and drug-related misdemeanors and found that this intervention reduced firearm violence in vulnerable populations (292). Therefore, if the model developed for this PhD could explain the AHP it would have been possible to test the impact of interventions such as the redistribution of fundamental resources, targeting more deprived communities with risk, prevention and treatment information, and lowering the resource threshold for the most deprived, on inequalities in alcohol specific death.

10.7 Conclusion

This PhD used a mechanism-based six step approach to advance our understanding of socioeconomic inequalities in alcohol harm in Scotland from 2001-2019. This is the first study to draw on health inequality theory and use ABM as a method to understand the causes of the AHP.

Risk behaviour related explanations have been central to research exploring the causes of the AHP, while other hypothetical explanations often related to explanations for health inequalities more generally remain untested. Health inequality theory has been under-utilised in the existing literature and there are many potential opportunities for future research investigating the causes of the AHP to draw on health inequality theory.

A mechanism-based approach can tell us how observable phenomenon are generated and combining this approach with ABM methodology is particularly well suited to test explanations for the AHP that involve a role for individuals' interactions with each other and their environment.

FCT as implemented in the ABM presented in this thesis cannot explain the socioeconomic gradient in alcohol harm observed in Scotland. However, there are several model limitations that should be addressed in future research before we can conclude that FCT cannot explain the AHP.

Future research should continue to investigate the causes of the paradox either using a mechanism-based approach and ABM as illustrated in this thesis or other research methods to test the explanatory values of alternative explanations for AHP. Until we understand the causes of this phenomenon we cannot identify potential solutions to intervene and reduce inequalities in alcohol harm between the most and least deprived groups in society.

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Appendices

Appendix A: Supplementary Material Published alongside the Systematic Review

Table S1: Systematic Search Strategy

Concept	Search terms				
Alcohol (.mp.) (MEDLINE & Embase)	Alcohol* adj3 drink*	Heavy adj3 drink*	Binge drink*	*alcohol consumption/ or *binge drinking/ or *heavy drinking	Alcohol*.ti.
Alcohol (PsychInfo)	Alcohol* adj3 drink* (.mp.)	Alcohol drinking patterns/	Heavy adj3 drink* (.mp.)	*alcohol consumption/ OR *binge drinking/ OR *heavy drinking/	Alcohol*.ti.
Health Inequalities (MEDLINE & Embase)	Health Status Disparities/ or exp Socioeconomic Factors/	Health adj2 inequalit* (.mp.)	Socioeconomic or socio-economic (.mp.)		
Health Inequalities (PsychInfo)	Health status disparities (.mp.)	Socioeconomic status/	Health adj2 inequalit* (.mp.)		
Socioeconomic Status (MEDLINE & Embase)	Disadvantage* OR inequit* OR inequal* OR poverty OR low income OR unemploy* OR employ	High income OR deprived OR social class OR upper class OR middle class OR working class	Deprivation (.mp.)		
Socioeconomic Status (PsychInfo)	Disadvantage* OR inequit* OR inequal* OR poverty OR low income OR unemploy* OR employ (.mp.)	High income OR deprived OR social class OR upper class OR middle class OR working class (.mp.)	Deprivation (.mp.)	*social class/ OR *socioeconomic status/	

Concept	Search terms			
Exclusions for: MEDLINE & Embase	Therapeutics/ OR psychotherapy/ OR intervention.ti. OR brief intervention.ab. OR effectiveness.ti.	(Brain OR bacter* OR pathogen* OR methyl* OR memor* OR cortex OR neur* OR temporal).ti.	(Africa* OR chin* OR india* OR Russia* OR thai* OR vietn* OR Uganda OR brazil OR Nepal).ti.	Addiction.ti. OR rehabilitation.mp. OR psych*.ti. OR rats.mp. OR vehicle.mp.
Exclusions for: PsycInfo	(Addiction OR rehabilitation OR alcoholi*).ti.	Therapeutics/ OR psychotherapy/ OR intervention.ti. OR brief intervention.ab. OR effectiveness.ti.		

S2: Critical Appraisal

Quality Assessment

Overall, the quality of included papers was assessed as good. The key quality concern was non-response bias in cross-sectional studies. Many used secondary data and therefore did not report response rates (e.g. (18,23)) while others reported response rates but did not take measures to address potential biases (e.g. data was not weighted and there were no attempts to contact or categorise non-responders) (17,24,25) (see Tables S2 in supplementary material for full details). Another limitation was studies mainly used self-report measures of consumption and SEP. However, these were often established and validated measures. A focus on physical health harms as an objective outcome measure was a strength of included studies.

NB: Numbers in the top row represent question numbers from respective checklists.

Key: ✓ = meets criteria, DR = do not report, N/A = not applicable, ~ = partially meets criteria, X = does not meet criteria

Table S2.1: AXIS Critical Appraisal for included cross-sectional studies.

	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.	14.	15.	16.	17.	18.	19.	20.
(1)	✓	✓	✓	✓	✓	✓	DR	✓	✓	✓	✓	✓	DR	N/A	✓	✓	✓	✓	✓	✓
(2)	✓	✓	✓	✓	✓	✓	X	✓	✓	✓	✓	✓	X 23.3%RR	X	N/A	✓	✓	✓	✓	✓
(3)	✓	✓	✓	✓	✓	✓	N/A	✓	✓	✓	✓	✓	N/A	N/A	N/A	✓	✓	X	✓	✓
(4)	✓	✓	✓	✓	✓	✓	X	✓	✓	✓	✓	✓	X (25.5%RR, 65.8% of those then returned the questionnaire)	X	✓	✓	✓	✓	DR	✓
(5)	✓	✓	✓	✓	✓	✓	✓	✓	~	✓	✓	✓	✓	X	DR	✓	✓	✓	✓	✓
(6)	✓	✓	✓	✓	✓	✓	N/A	✓	✓	✓	✓	✓	✓	N/A	N/A	✓	✓	X	✓	✓
(7)	✓	✓	✓	✓	✓	✓	N/A	✓	✓	✓	✓	✓	✓	N/A	N/A	✓	✓	✓	✓	✓
(8)	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	N/A	N/A	✓	✓	✓	✓	✓
(9)	✓	✓	✓	✓	✓	✓	N/A	✓	✓	✓	✓	✓	N/A	N/A	N/A	✓	✓	✓	✓	✓
(10)	✓	✓	✓	✓	✓	✓	N/A	✓	✓	✓	✓	✓	✓	N/A	N/A	✓	✓	✓	DR	✓
(11)	✓	✓	✓	✓	✓	✓	N/A	✓	✓	DR	✓	✓	N/A	N/A	N/A	✓	✓	X	DR	✓
(12)	✓	✓	✓	✓	✓	✓	N/A	✓	✓	✓	✓	✓	N/A	N/A	N/A	✓	✓	✓	X	✓
(13)	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	N/A	N/A	✓	✓	✓	DR	✓
(14)	✓	✓	✓	✓	✓	✓	X	✓	✓	✓	✓	✓	~	N/A	N/A	✓	✓	✓	✓	✓

(15)	✓	✓	✓	✓	✓	✓	N/A	✓	✓	✓	✓	✓	N/A		N/A	N/A	✓	✓	✓	DR	N/A
(16)	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓		✓	✓	✓	✓	✓	✓	N/A
(17)	✓	✓	✓	✓	✓	✓	N/A	✓	~	✓	✓	✓	N/A		N/A	DR	✓	✓	✓	✓	✓
(18)	✓	✓	✓	✓	✓	X	X	✓	✓	✓	✓	✓	X		X	✓	✓	✓	✓	✓	✓
(19)	✓	✓	✓	✓	✓	✓	N/A	✓	~	✓	✓	✓	✓		X	DR	✓	✓	✓	✓	✓

Table S2.2: CASP Critical Appraisal for included case-control studies.

	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.
(20)	✓	✓	✓	✓	✓	N/A	✓	N/A	N/A	✓	✓	✓
(21)	✓	✓	✓	✓	✓	N/A	✓	N/A	N/A	✓	✓	✓
(22)	✓	✓	✓	✓	✓	N/A	✓	N/A	N/A	✓	✓	✓
(23)	✓	✓	✓	✓	✓	N/A	✓	N/A	N/A	✓	✓	✓

Table S2.3*:* CASP Critical Appraisal for included cohort studies.

	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.	14.
(24)	✓	✓	✓	✓	✓	✓	✓	✓	N/A	✓	✓	✓	✓	N/A
(25)	✓	✓	✓	✓	✓	✓	✓	✓	N/A	✓	✓	✓	✓	N/A
(26)	✓	✓	✓	✓	✓	✓	✓	✓	N/A	✓	✓	✓	✓	N/A
(27)	✓	✓	✓	✓	✓	✓	✓	✓	N/A	✓	✓	✓	✓	N/A
(28)	✓	✓	✓	✓	✓	✓	✓	✓	N/A	✓	✓	✓	✓	N/A
(29)	✓	✓	✓	✓	X	X	✓	✓	N/A	DR	✓	✓	✓	N/A
(30)	✓	✓	✓	✓	✓	✓	✓	✓	N/A	✓	✓	✓	✓	N/A
(31)	✓	✓	✓	✓	✓	✓	✓	✓	N/A	✓	✓	✓	✓	N/A
(32)	✓	✓	✓	✓	X	X	✓	✓	N/A	✓	✓	✓	✓	N/A
(33)	✓	✓	✓	✓	✓	✓	✓	✓	N/A	✓	✓	✓	✓	N/A
(34)														
(35)	✓	✓	✓	✓	✓	✓	✓	✓	N/A	✓	✓	✓	✓	N/A
(36)	✓	✓	✓	✓	X	X	✓	✓	N/A	✓	✓	✓	✓	N/A
(37)	✓	✓	✓	✓	X	X	✓	✓	N/A	✓	✓	✓	✓	N/A
(38)	✓	✓	✓	✓	✓	✓	✓	✓	N/A	✓	✓	✓	✓	N/A
(39)	✓	✓	✓	✓	✓	✓	✓	✓	N/A	✓	✓	✓	✓	N/A
(40)	✓	✓	✓	✓	X	X	✓	✓	N/A	DR	✓	✓	✓	N/A
(41)	✓	✓	✓	✓	✓	✓	✓	✓	N/A	✓	✓	✓	✓	N/A
(42)	✓	✓	✓	✓	✓	✓	✓	✓	N/A	✓	✓	✓	✓	N/A
(43)	✓	✓	✓	✓	X	X	✓	✓	N/A	DR	✓	✓	✓	N/A
(44)	✓	✓	✓	✓	✓	✓	✓	✓	N/A	✓	✓	✓	✓	N/A
(45)	✓	✓	✓	✓	X	X	✓	✓	N/A	✓	✓	✓	✓	N/A
(46)	✓	✓	✓	✓	X	X	✓	✓	N/A	✓	✓	✓	✓	N/A
(47)	✓	✓	✓	✓	✓	✓	N/A	✓	✓	✓	✓	✓	✓	N/A
(48)	✓	✓	✓	✓	✓	✓	N/A	✓	✓	✓	✓	✓	✓	N/A
(49)	✓	✓	✓	✓	X	X	✓	✓	N/A	✓	✓	✓	✓	N/A
(50)	✓	✓	✓	✓	✓	✓	X	✓	N/A	✓	✓	✓	✓	N/A
(51)	✓	✓	✓	✓	X	X	✓	✓	N/A	✓	✓	X	✓	N/A
(52)	✓	✓	✓	✓	X	X	✓	✓	N/A	DR	✓	✓	✓	N/A

Table S2.4*:* CASP Quality Appraisal for included qualitative studies

	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.
(53)	✓	✓	✓	✓	✓	X	✓	✓	✓	✓
(54)	✓	✓	✓	✓	✓	X	✓	✓	✓	✓

Table S2.5: CASP Quality Appraisal for included systematic reviews.

	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.
(55)	✓	✓	✓	✓	✓	N/A	✓	✓	✓	N/A
(56)	✓	✓	✓	✓	✓	N/A	✓	✓	✓	N/A
(57)	✓	✓	✓	X	N/A	N/A	N/A	✓	✓	N/A

S3: PRISMA Checklist

Section/topic	#	Checklist item	Reported on page #
TITLE			
Title	1	Identify the report as a systematic review, meta-analysis, or both.	1
ABSTRACT			
Structured summary	2	Provide a structured summary including, as applicable: background; objectives; data sources; study eligibility criteria, participants, and interventions; study appraisal and synthesis methods; results; limitations; conclusions and implications of key findings; systematic review registration number.	2
INTRODUCTION			
Rationale	3	Describe the rationale for the review in the context of what is already known.	3
Objectives	4	Provide an explicit statement of questions being addressed with reference to participants, interventions, comparisons, outcomes, and study design (PICOS).	3
METHODS			
Protocol and registration	5	Indicate if a review protocol exists, if and where it can be accessed (e.g., Web address), and, if available, provide registration information including registration number.	4
Eligibility criteria	6	Specify study characteristics (e.g., PICOS, length of follow-up) and report characteristics (e.g., years considered, language, publication status) used as criteria for eligibility, giving rationale.	4
Information sources	7	Describe all information sources (e.g., databases with dates of coverage, contact with study authors to identify additional studies) in the search and date last searched.	4
Search	8	Present full electronic search strategy for at least one database, including any limits used, such that it could be repeated.	S1
Study selection	9	State the process for selecting studies (i.e., screening, eligibility, included in systematic review, and, if applicable, included in the meta-analysis).	4
Data collection process	10	Describe method of data extraction from reports (e.g., piloted forms, independently, in duplicate) and any processes for obtaining and confirming data from investigators.	4, 5
Data items	11	List and define all variables for which data were sought (e.g., PICOS, funding sources) and any assumptions and simplifications made.	4, 5

Risk of bias in individual studies	12	Describe methods used for assessing risk of bias of individual studies (including specification of whether this was done at the study or outcome level), and how this information is to be used in any data synthesis.	5, S2
Summary measures	13	State the principal summary measures (e.g., risk ratio, difference in means).	N/A
Synthesis of results	14	Describe the methods of handling data and combining results of studies, if done, including measures of consistency (e.g., I^2) for each meta-analysis.	N/A

Section/topic	#	Checklist item	Reported on page #
Risk of bias across studies	15	Specify any assessment of risk of bias that may affect the cumulative evidence (e.g., publication bias, selective reporting within studies).	S2
Additional analyses	16	Describe methods of additional analyses (e.g., sensitivity or subgroup analyses, meta-regression), if done, indicating which were pre-specified.	N/A
RESULTS			
Study selection	17	Give numbers of studies screened, assessed for eligibility, and included in the review, with reasons for exclusions at each stage, ideally with a flow diagram.	5
Study characteristics	18	For each study, present characteristics for which data were extracted (e.g., study size, PICOS, follow-up period) and provide the citations.	5, 6
Risk of bias within studies	19	Present data on risk of bias of each study and, if available, any outcome level assessment (see item 12).	S2
Results of individual studies	20	For all outcomes considered (benefits or harms), present, for each study: (a) simple summary data for each intervention group (b) effect estimates and confidence intervals, ideally with a forest plot.	N/A
Synthesis of results	21	Present results of each meta-analysis done, including confidence intervals and measures of consistency.	N/A
Risk of bias across studies	22	Present results of any assessment of risk of bias across studies (see Item 15).	S2
Additional analysis	23	Give results of additional analyses, if done (e.g., sensitivity or subgroup analyses, meta-regression [see Item 16]).	N/A
DISCUSSION			
Summary of evidence	24	Summarize the main findings including the strength of evidence for each main outcome; consider their relevance to key groups (e.g., healthcare providers, users, and policy makers).	9
Limitations	25	Discuss limitations at study and outcome level (e.g., risk of bias), and at review-level (e.g., incomplete retrieval of identified research, reporting bias).	10

Conclusions	26	Provide a general interpretation of the results in the context of other evidence, and implications for future research.	9, 10, 11
FUNDING			
Funding	27	Describe sources of funding for the systematic review and other support (e.g., supply of data); role of funders for the systematic review.	1

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Appendix B: Overview, Design concepts and Details (ODD) protocol for FCT AB

R Script for the propensity score matching procedure.

```
library(dplyr)
library(tidyr)
library(MatchIt)
library(optmatch)

gc()
memory.limit(size=500000)

MicroData <- read.csv("Imputed_MicroData.csv")

summary(MicroData)

GHS <- read.csv("CleanGHS.csv")

summary(GHS)

GHS$sex <- as.factor(GHS$sex)
GHS$marstat <- as.factor(GHS$marstat)
GHS$economic_status <- as.factor(GHS$economic_status)

GHS_subset <-
  GHS %>%
  select(age, sex, marstat, economic_status, equWeeklyHHInc, drating) %>%
  rename(marital_status = marstat) %>%
  rename(economic_activity = economic_status) %>%
  rename(EquNetHHInc = equWeeklyHHInc)

UNIQID <- rep(c("X"), 16443)

GHS_subset <- cbind(GHS_subset, UNIQID)

summary(GHS_subset)

#### Format MicroData variables to match GHS ####

MicroData$sex <- as.factor(MicroData$sex)
MicroData$marital_status <- as.factor(MicroData$marital_status)
MicroData$economic_activity <- as.factor(MicroData$economic_activity)

summary(MicroData$marital_status)

MicroData <-
  MicroData %>%
  mutate(marital_status = case_when(marital_status == "Never married" ~ "Single",
                                    TRUE ~ as.character(marital_status)))

MicroData$marital_status <- as.factor(MicroData$marital_status)

summary(MicroData$economic_activity)

MicroData <-
  MicroData %>%
  mutate(economic_activity = case_when(economic_activity == "Family care" ~ "Economically
Inactive",
                                       economic_activity == "FT studt, school" ~ "Student",
                                       economic_activity == "Gvt trng scheme" ~ "Employed",
                                       economic_activity == "LT sick, disabld" ~ "Economically Inactive",
                                       economic_activity == "Maternity leave" ~ "Employed",
```

```
                              economic_activity == "Retired" ~ "Economically Inactive",
                              economic_activity == "Self-employed" ~ "Employed",
                              TRUE ~ as.character(economic_activity)
                                ))

MicroData$economic_activity <- as.factor(MicroData$economic_activity)

summary(MicroData$economic_activity)

#### Add the survey variable to both datasets ####

Surv<- rep(c(0), each = 16443)

GHS_subset <- cbind(GHS_subset, Surv)

Surv <- rep(c(1), each = 14803)

MicroData <- cbind(MicroData, Surv)

#### Subset the MicroData to just contain the variables that match the GHS ####

MicroData_subset <-
  MicroData %>%
  select(UNIQID, age, sex, marital_status, economic_activity, EquNetHHInc, Surv)

drating <- rep(c("X"), each = 14803)

MicroData_subset <- cbind(MicroData_subset, drating)


#### Remove 15 year olds from the MicroData ####

MicroData_subset <-
  MicroData_subset %>%
  filter(age >= 16)

#### Check both datasets match ####

summary(GHS_subset)
summary(MicroData_subset)

#### Merge both datasets together ####

PropensityScoreDF <- rbind(GHS_subset, MicroData_subset)


#### Propensity Score model ####

psFormula <- Surv ~ age + sex + marital_status + economic_activity

glm1 <- glm(psFormula, family = binomial, data = PropensityScoreDF)
summary(glm1)

pscores <- fitted(glm1)
PropensityScoreDF$pscores <- pscores

#### Matching procedure ####

m.out.nearest = matchit(Surv ~ age + sex + marital_status +economic_activity,
            data = PropensityScoreDF, method = "nearest",
```

```
            ratio = 1)

summary(m.out.nearest)


#### Obtain match data ####

nearestMatchDF <- match.data(m.out.nearest)

write.csv(nearestMatchDF, "matchedData.csv")

GHS_propensity <-
  nearestMatchDF %>%
  filter(UNIQID == "X")

GHS_alcohol <-
  GHS_propensity %>%
  select(subclass, drating)

MicroData_propensity <-
  nearestMatchDF %>%
  filter(Treat == 1)

MicroData_ID <-
  MicroData_propensity %>%
  select(UNIQID, subclass)

#### Assign alcohol consumption to microdata ####

MicroData_alcohol <- left_join(MicroData_ID, GHS_alcohol, by = "subclass")

write.csv(MicroData_alcohol, "MicroDataAlcohol.csv")
```

Appendix C: Overview, Design concepts and Details (ODD) protocol for FCT ABM.

Using Agent-based Modelling to understand the causes of the Alcohol Harm Paradox: A Scottish case study investigating Fundamental Cause Theory: ODD Protocol

Overview

1. Purpose and patterns

This model aims to use mechanisms from Fundamental Cause Theory (FCT) to generate patterns in alcohol-related mortality by SIMD quintile in Scotland between 2001 and 2019. In FCT it is purported that the root cause of health inequality is the unequal distribution of fundamental resources: money, power, prestige, knowledge and social connections, that generate and sustain differential health outcomes between socioeconomic groups (96,97). Advantaged individuals have greater access to these resources and therefore can deploy them to adapt to risk or prevention information and uptake new treatments, whereas the disadvantaged are not afforded the opportunity to do so. It is this lack of adaptation that results in worse health outcomes for more disadvantaged socioeconomic groups. This theory has been used to attempt to explain differences in mortality outcomes from smoking-related causes (181). Here we use this theory to attempt to explain differences in alcohol-related mortality.

The patterns we use as criteria for evaluating the model's suitability for purpose are the trends in alcohol-related mortality in the Scottish population between 2001 and 2019. These trends are used as calibration targets for simulation and are based on data from the National Records for Scotland.

The main application of this model is to explore the explanatory value of Fundamental Cause Theory in explaining inequalities in alcohol-related harm.

2. Entities, state variables and scales

Agents in the model are individual's representative of the population of Scotland in 2001. They are sampled from a representative microsimulation model of the Scottish population. Briefly, agents are initialized using the British Household Panel Survey 2001, and then reweighted to the Scottish Census 2001 data. Agents have several attributes that they are initialized at baseline with (see Table 1). Temporal scale is set to weeks because alcohol use in this model is measured as average units per week and it is more probable that FCT "events" would occur less frequently than at daily timesteps. A tick in this model is equivalent to 1 week.

Table 1: *Attributes of individual agents in the simulation. Some attributes are initialized from the microsimulation model and some are allocated during the simulation.*

Variable name	Variable type and units	Meaning	Data source
Age	Numeric	Agent's age in years, range 16-95.	Microsimulation
Age Group	Categorical	Agent's age categorized into groups coded "16-24", "25-34", "35-44", "45-54", "55-64", "65-74" and "75+".	Microsimulation
Sex	Binary	Agent's sex, coded "Male", "Female".	Microsimulation

Education Group	Categorical	Agent's Education Group, coded, "No educational qualifications", "Group 1", "Group 2", "Group 3", "Group 4" .	Microsimulation
Original Quintile	Categorical	Agent's deprivation quintile at point of initialization, coded 1-5, where 1 is the most deprived and 5 is the least deprived quintile.	Microsimulation
Deprivation Quintile	Categorical	Agent's deprivation quintile for years post 2001, coded 1-5, where 1 is the most deprived and 5 is the least deprived.	ABM Simulation
Average Units Per Week	Numeric	The average number of units of alcohol consumed per week.	Microsimulation
Drinking Status	Categorical	Agent's drinking status based on their average units per week, coded "Abstainer", "Light Drinker", Moderate Drinker", "Heavy Drinker" and "Risky Drinker".	Microsimulation
Number Connections	Numeric	Number of connections to other agents formed during the social network set up procedure.	ABM Simulation
Power	Numeric	Measurement of an individual's power on a scale of 0-1 constructed from variables representing the ability and capacity to achieve goals.	Microsimulation
Prestige	Numeric	Measurement of an individual's prestige on a scale of 0-1 constructed from variables representing social grade, occupation	Microsimulation

		and managerial roles.	
Money	Numeric	Measurement of an individual's money on a scale of 0-1 constructed from Equivalent Net Household Income and Equivalent Real Household Annual Income.	Microsimulation
Knowledge	Numeric	Measurement of an individual's knowledge on a scale of 0-1 constructed from education level.	Microsimulation
Social Connections	Numeric	Measurement of an individual's social connections on a scale of 0-1 constructed from both the count of connections and the quality (those connected to individuals with greater prestige than themselves given prestige bonus)	ABM Simulation
Total Resources	Numeric	Total sum of FCT resources: Power, Prestige, Money, and Knowledge.	ABM Simulation
Probability Communicate	Numeric	The likelihood that agents will communicate information about an event to a social connection.	Sampled parameter
Knowledge Gain	Numeric	A value added to the agents existing knowledge in the instance where an agent successfully adapts to an event and if the knowledge of that agent is not already reached the maximum value 1.	Sampled parameter
Strategy Multiplier	Numeric	The number used to enhance the number of available resources an individual possesses	Sampled parameter

		for the tick that it is applied.	
Resource Depletion	Numeric	The number subtracted from the total resource pool of individuals when they successfully deploy resources to adapt to an event.	Sampled parameter

3. Process overview & scheduling

The process in the simulation model follows the micro-macro scheme described in MBSSM architecture (160). At the micro level, there are agents that represent individuals. At the macro level, there are two social structural entities: the communicator and the environment.

Each week a series of three different types of mechanisms are carried out. Firstly, situational mechanisms (macro-to-micro): the communicator entity assesses whether an event (representing new prevention, risk information or treatment) occurs during each tick, and if an event occurs in that tick the communicator entity relays this new information to a proportion of the individuals in the simulation. Secondly, the individuals in the simulation perform action mechanisms: the first of which is to select an event from their event list to attempt to solve, then they can communicate that event to another individual in their social network, following this agents check whether they are aware of any strategies to solve the event that they have selected (if they are aware of a strategy this enhances the resources available to them for this tick), individuals then attempt to adapt to the event by deploying the total resources available to them in the current tick and finally individuals can communicate their own strategy to another individual in their social network if they successfully adapt to the event. Finally, the transformational mechanism is the individuals can swap SIMD quintile which changes the environment as they swap from one community to another (this is based on an existing model described in section 7).

Scheduling: On each week of the simulation, situational, action and transformational mechanisms are performed.

Annually, every 52 ticks of the simulation, the probability that an agent will die from an alcohol-related cause is calculated, by calculating the ratio of successful adaptations to unsuccessful adaptations which is then used to modify the absolute risk of death from an alcohol-related cause. Individuals also may die of a non-alcohol related cause, this is based on Scottish lifetables obtained from:https://www.ons.gov.uk/peoplepopulationandcommunity/birthsdeathsandmarriages/lifeexpectancies/datasets/nationallifetablesscotlandreferencetables which are adjusted by removing the proportion of all-cause mortality attributed to alcohol (31.3 deaths per 100,000 males, and 12.7 deaths per 100,000 females) (https://www.nrscotland.gov.uk/files/statistics/alcohol-deaths/2020/alcohol-specific-deaths-20-report.pdf).

For each individual, monetary resources are replenished, and education level and drinking are updated annually.

Design

4. Design concepts

Basic Principles

Situational mechanisms

At the macro level, the communicator entity assesses whether an event has occurred at the beginning of the tick. This is based on the probability of an event which is a calibrated value. If an event occurs in that tick the communicator entity stores it in a list representing event history. The current event is also stored numerically in a variable labelled total event. The communicator entity then communicates this event to a proportion of individuals in the simulation, this proportion is also a calibrated value.

Action mechanisms

At the micro level (see Figure 1 for an overview of actions at the micro-level), individuals can correctly perceive the event communicated to them from the communicator entity based on their own knowledge – conditional on knowledge being equal to or greater than the mean. If they successfully perceive the event they store it in a list variable. Individuals randomly select an event that they are aware of and are going to attempt to resolve on each week of the simulation. Events are stored numerically as they occur and can be communicated to agents directly from the communicator entity or indirectly from another individual in their social network. Individuals store events in three lists: all events, resolved events and unresolved events. If an event is unresolved it can be selected by the individual. There is no limit on how many times an event can appear in the list e.g., the list [1,1,1,2,3] represents that event 1 has been communicated to the individual three times – this increases the probability that this event is selected. However, once the event has been resolved all instances of that event are removed from the unresolved event list. So, in the example above when event 1 is resolved the list unresolved list would then become [2,3]. Individuals can only attempt to resolve one event per tick. Once selected, individuals can then communicate the active event to a randomly selected member of their social network based on the probability that they will communicate, this probability is a calibrated value.

Following this, individuals can check whether they are aware of any strategies to resolve the event they have selected. This is done by cross-checking whether the active event is present on another list variable called tips events. The variable tips event consists of a list of events that individuals have received strategies for from other individuals in their social network. If they have been provided with a strategy to resolve the active event then a strategy multiplier is used to calculate an enhanced value for total resources to deploy to resolve the event in that tick. To deploy their resources to attempt to adapt to the event, individuals assess whether their total resources is greater than the adaptation threshold, this threshold is a calibrated parameter. If they possess enough resources then they deploy these resources to successfully adapt, and the resource depletion value is subtracted from their total resource pool. If they successfully adapt this also comes with a knowledge gain which is added to the knowledge variable and the successful adaptation is recorded. However, if they do not possess enough resources to meet the adaptation threshold then they unsuccessfully adapt and this too is recorded.

Individuals also have the opportunity to communicate a strategy regarding one of their resolved events to another individual in their social network. Communicating a strategy is based on two conditions: that the individual has an event stored in their resolved event list and the probability that they will communicate with a social connection.

Transformational mechanisms

Annually individuals then have opportunity to swap locations with another individual belonging to a higher or lower SIMD quintile. This deprivation swap model is discussed in more detail in section 7.

Updating resources, education level and weekly alcohol consumption over time

Annually (every 52 ticks) the total resources available to individuals in the simulation is replenished based on the values from the microsimulation for money. Education level is also updated annually for individuals between the age of 16-30 whom have the opportunity to move up an educational group based on the proportion of individuals over the age of 30 belonging to each education group.

At baseline propensity score matching was used to give each individual an initial average units per week from the General Household Survey 2001 using age, sex, marital status and economic activity. Alcohol consumption, measured in average units per week is also updated annually for each individual. These values are taken from the General Household Survey (2002-2006) and the Scottish Health Survey (2008-2019). Each individual in synthetic population was given a value for average units per week for each year of the survey data by matching them to individuals from the surveys ranked by age group, sex, and economic status.

Alcohol-specific mortality

Alcohol-specific mortality was calculated annually. To calculate the risk of alcohol-specific mortality a model calculating the absolute risk from alcohol consumption (see Section 7 for more details) was adjusted using the ratio of successfully adapted events versus unsuccessfully adapted events. A risk modifier is first calculated by taking the number of failures and dividing by the sum of all successes and failures each individual has experienced in the simulation. To stabilise the risk modifier it is exponentiated using a βMod which is a parameterised value. The absolute risk previous calculated in is then adjusted by multiplying it by the calculated RiskModβ to produce an adjusted risk. A greater number of successes results in a reduced risk of alcohol-specific death, while a greater number of failures resulted in an increased risk.

Mortality from all other causes

Each individual could also die from a cause unrelated to alcohol. This was estimated using probabilities of death split by age and sex taken from the Scottish Lifetables for the years 2001-2020. To simplify the model an average probability across the time period for each age and sex group was used.

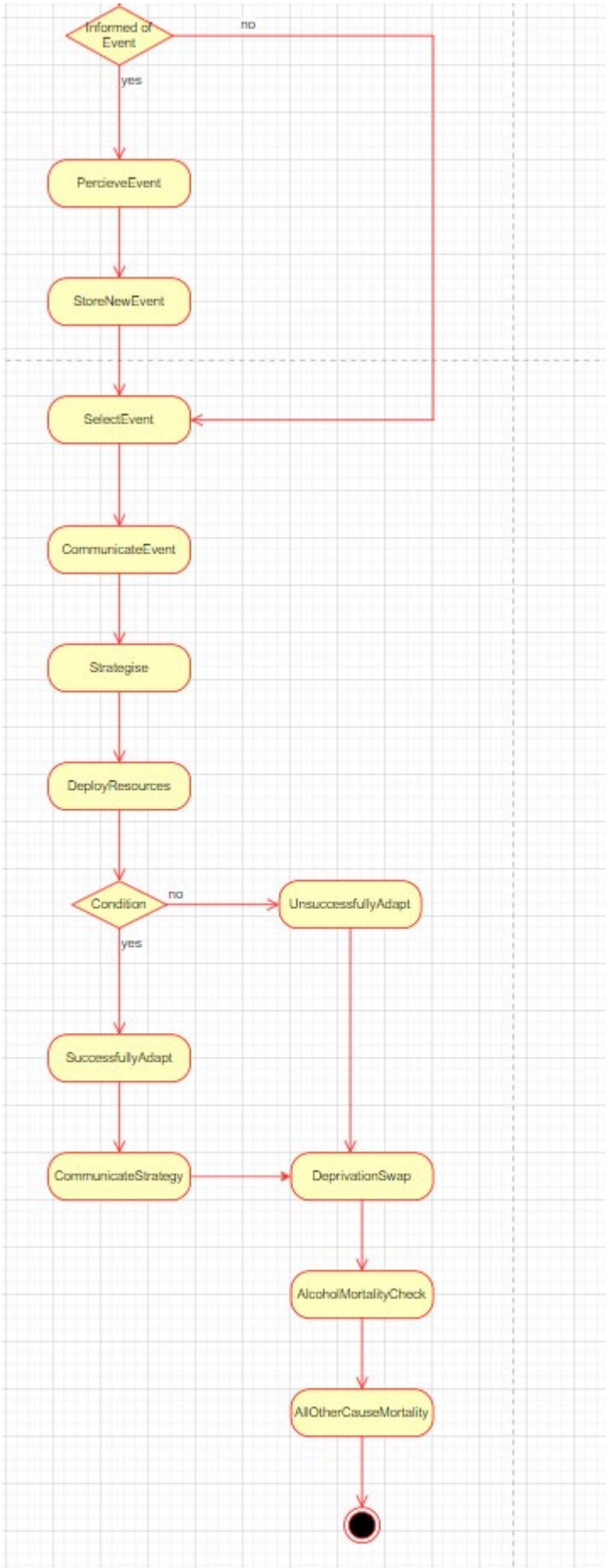


Figure 1: Activity Diagram of Individual Agents Action during the Simulation. *Note Deprivation Swap, Alcohol Mortality and Mortality from all other causes only occurs annually (every 52 ticks).

Emergence

The key outcome of the models are alcohol-specific deaths split by SIMD quintile over time.

Adaptation

No adaptation.

Objectives

No objectives.

Learning

Individuals gain knowledge when they successfully adapt to an event.

Prediction

No prediction.

Sensing

Individuals in the simulation perceive the event from the communicator entity however this is subject to the knowledge they possess.

Interaction

The communicator entity interacts with individual agents to inform them of the new event that has occurred in that tick. The simulated individuals also interact with one another communicating the event that they are actively trying to solve that tick and if they successfully resolve an event then they can communicate a strategy to a member of their social network.

Stochasticity

Stochasticity is used to set up the social network. Individuals assess their similarity to another individual based on a set of probabilities. The method to create the social network and the values for the probabilities were obtained from the authors of a published paper (212). Stochasticity is also used to update education group annually based on the proportion of individuals in each group in the baseline microsimulation population over the age of 30. Whether an event occurs in each tick, and when individuals communicate an event or adaptation strategy to a network connection is also determined stochastically. The deprivation quintile swap model (described in section 7) is also stochastic. Lastly, stochasticity is used to select which individual will die from an alcohol-specific cause (see section 7 for further description) or any other cause based on the average mortality rate from 2001-2019 split by age group and sex.

Collectives

The individuals belong to a social network that was constructed through a stochastic friend selection process based on spatial proximity, sex, education level, age and drinking status (see initialization for full details). During the simulation, modelled individuals could not create new network connections, however connections where broken when death occurred and could be made by new individuals migrating into the model.

Observation

Whether a simulated individual has died from an alcohol-specific cause is recorded in the model. The count of alcohol-related deaths by SIMD quintile where used to calculate the rate of alcohol-specific mortality.

Details

5. Initialisation

The individual agents are initialized using a microsimulation described in Section 7. We use survey data to provide baseline data for socio-demographics (including the fundamental cause related variables: money, knowledge, prestige and power) and average units of alcohol consumed per week.

A social network was initialised using the following procedure:

Social Network set up

➔ For each agent:
- Assign a number of target connections.
- While the selected individuals ("a") still has fewer than their assigned target connections and less than 10,000 attempts have been made to create social connections:
  - Randomly select another agent from the population ("b").
  - If agent "b" is not already linked to agent "a" and agent "b" does not have his/her assigned number of social connections:
    - Select a random number from 0 to 1.
    - If the random number is less than the assigned proportion of social network links that will be based on spatial proximity (25%):
      - Determine whether agents "a" and "b" are located within the same SIMD quintile.
    - Select a random number from 0 to 1.
    - If random number is less than the specified proportion of social network links that will be based on age (81.5%):
      - Determine whether agents "a" and "b" are in the same age group.
    - Select a random number from 0 to 1.
    - If random number is less than the specified proportion of social network links that will be based on sex (0.5%):
      - Determine whether agents "a" and "b" are of the same sex.
    - Select a random number from 0 to 1.
    - If random number is less than the specified proportion of social network links that will be based on education (75%):
      - Determine whether agents "a" and "b" have the same educational level.
    - Select random number from 0 to 1.
    - If random number is less than the specified proportion of social network links that will be based on drinking status (15%):
      - Determine whether agents "a" and "b" have the same drinking status.

- If agents “a” and “b” match on spatial proximity, age, gender, education level and drinking status:
    - Create a link between agents “a” and “b”.
    - Increase count of social connections for agents “a” and “b” by one.

6. Input data

We used several surveys to determine inputs for the model. Agents are individuals from the British Household Panel Survey (2001), reweighted using the Scottish 2001 Census (described in detail in Section 7). We also used propensity score matching to initialize agents with average units of alcohol consumed per week from the General Household Survey.

7. Submodels

There are three sub-models within the agent-based model. The first is a static microsimulation model of the population of Scotland in 2001. The second a deprivation swap model is used to estimate individual transitions in IMD quintile. Finally, there is a sub-model used to calculate the absolute risk of alcohol-related mortality.

a. Microsimulation model

Microsimulation modelling was used to generate a synthetic population representative of the Scottish population following the approach outlined by Wu and colleagues (293). This approach estimates the characteristics of a population using a combination of attribute rich individual data and geographical aggregate counts of individuals. The micro units in this simulation are individuals which are simulated by assigning them attributes from other data sources. We applied a static microsimulation approach to produce a synthetic population of individuals at Data Zone scale (500-1000 individuals per data zone) for Scotland, in the year 2001.

We used the Scottish Census 2001 which provided constraint tables containing aggregate counts for each data zone, and the British Household Panel Survey 2001, to provide the non-geographical and attribute rich individual level data. In order to calculate the weights for each individual in the survey data, linking variables available in both the aggregate and individual level data are required. A microsimulation model was created and run for adults (aged 16 and over).

We used the existing Flexible Modelling Framework (FMF) software to generate the microsimulation model. The FMF implements a simulated annealing approach to generate a microsynthetic population. This approach randomly selects individuals from the microdata (the British Household Panel Survey) and considers whether they should be admitted to a data zone based on the goodness of fit to the constraint tables (Census aggregate counts). This process is repeated and individuals are replaced in each data zone if the fit is improved. This software was developed by the University of Leeds as a tool to build microsimulation models (244).

The individual level data for this model is taken from the British Household Panel Survey (BHPS) collected in 2001. This survey began in 1991 and follows the same representative sample of individuals until 2009, when it was then replaced with the Understanding Society Survey. The BHPS’s 2001 sample consisted of 15,519 individuals sampled from across the UK and collects information on a range of topics including demographic, social, health,

economic and behavioural questions. The particular variables that make this survey suitable to operationalize Fundamental Cause Theory were equivalized net household income, equivalized real household annual income, level of qualification, social class, occupational grade, whether respondents had managerial experience, and questions that reflected the ability to achieve your goals. In addition to this information, the BHPS also includes sociodemographic information that overlaps with the variables in the aggregate area-level counts making it possible to generate a synthetic population. The sociodemographic variables used in this model were personal identifier, sex, age, economic activity, highest educational qualification and marital status. Ethnic group was not included as it was missing for more than half of the cases in the BHPS 2001. Missing cases for the other sociodemographic variables were excluded leaving 14,623 cases in the microdata. In order to match to the constraint aggregate counts the sociodemographic variables were formatted as shown in Table 2.

Table 2: *Summary of variables in the British Household Panel Survey adult microdata.*

Variable	Description	Values and categories
UNIQID	ID number assigned to each individual in the dataset	e.g., 1, 2, 3, 4, etc.
JSex/Jage	Sex and age group	M_16_24 (Male, aged 16-24)
		M_25_34 (Male, aged 25-34)
		M_35_49 (Male, aged 35-49)
		M_50_64 (Male, aged 50-64)
		M_65_74 (Male, aged 65-74)
		M_75+ (Male, aged 75 and over)
		F_16_24 (Female, aged 16-24)
		F_25_34 (Female, aged 25-34)
		F_35_49 (Female, aged 35-49)
		F_50_64 (Female, aged 50-64)
		F_65_74 (Female, aged 65-74)
		F_75+ (Female, aged 75 and over)
Jjbstat	Economic status	In_paid_employment
		Self_employed
		Student
		Unemployed
		Retired
		Other_economically_inactive
jqfedhi	Highest educational qualification	No qualifications
		Group 1 (GCE O-levels or equiv)
		Group 2 (GCE A-levels or equiv)
		Group 3 (CSE Grade 2-5, Scot Grade 4-5)
		Group 4 (Degree level)
jmlstat	Marital status	Married
		Separated
		Divorced
		Widowed
		Single

The Scottish 2001 census data was used to form the constraint tables and contains information about the counts of individuals in each sociodemographic category for every data zone in Scotland available from: https://www.scotlandscensus.gov.uk/census-

results/download-data/census-table-data/. See tables 3 and 4 for extracts of the constraint tables used in this model.

Table 3: *Extracted sample of the sex/age constraint table.*

DataZone	M_16_24	M_25_34	M_35_49	M_65_74	M_75+	…	F_75+
S01000001	23	109	108	21	13		32
S01000002	9	141	90	3	0		2
S01000003	15	104	143	4	5		4

Table 4: *Extracted sample of the economic status constraint table.*

DataZone	In paid employment	Self-employed	Student	Retired	Other_ economically_ inactive	Unemployed
S01000001	371	72	37	72	80	22
S01000002	423	25	14	15	36	6
S01000003	467	29	24	18	49	14

Both the microdata taken from the survey and constraint tables are used as inputs into the Flexible Modelling Framework. Upon completion of the microsimulation process a list of individual identifiers and the codes for data zones that each individual is located in is produced. This represents the synthetic population and the identifiers can be used to join the individual level attribute rich data available from the full survey with each individual in the synthetic population.

To ensure that the synthetic population produced is representative of the desired population model validation is required. Internal validation was assessed by examining several commonly used fit statistics. The goodness of fit statistics displayed in table 5 show a good fit between the simulated and observed data.

Table 5: *Validation metrics for the comparison of simulated and actual counts in each constraint*

Constraint	R^2	SRSME	TAE	SAE
Age/sex	1.0	0	0	0
Marital status	1.0	0	0	0
Economic status	0.969961	0.198851	257141	0.066611
Highest educational qualification	0.924808	0.215284	358867	0.096183

We also externally validated the microsimulation by comparing the simulated results to a different dataset, external to the model. The results are compared at the aggregate data zone level with estimates from the Scottish Index of Multiple Deprivation (SIMD) (248). SIMD is a relative measure of area-level deprivation, it ranks the 6,505 datazones in Scotland from most deprived (rank 1) to least deprived (rank 6,505). There is a well-established relationship between deprivation and personal or household income. Therefore, we expect the SIMD rank of each data zone will strongly correlate with these outcomes. As expected a

higher rank was strongly positively correlated with monthly income (r = 0.8313, p<0.0001) and annual income (r = 0.8185, p<0.0001), and strongly negatively correlated with annual benefits receipt (r = -0.7714, p<0.0001).

Highest educational qualification was used to represent the fundamental resource knowledge. To construct the other variables that represent the fundamental resources power, prestige and money we carried out exploratory factor analysis on a set of relevant variables available in the 2001 British Household Panel Survey (see Table 6).

Table 6: *Factor Loadings for Exploratory Factor Analysis used to create latent variables for fundamental resources. *Note factors were produced as 1-4 and we attached names which matched the fundamental resource they seemed to represent.*

	F1: Prestige	**F2: Money**	**F3: Power 1**	**F4: Power 2**
SocialClass_RG	0.82			
Occupation	0.94			
Manager_recent	0.4			
EquNetHHInc		0.96		
EquRealHHAnnInc		0.81		
PayHoliday			0.69	
BuyNewClothes			0.32	
CarPrivateUse			0.35	
FinancialSituation			0.41	
ProblemsOvercomingDifficulties				0.88
CapableOfDecisions				0.37

Df (24), X^2 = 71567.28, p <0.001.

The EFA model provided a four-factor solution. Factor 1 consisted of a measure of social class, occupation and whether the individual had managerial duties recently. We interpreted this factor to represent the fundamental resource prestige. In FCT prestige is associated with an individual's occupational status, jobs such as doctors or lawyers are seen to be more prestigious occupations. Factor 2 consisted of the variables equivalized net household income and equivalized real household annual income and therefore represents monetary resource. Factor 3 consisted of variables associated with being financially able to achieve your goals (e.g., whether you can afford to pay for holidays when you want to take them) and therefore we interpreted this factor to be a sub-component of the resource power. Factor 4 consisted of variables which represented whether you are psychologically capable to achieve your goals (e.g., do you feel capable of making your own decisions), and therefore we interpreted this factor to represent a facet of the resource power. Each latent variable was created by aggregating the component variables and then normalizing these aggregate variables to values between 0 and 1 for ease of use in the ABM. The power latent variable was created by aggregating both variables in Factor 3 and 4.

b. Deprivation Swap model

The general method and swap event parameters were taken from the Human population with swap model (264)available from: https://cran.r-project.org/web/packages/IBMPopSim/vignettes/IBMPopSim_human_pop_IMD.pdf . It should be noted that this model labels IMD quintiles as 1 = least deprived and 5 = most deprived, whereas the agent-based model labels IMD quintiles as 1 = most deprived and 5 = least deprived.

This model simulates the movement of individuals and therefore the change in SIMD quintile. However, as SIMD is a relative measure of deprivation these movements are required to be swap events, one individual must replace another in a different quintile. The absence of this swap mechanism would make it possible for all agents to move to one quintile or move out of one particular quintile. Based on the existing model young individuals aged 16-30 and in less deprived quintiles have the chance to move to more deprived areas, for example to continue education. While older adults ages 30-45, and in deprived areas can move to a less deprived area, for example to settle down and start a family.

In the model individuals first assess whether they can attempt to move deprivation quintile based on their age and their current deprivation quintile. For those aged 16-30 in deprivation quintile 5 the probability that that will attempt to swap is 0.001666, in deprivation quintile 4 and 3 the probability is 0.000833. While those aged 30-45 and in deprivation quintile 2 have the probability to attempt to swap of 0.0183 and those in quintile 1 have the probability of 0.0208. These values where obtained from the swap intensity parameters listed in the existing model. If the random-float value is less than the probability, individuals indicate that want to attempt to move deprivation quintile.

If individuals indicate that they will attempt to move they will then attempt to switch with another individual in a different quintile who has also indicated that they are going to attempt to switch. For each individual that has indicated they wish to move up deprivation quintile they create a link to all other individuals that have indicated they wish to move down, and vice versa. The swap will either be successful or unsuccessful based on the probabilities displayed in Table 2.

***Table 7**: Probabilities of a successful swap to another deprivation quintile.*

	IMD1	**IMD2**	**IMD3**	**IMD4**	**IMD5**
Move Down: 16-30 years	0.3	0.3	0.4	0.0	0.0
Move Up: 30-45	0.0	0.0	0.0	0.4	0.6

For example, if someone that has indicated that they want to move down IMD quintile is attempting to swap with an individual in quintile 3 who has indicated they wish to move up IMD quintile, the probability of a successful swap is 0.4. If the swap is unsuccessful the individual will continue to search for other potential swaps until it has exhausted the pool of potential swaps. Individuals can only attempt to swap with the same individual once, as once the swap is unsuccessful the link is broken between those individuals. If the swap is a success then the individuals switch co-ordinates, move to their new location and update their IMD quintile.

c. Alcohol-specific mortality model

To calculate the absolute risk of death from alcohol consumption we adapted the method described by Meier and colleagues (265). We assumed that the starting threshold for absolute risk was 14 units per week for both males and females in line with the current drinking guidelines (266). We also capped consumption for the purposes of estimating harm at 131.25 units per week for two reasons: 1) There are relatively few drinkers exceeding 131.25 units per week in cohort studies that are used to generate dose response curves, and those that are likely to die of another cause, and 2) The specific approach used to fit

curves (fractional polynomials) is sensitive, and as a result a small change in input data can lead to a big change in the shape of the curve at high values of consumption.

We used the following absolute risk function based on consumption (for which a slope is defined) and threshold:

AR(c) = 0 if c < T

AR(c) = β (c – T) otherwise

Where AR = absolute risk, c = alcohol consumption, T = threshold and β = slope parameter.

Given sex differences in the risk of alcohol-specific death, we defined two separate beta slope parameters; one for males and one for females.

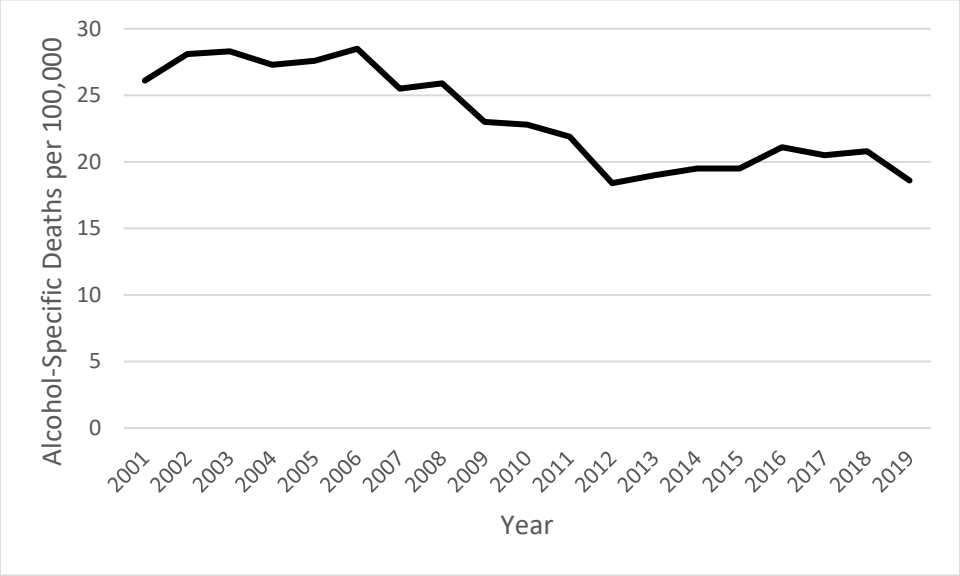
This calculated absolute risk was then adjusted using the ratio of successful versus unsuccessful adaptations as outlined in section 4.

Appendix D: Calibrating the Absolute Risk Function to population level alcohol specific deaths.

D.1 Targets, Calibration Procedure and Results.

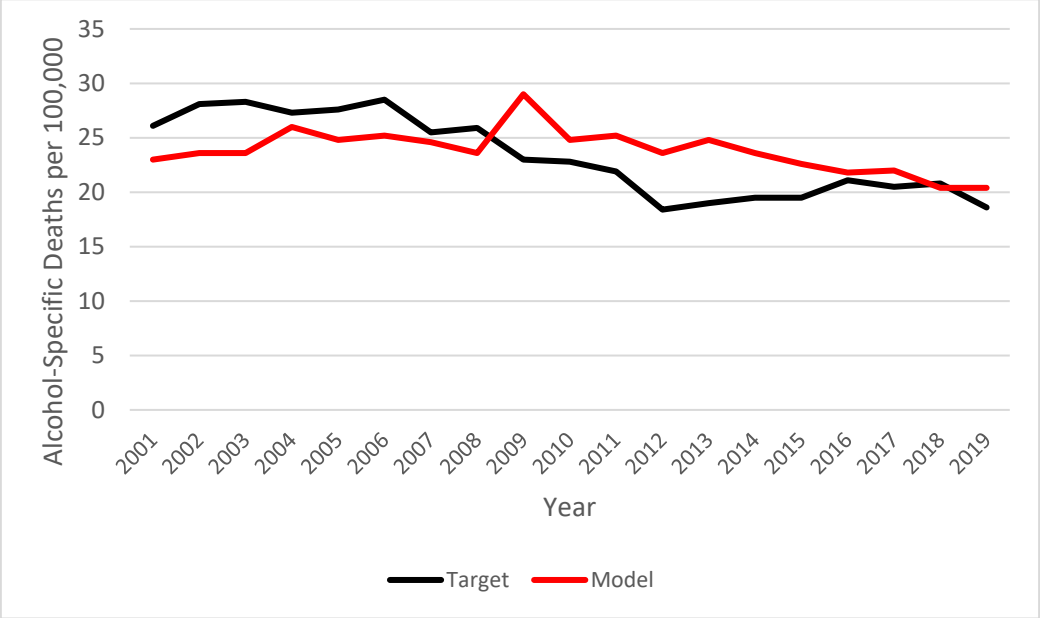
To calculate absolute risk as described in section 7c of the ODD protocol a beta slope parameter needs to be estimated for each sex. To estimate values for each beta slope we calibrated a model which operationalized the absolute risk function described by Meier and colleagues to population level alcohol specific deaths in Scotland from 2001-2019 (see Figure C.1).

Figure C.1: *Alcohol specific death per 100,000 in Scotland from 2001-2019.*



To calibrate the model, we used the same approach applied in chapter 9 to calibrate the ABM. We used the nlrx package and a Latin hypercube sampling design, however given that the absolute risk model was much simpler and we were only calibrating two parameters: BetaSlopeMale and BetaSlopeFemale a larger sample of 15,000 parameter settings were simulated. The prior distributions for both the BetaSlopeMale and BetaSlopeFemale parameters were uniform distributions with a minimum value of 0.001 and a maximum value of 0.01. The best identified model setting had an implausibility value of 0.90. The model outputs compared to the target data are shown in Figure C.2.

Figure C.2: *The best calibrated model compared to the observed rate of alcohol specific deaths for the whole Scottish population per 100,000 from 2001-2019.*



The best estimated model slightly underestimates alcohol specific deaths from 2001-2008 and slightly overestimates deaths from 2009-2017. This is either due to a spike in alcohol consumption from 2008, particularly in the most deprived quintile (as shown in Chapter 8, Figure 8.2) or that as we use two separate data sources to estimate alcohol consumption, the GHS for years 2001-2006 and SHeS for years 2008-2019. Specifically, the population recruited in the GHS is UK wide whereas the population recruited in the SHeS is Scottish only which means estimations of alcohol consumption across time in the microsimulation may not be consistent. For males the beta slope value identified that best fit the data was 0.00398 and for females it was 0.00101. These values were fixed in the FCT ABM to ensure that absolute risk was calculated accurately before it was adjusted using the behavioural outcomes from the FCT mechanisms.

D.2 The NetLogo Model Code for calculating the absolute risk of alcohol specific death from alcohol consumption.

```
extensions [nw csv profiler array]

;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;
;;;;;;;;;;;;;;;; GLOBAL VARIABLES SETUP ;;;;;;;;;;;;;;;;;;;
;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;

globals [
  next-replenish
  year
  thresholdConsumption
  thresholdLog
  females-dead-alcohol
  males-dead-alcohol
  number-dead-other

  Femaledeaths2001
  Femaledeaths2002
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  Maledeaths2017
```

```
  Maledeaths2018
  Maledeaths2019

  countMales
  countFemales

  inflationFemale
  inflationMale
]

;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;
;;;;;;;;;;;;Breeds definitions ;;;;;;;;;;

breed [individuals individual]

individuals-own[
  gender
  age
  microsimID
  UPW
  UPW2002
  UPW2005
  UPW2008
  UPW2011
  UPW2014
  UPW2017
  UPWlog
  absoluteRisk
]

;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;
;;;;;;;;;;;;;;;; SETUP PROCEDURE ;;;;;;;;;;;;;;;;;;;;;;;;;
;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;

to setup
  ;;clear all
  clear-all
  reset-ticks

  file-close-all
  file-open "C:/Fundamental Cause Theory ABM/PopulationReadInCSVExtension.csv"
  let headings csv:from-row file-read-line

  while [ not file-at-end?] [
    let data csv:from-row file-read-line
    print data
    create-individuals 1 [
      set microsimID item 0 data
      set age item 1 data
      set gender item 3 data
      set UPW item 12 data
      set UPW2002 item 13 data
      set UPW2005 item 14 data
      set UPW2008 item 15 data
      set UPW2011 item 16 data
```

```
    set UPW2014 item 17 data
    set UPW2017 item 18 data
    setxy random-xcor random-ycor
    set size 1
    set shape "person"
   ]
  ]
  file-close-all

  set next-replenish 52
  set year 2001
  set thresholdConsumption 14
  set thresholdLog log thresholdConsumption 10
  set countMales (count individuals with [gender = "Male"])
  set countFemales (count individuals with [gender = "Female"])
end

;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;
;;;;;;;;;;;;;;;; GO PROCEDURE ;;;;;;;;;;;;;;;;;;;;;;;;;;
;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;

to go
  if ticks = next-replenish [
  ask individuals [
    if age >= 16 [calculateMortality]
    set age age + 1
    if age >= 16 [updateDrinking]
   ]
   reportDeaths
   set year year + 1
   set next-replenish ticks + 52
  ]
  set countMales (count individuals with [gender = "Male"])
  set countFemales (count individuals with [gender = "Female"])
  tick

  if ticks = 990 [
   stop
  ]
end



;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;
;;;;;;;;;;;;;;;; ABSOLUTE RISK DEATHS;;;;;;;;;;;;;;;;;;;;
;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;


to calculateMortality
  calculateRisk
  alcoholMortalityCheck
  allCauseMortalityCheck
end

to calculateRisk
```

```
  if UPW < 0.5 [set UPW 0]
  if UPW >= 0.5 and UPW <= 1 [set UPW 1]
  if UPW > 131.25 [set UPW 131.25]
  if UPW > 0 [set UPWlog log UPW 10]

  if gender = "Female" [
    set inflationFemale 100000 / countFemales
  ifelse UPW > thresholdConsumption
    [set absoluteRisk (inflationFemale * (betaSlopeFemale * (UPWlog - thresholdLog)))]
    [set absoluteRisk 0]
  ]

  if gender = "Male" [
    set inflationMale 100000 / countMales
    ifelse UPW > thresholdConsumption
    [set absoluteRisk (inflationMale * (betaSlopeMale * (UPWlog - thresholdLog)))]
    [set absoluteRisk 0]
  ]

end

to alcoholMortalityCheck
  if gender = "Female" [
  if random-float 1 < absoluteRisk [
    set females-dead-alcohol females-dead-alcohol + 1
  ]
  ]
  if gender = "Female" [
   let actualRisk (absoluteRisk / inflationFemale)
      if random-float 1 < actualRisk [
        die
  ]
  ]

   if gender = "Male" [
  if random-float 1 < absoluteRisk [
    set males-dead-alcohol males-dead-alcohol + 1
  ]
  ]

  if gender = "Male" [
   let actualRisk (absoluteRisk / inflationMale)
      if random-float 1 < actualRisk [
        die
  ]
  ]

end


to allCauseMortalityCheck
  if (age >= 16 and age <= 25) and gender = "Male" [
    if random-float 1 < 0.000895 [
      set number-dead-other number-dead-other + 1
      die
```

```
  ]
]
if (age >= 26 and age <= 35) and gender = "Male" [
  if random-float 1 < 0.001456 [
    set number-dead-other number-dead-other + 1
    die
  ]
]
 if (age >= 36 and age <= 45) and gender = "Male" [
  if random-float 1 < 0.002677 [
    set number-dead-other number-dead-other + 1
    die
  ]
]
if (age >= 46 and age <= 55) and gender = "Male" [
  if random-float 1 < 0.005320 [
    set number-dead-other number-dead-other + 1
    die
  ]
]
if (age >= 56 and age <= 65) and gender = "Male" [
  if random-float 1 < 0.012868 [
    set number-dead-other number-dead-other + 1
    die
  ]
]
if (age >= 66 and age <= 75) and gender = "Male" [
  if random-float 1 < 0.031500 [
    set number-dead-other number-dead-other + 1
    die
  ]
]
if (age > 75) and gender = "Male" [
  if random-float 1 < 0.082420 [
    set number-dead-other number-dead-other + 1
    die
  ]
]

if (age >= 16 and age <= 25) and gender = "Female" [
  if random-float 1 < 0.000338 [
    set number-dead-other number-dead-other + 1
    die
  ]
]
if (age >= 26 and age <= 35) and gender = "Female" [
  if random-float 1 < 0.000567 [
    set number-dead-other number-dead-other + 1
    die
  ]
]
 if (age >= 36 and age <= 45) and gender = "Female" [
  if random-float 1 < 0.001447 [
    set number-dead-other number-dead-other + 1
    die
```

```
    ]
  ]
  if (age >= 46 and age <= 55) and gender = "Female" [
    if random-float 1 < 0.003260 [
      set number-dead-other number-dead-other + 1
      die
    ]
  ]
  if (age >= 56 and age <= 65) and gender = "Female" [
    if random-float 1 < 0.008018 [
      set number-dead-other number-dead-other + 1
      die
    ]
  ]
  if (age >= 66 and age <= 75) and gender = "Female" [
    if random-float 1 < 0.020539 [
      set number-dead-other number-dead-other + 1
      die
    ]
  ]
  if (age > 75) and gender = "Female" [
    if random-float 1 < 0.059150 [
      set number-dead-other number-dead-other + 1
      die
    ]
  ]
end



;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;
;;;;;;;;;;;;;;;; UPDATE DRINKING ;;;;;;;;;;;;;;;;;;;;;;
;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;


to updateDrinking
  if year = 2002 [set UPW UPW2002]
  if year = 2005 [set UPW UPW2005]
  if year = 2008 [set UPW UPW2008]
  if year = 2011 [set UPW UPW2011]
  if year = 2014 [set UPW UPW2014]
  if year = 2017 [set UPW UPW2017]
end


;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;
;;;;;;;;;;;;;;;; REPORTERS ;;;;;;;;;;;;;;;;;;;;;;;;;;;;;
;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;

to reportDeaths
  if year = 2001 [
    set Femaledeaths2001 females-dead-alcohol
    set females-dead-alcohol 0
    set Maledeaths2001 males-dead-alcohol
```

```
    set males-dead-alcohol 0
  ]

if year = 2002 [
    set Femaledeaths2002 females-dead-alcohol
    set females-dead-alcohol 0
    set Maledeaths2002 males-dead-alcohol
    set males-dead-alcohol 0
  ]

if year = 2003 [
    set Femaledeaths2003 females-dead-alcohol
    set females-dead-alcohol 0
    set Maledeaths2003 males-dead-alcohol
    set males-dead-alcohol 0
  ]

…repeated for each year until 2019…

if year = 2019 [
    set Femaledeaths2019 females-dead-alcohol
    set females-dead-alcohol 0
    set Maledeaths2019 males-dead-alcohol
    set males-dead-alcohol 0
  ]

end
```

Appendix E: FCT ABM NetLogo Code.

```
extensions [nw csv profiler array]

;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;
;;;;;;;;;;;;;;;; GLOBAL VARIABLES SETUP ;;;;;;;;;;;
;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;


globals [
  seed-positions
  number-of-subareas
  list-subareas
  next-replenish
  year
  thresholdConsumption
  number-dead-alcohol
  number-dead-other
  start-population
  end-population
  thresholdLog
  IMD1-deaths
  IMD2-deaths
  IMD3-deaths
  IMD4-deaths
  IMD5-deaths

  IMD1-resources
  IMD2-resources
  IMD3-resources
  IMD4-resources
  IMD5-resources

  IMD1deaths2001
  IMD2deaths2001
  IMD3deaths2001
  IMD4deaths2001
  IMD5deaths2001

  IMD1deaths2002
  IMD2deaths2002
  IMD3deaths2002
  IMD4deaths2002
  IMD5deaths2002

  IMD1deaths2003
  IMD2deaths2003
  IMD3deaths2003
  IMD4deaths2003
  IMD5deaths2003

  IMD1deaths2004
  IMD2deaths2004
  IMD3deaths2004
  IMD4deaths2004
  IMD5deaths2004
```

IMD1deaths2005
IMD2deaths2005
IMD3deaths2005
IMD4deaths2005
IMD5deaths2005

IMD1deaths2006
IMD2deaths2006
IMD3deaths2006
IMD4deaths2006
IMD5deaths2006

IMD1deaths2007
IMD2deaths2007
IMD3deaths2007
IMD4deaths2007
IMD5deaths2007

IMD1deaths2008
IMD2deaths2008
IMD3deaths2008
IMD4deaths2008
IMD5deaths2008

IMD1deaths2009
IMD2deaths2009
IMD3deaths2009
IMD4deaths2009
IMD5deaths2009

IMD1deaths2010
IMD2deaths2010
IMD3deaths2010
IMD4deaths2010
IMD5deaths2010

IMD1deaths2011
IMD2deaths2011
IMD3deaths2011
IMD4deaths2011
IMD5deaths2011

IMD1deaths2012
IMD2deaths2012
IMD3deaths2012
IMD4deaths2012
IMD5deaths2012

IMD1deaths2013
IMD2deaths2013
IMD3deaths2013
IMD4deaths2013
IMD5deaths2013

IMD1deaths2014

```
  IMD2deaths2014
  IMD3deaths2014
  IMD4deaths2014
  IMD5deaths2014

  IMD1deaths2015
  IMD2deaths2015
  IMD3deaths2015
  IMD4deaths2015
  IMD5deaths2015

  IMD1deaths2016
  IMD2deaths2016
  IMD3deaths2016
  IMD4deaths2016
  IMD5deaths2016

  IMD1deaths2017
  IMD2deaths2017
  IMD3deaths2017
  IMD4deaths2017
  IMD5deaths2017

  IMD1deaths2018
  IMD2deaths2018
  IMD3deaths2018
  IMD4deaths2018
  IMD5deaths2018

  IMD1deaths2019
  IMD2deaths2019
  IMD3deaths2019
  IMD4deaths2019
  IMD5deaths2019

  eventsN

  betaSlopeMale
  betaSlopeFemale

]

;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;
;;;;;;;;;;;Breeds definitions ;;;;;;;;;
;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;

breed [individuals individual]
breed [subareas subarea]
breed [communicators communicator]

directed-link-breed [Friendships Friendship]
directed-link-breed [deprivationSwaps deprivationSwap]
```

```
;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;
;;;;;;;;;;;; AGENT SETUP;;;;;;;;;;;;;;;;;;;;;
;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;

individuals-own [
  gender
  age
  ageGroup
  educationGroup
  microsimID
  originalQuintile
  deprivationQuintile
  zone
  GPD
  UPW
  UPW2002
  UPW2005
  UPW2008
  UPW2011
  UPW2014
  UPW2017
  drinkingStatus
  targetConnections
  numberConnections


  power
  prestige
  money
  knowledge

  eventList
  resolvedEvents
  unresolvedEvents
  activeEvent
  totalEvents
  strategyEvent
  tipsEvent
  strategise

  totalResources
  successfullyAdapted
  unsuccessfullyAdapted

  moveUpDeprivationQuintile
  moveDownDeprivationQuintile
  new-xcor
  new-ycor

  UPWlog
  absoluteRisk
  dead
  ratio
  adjustedRisk1
```

```
  adjustedRisk2
]

subareas-own [
  deprivationQuintile
  number
]

patches-own [
  subareaNumber
]

friendships-own [
  new-link?
  prestigeBonus
]

deprivationSwaps-own [
  swap
]

communicators-own [
  currentEvent
  eventHistory
  totalEvents
]

;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;
;;;;;;;;;;;;;;;; SETUP PROCEDURE ;;;;;;;;;;;;;;;;;
;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;

to setup
  ;;clear all
  clear-all
  set-seeds
  setup-subareas
  reset-ticks

  file-close-all
  file-open "C:/Fundamental Cause Theory ABM/PopulationReadInCSVExtension.csv"
  let headings csv:from-row file-read-line

  while [ not file-at-end?] [
    let data csv:from-row file-read-line
    print data
    create-individuals 1 [
      set microsimID item 0 data
      set age item 1 data
      set ageGroup item 2 data
      set gender item 3 data
      set originalQuintile item 4 data
      set deprivationQuintile item 5 data
      set knowledge item 6 data
      set money item 7 data
      set power item 8 data
```

```
    set prestige item 9 data
    set drinkingStatus item 10 data
    set educationGroup item 11 data
    set UPW item 12 data
    set UPW2002 item 13 data
    set UPW2005 item 14 data
    set UPW2008 item 15 data
    set UPW2011 item 16 data
    set UPW2014 item 17 data
    set UPW2017 item 18 data
    setxy random-xcor random-ycor
    set size 1
    set shape "person"
  ]
 ]
 file-close-all

 ask individuals [
  ;;sets up grid matched by deprivation quintile
  move-to one-of patches with [subareaNumber = [deprivationQuintile] of myself]
  set targetConnections random 7
 ]
 setup-network
 setup-socialConnections
 ask individuals [
  ask my-links [hide-link]
  set totalResources (power + prestige + money + knowledge)
  set eventList []
  set resolvedEvents []
  set unresolvedEvents []
  set tipsEvent []
 ]


create-communicators 1
 ask communicators [
  set eventHistory []
 ]
 set next-replenish 52
 set year 2001
 set thresholdConsumption 14
 set thresholdLog log thresholdConsumption 10
 set betaSlopeFemale 0.00101
 set betaSlopeMale 0.00398


end


;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;
;;;;;;;;;;;;;;;;;; SUBAREA SETUP ;;;;;;;;;;;;;;;;;;;;;
;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;

to set-seeds
 ca
```

```
  set seed-positions (list (list 15 15) (list 15 45) (list 30 30) (list 45 15) (list 45 45))
  foreach seed-positions [ pos ->
    ask patch (first pos) (last pos) [sprout-subareas 1 [set label who + 1]]]
  ask subareas [subarea-init-components]
  ask subareas [set number label]
end

to subarea-init-components
  let num who + 1
  ask patch-here [set subareaNumber num]
end

to setup-subareas
  if (not any? patches with [subareaNumber = 0]) [stop]
  let i 1
  while [(i < 1000) and (any? patches with [subareaNumber = 0])] [
    set i i + 1
    ask patches [
      if (subareaNumber > 0) [
        ask neighbors4 with [subareaNumber = 0]
        [ set subareaNumber [subareaNumber] of myself
          patch-updateview
        ]
      ]
    ]
  ]
end

;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;
;;;;;;;;;;;;;;;; SOCIAL NETWORK SETUP ;;;;;;;;;;;;
;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;

to setup-network
  ask individuals with [age >= 16] [
    let i 0
    while [(i <= 10000) and (numberConnections < targetConnections)] [
      create-Friendships-to n-of 1 other individuals with [age >= 16] [set new-link? true]
      if random-float 1 < proportionSpatialConnections [assess-spatial-match]
      if random-float 1 < proportionGenderConnections [assess-gender-match]
      if random-float 1 < proportionEducationConnections [assess-education-match]
      if random-float 1 < proportionAgeConnections [assess-age-match]
      if random-float 1 < proportionDrinkingConnections [assess-drinking-match]
      assess-prestige
      set numberConnections count my-friendships
      ask Friendships [set new-link? false]
      set i i + 1
    ]
  ]
end

to assess-spatial-match
  ask Friendships [
    if new-link? [
    ifelse ([deprivationQuintile] of end1) = ([deprivationQuintile] of end2)
      [set color blue]
```

```
      [die]
    ]
  ]
end

to assess-gender-match
  ask Friendships [
    if new-link? [
    ifelse ([gender] of end1) = ([gender] of end2)
      [set color blue]
      [die]
  ]
  ]
end

to assess-education-match
  ask Friendships [
    if new-link? [
    ifelse ([educationGroup] of end1) = ([educationGroup] of end2)
      [set color blue]
      [die]
  ]
  ]
end

to assess-age-match
  ask Friendships [
    if new-link? [
      ifelse ([ageGroup] of end1) = ([ageGroup] of end2)
      [set color blue]
      [die]
    ]
  ]
end

to assess-drinking-match
  ask Friendships [
    if new-link? [
      ifelse ([drinkingStatus] of end1) = ([drinkingStatus] of end2)
      [set color blue]
      [die]
    ]
  ]
end



;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;
;;;;;;;;;;;;;;;; GO PROCEDURE ;;;;;;;;;;;;;;;;;;;;
;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;

;model scheduler
to go
  ;; each tick is 1-week, we have 14 years of data for calibration - 52 x 14 = 728
  ;; resources are annual so will replenish after 1 year
```

```
  doSituation
  ask individuals [
    if age >= 16 [
    doActions
    ]
  ]
  if (ticks = next-replenish) [
    set start-population count individuals with [age >= 16]
    ask individuals [
      calculateMortality
    ]
    set end-population count individuals with [age >= 16]

    doTransformation

    ask individuals [
      set age age + 1
      if age = 15 [
        create-Friendships-to n-of 3 other individuals with [age >= 15]
      ]
      if age >= 16 [
      ;;replenish annual material resources
      set totalResources (totalResources + money)
      updateEducation
      updateDrinking
    ]
   ]
    reportDeaths
    set year year + 1
    set next-replenish ticks + 52
  ]
  tick

  if (ticks = 780) [
    stop
  ]
end



;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;
;;;;;;;;;;;;; ANNUAL TRANSITIONS ;;;;;;;;;;;;;;;;;;
;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;

to updateEducation
  if (age <= 30) and (educationGroup != "Group 4") [
    if educationGroup = "No qualifications" [
      ;; the proportion of individuals in Group 1 over 30 is 24% but the age range is from 30-90
so divided 0.24/14 (16-30) so that not 24% aren't moving up every year
      if random-float 1 < 0.017 [
        set educationGroup "Group 1"
        ;; the average knowledge of someone in group 1 is 0.25
        if knowledge < 0.25 [
          set knowledge 0.25
        ]
```

```
      ]
    ]
    if educationGroup = "Group 1" or educationGroup = "No qualifications" [
      ;; the proportion of individuals in Group 2 over 30 is 10% but the age range is from 30-90
so divided 0.10/14 (16-30)
      if random-float 1 < 0.007 [
        set educationGroup "Group 2"
        ;; the average knowledge of someone in group 2 is 0.43
        if knowledge < 0.43 [
          set knowledge 0.43
        ]
      ]
    ]
    if educationGroup = "Group 1" or educationGroup = "Group 2" [
      ;; the proportion of individuals in Group 3 over 30 is 5% but the age range is from 30-90
so divided 0.05/14 (16-30)
      if random-float 1 < 0.003 [
        set educationGroup "Group 3"
        ;; the average knowledge of someone in group 3 is 0.57
        if knowledge < 0.57 [
          set knowledge 0.57
        ]
      ]
    ]
    if  educationGroup = "Group 2" or educationGroup = "Group 3" [
      ;; the proportion of individuals in Group 4 over 30 is 39% but the age range is from 30-90
so divided 0.39/14 (16-30)
      if random-float 1 < 0.028 [
        set educationGroup "Group 4"
        ;; the average knowledge of someone in group 4 is 0.94
        if knowledge < 0.94 [
          set knowledge 0.94
        ]
      ]
    ]
  ]
end

to updateDrinking
  if year = 2002 [set UPW UPW2002]
  if year = 2005 [set UPW UPW2005]
  if year = 2008 [set UPW UPW2008]
  if year = 2011 [set UPW UPW2011]
  if year = 2014 [set UPW UPW2014]
  if year = 2017 [set UPW UPW2017]
end



;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;
;;;;;;;;;;;;; SITUATIONAL MECHANISMS ;;;;;;;;;;;;;
;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;
```

```
;; Situational Mechanisms
to doSituation
  ;; agents assign their totalResources to communityResource variable in subarea
  update-community-resources
  ;; communicator entity assess whether a new event has occured in the current tick
  assess-events
  ;; communicator entity communicates new event to proportion of agents
  communicate-event
end

to update-community-resources
  set IMD1-resources sum [totalResources] of individuals with [deprivationQuintile = 1]
  set IMD2-resources sum [totalResources] of individuals with [deprivationQuintile = 2]
  set IMD3-resources sum [totalResources] of individuals with [deprivationQuintile = 3]
  set IMD4-resources sum [totalResources] of individuals with [deprivationQuintile = 4]
  set IMD5-resources sum [totalResources] of individuals with [deprivationQuintile = 5]
end

to assess-events
  ask Communicators [
  ifelse (random-float 1 < probabilityEvent) [set currentEvent 1] [set currentEvent 0]
    set totalEvents totalEvents + currentEvent
    set eventsN eventsN + 1
    if (currentEvent = 1) [
      set eventHistory lput totalEvents eventHistory
    ]
  ]
end

to communicate-event
  if ([currentEvent] of one-of communicators = 1) [
    ask n-of (count individuals with [age >= 16] * %agentsKnowEvent) individuals with [age >=
16] [
      ;; only individuals with knowledge greater than random float 1 then they correctly
perceive the event
      if knowledge >= random-float 1 [
      set eventList lput [totalEvents] of one-of communicators eventList
      set totalEvents totalEvents + 1
      ]
  ]
 ]
end


;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;
;;;;;;;;;;;; ACTION MECHANISMS ;;;;;;;;;;;;;;;;
;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;


to doActions
  ;; pick an event this tick to try to resolve
  select-event
  ;;agents communicate with another agent in their social network to inform them of an event
they know about
  communicate-event-with-network
```

```
  ;; agents assess whether they have been given a strategy from a social connection to adapt
to the event - if they have there resources are changed with a strategy multiplier
  check-strategise
  ;; agents deploy their available resources in an attempt to adapt to an event in their list that
they have not yet adapted to
  deploy-resources
  ;; agents that have successfully adapted to an event communicate their strategy to another
agent in their social network
  communicate-strategy-with-network
end

to select-event
  if (not empty? eventList) [
  set unresolvedEvents []
  ;; use filter to select all of the values that appear in the eventList which do not appear in the
resolvedEvents list
  set unresolvedEvents filter [x -> not member? x resolvedEvents] eventList
  ;; agents select a random unresolved event from the list
    if (not empty? unresolvedEvents) [
  set activeEvent one-of unresolvedEvents
    ]
  ]
end

to communicate-event-with-network
  ;; probability that agents who know about event will communicate that with another agent in
their network
  if (any? friendship-neighbors) [
  if (random-float 1 < probabilityCommunicate) and (activeEvent > 0)  [
      ask one-of friendship-neighbors [ set eventList lput [activeEvent] of myself eventList ]
    ]
 ]
end

to check-strategise
 set strategise 0
 if (activeEvent > 0) and (not empty? tipsEvent) [
    let tip one-of tipsEvent
    if tip = activeEvent [
     set totalResources (totalResources * strategyMultiplier)
     set strategise 1
    ]
  ]
end

to deploy-resources
 if (activeEvent > 0) [
  ifelse (totalResources > adaptationThreshold) [
    set resolvedEvents lput activeEvent resolvedEvents
    set successfullyAdapted successfullyAdapted + 1
    set activeEvent 0
    let knowledgeUpdate (knowledge + knowledgeGain)
      if (knowledgeUpdate > 1) [set knowledgeUpdate 1]
      if (strategise = 1) [set totalResources (totalResources / strategyMultiplier)
    set totalResources (totalResources - resourceDepletion)
```

```
    set totalResources (totalResources - knowledge)
    set totalResources (totalResources + knowledgeUpdate)
      if totalResources < 0 [set totalResources 0]

  ]
 ]
    [set unsuccessfullyAdapted unsuccessfullyAdapted + 1
     if (strategise = 1) [set totalResources (totalResources / strategyMultiplier)]
  ]
 ]
end

to communicate-strategy-with-network
  if (any? friendship-neighbors) [
  if (not empty? resolvedEvents) [
  set strategyEvent one-of resolvedEvents
  if (random-float 1 < probabilityCommunicate) [
      ask one-of friendship-neighbors [ set tipsEvent lput [strategyEvent] of myself tipsEvent]
    ]
  set tipsEvent filter [x -> not member? x resolvedEvents] tipsEvent
  ]
 ]
end



;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;
;;;;;;;;; TRANSFORMATIONAL MECHANISMS ;;;;;;;;;;;;
;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;


;;Transformational Mechanisms
to doTransformation
  updateIMDQuintile
end

to updateIMDQuintile
  ask individuals with [age >= 16] [
    ask my-DeprivationSwaps [ die ]
    assess-swap
    attempt-switch
    move-quintile
    update-Quintile
    check
  ]
end

to assess-swap
  ;; use probabilities of swap for each quintile from the existing model https://cran.r-project.org/web/packages/IBMPopSim/vignettes/IBMPopSim_human_pop_IMD.pdf
    if (age <= 29) and (deprivationQuintile = 5) [
      if random-float 1 < 0.001666 [
        set moveDownDeprivationQuintile 1
      ]
    ]
```

```
  if (age <= 29) and (deprivationQuintile = 4) [
    if random-float 1 < 0.000833 [
      set moveDownDeprivationQuintile 1
    ]
  ]
    if (age <= 29) and (deprivationQuintile = 3) [
    if random-float 1 < 0.000833 [
      set moveDownDeprivationQuintile 1
    ]
  ]
  if (age >= 30) and (age <= 45) and (deprivationQuintile = 2) [
    if random-float 1 < 0.0183 [
      set moveUpDeprivationQuintile 1
    ]
  ]
  if (age >= 30) and (age <= 45) and (deprivationQuintile = 1) [
    if random-float 1 < 0.0208 [
      set moveUpDeprivationQuintile 1
    ]
  ]
end

to attempt-switch
  ;; agents need to swap with another person do so by checking if anyone else in another
deprivation quintile has the opposite variable activated
  ;; agents create links with all other individuals that have the opposite swap variable
activated
  ;; based on some probabilities to do with the quintile they would move to the links are either
set to swap or deactivated by the command die (probabilities taken from the existing model
linked above)

  if (moveDownDeprivationQuintile = 1) [
  let potentialSwaps individuals with [moveUpDeprivationQuintile = 1]
  if any? potentialSwaps [create-DeprivationSwaps-to other potentialSwaps]
    ask DeprivationSwaps [
      if ([deprivationQuintile] of end2) = 3 [
        ifelse random-float 1 < 0.4
        [set swap 1]
        [die]
    ]
      if ([deprivationQuintile] of end2) = 2 [
        ifelse random-float 1 < 0.3
        [set swap 1]
        [die]
      ]
      if ([deprivationQuintile] of end2) = 1 [
        ifelse random-float 1 < 0.3
        [set swap 1]
        [die]
      ]
    ]
  ]

  if (moveUpDeprivationQuintile = 1) [
    let potentialSwaps individuals with [moveDownDeprivationQuintile = 1]
```

```
    if any? potentialSwaps [create-DeprivationSwaps-to other potentialSwaps]
    ask DeprivationSwaps [
       if ([deprivationQuintile] of end2) = 4 [
         ifelse random-float 1 < 0.4
         [set swap 1]
         [die]
       ]
       if ([deprivationQuintile] of end2) = 5 [
         ifelse random-float 1 < 0.6
         [set swap 1]
         [die]
       ]
    ]
  ]
end

to move-quintile
  if any? DeprivationSwap-neighbors [
  let swap-neighbor one-of DeprivationSwap-neighbors
  set new-xcor ([xcor] of swap-neighbor)
  set new-ycor ([ycor] of swap-neighbor)
  ask swap-neighbor [
    set xcor ([xcor] of myself)
    set ycor ([ycor] of myself)
  ]
  setxy new-xcor new-ycor
  set moveDownDeprivationQuintile 0
  set moveUpDeprivationQuintile 0
  ask swap-neighbor [
    set moveDownDeprivationQuintile 0
    set moveUpDeprivationQuintile 0
  ]
    ask DeprivationSwaps [die]
  ]
end

to update-Quintile
  set deprivationQuintile [subareaNumber] of patch-here
end

to check
  if originalQuintile != deprivationQuintile [
    set size 5
    set shape "triangle"
    set color 0
  ]
end

to calculateMortality
  calculateRisk
  alcoholMortalityCheck
  allCauseMortalityCheck
end

to calculateRisk
```

```
  if UPW < 0.5 [set UPW 0]
  if UPW >= 0.5 and UPW <= 1 [set UPW 1]
  if UPW > 131.25 [set UPW 131.25]
  if UPW > 0 [set UPWlog log UPW 10]

  if gender = "Female" [
  ifelse UPW > thresholdConsumption
    [set absoluteRisk (100 * (betaSlopeFemale * (UPWlog - thresholdLog)))]
    [set absoluteRisk 0]
  ]

  if gender = "Male" [
    ifelse UPW > thresholdConsumption
    [set absoluteRisk (100 * (betaSlopeMale * (UPWlog - thresholdLog)))]
    [set absoluteRisk 0]
  ]
end

to alcoholMortalityCheck
  if unsuccessfullyAdapted > 0 or successfullyAdapted > 0 [
  let sumEvents unsuccessfullyAdapted + successfullyAdapted
  let rMod 1 + (unsuccessfullyAdapted / sumEvents) - 0.5
  let rModBeta Rmod ^ betaModifier

  set adjustedRisk1 (absoluteRisk * rModBeta)

  if random-float 1 < adjustedRisk1 [
      set number-dead-alcohol number-dead-alcohol + 1
      if deprivationQuintile = 1 [set IMD1-deaths IMD1-deaths + 1]
      if deprivationQuintile = 2 [set IMD2-deaths IMD1-deaths + 1]
      if deprivationQuintile = 3 [set IMD3-deaths IMD1-deaths + 1]
      if deprivationQuintile = 4 [set IMD4-deaths IMD1-deaths + 1]
      if deprivationQuintile = 5 [set IMD5-deaths IMD1-deaths + 1]

      let actualRisk (adjustedRisk1 / 100)
      if random-float 1 < actualRisk [
        die
    ]
  ]
  ]
    if unsuccessfullyAdapted = 0 and successfullyAdapted = 0 [
    set adjustedRisk1 absoluteRisk

    if random-float 1 < adjustedRisk1 [
      set number-dead-alcohol number-dead-alcohol + 1
      if deprivationQuintile = 1 [set IMD1-deaths IMD1-deaths + 1]
      if deprivationQuintile = 2 [set IMD2-deaths IMD1-deaths + 1]
      if deprivationQuintile = 3 [set IMD3-deaths IMD1-deaths + 1]
      if deprivationQuintile = 4 [set IMD4-deaths IMD1-deaths + 1]
      if deprivationQuintile = 5 [set IMD5-deaths IMD1-deaths + 1]

      let actualRisk (adjustedRisk1 / 100)
      if actualRisk > random-float 1 [
        die
      ]
```

```
  ]
 ]
end

to allCauseMortalityCheck
 if (age >= 16 and age <= 25) and gender = "Male" [
  if random-float 1 < 0.000895 [
   set number-dead-other number-dead-other + 1
   die
  ]
 ]
 if (age >= 26 and age <= 35) and gender = "Male" [
  if random-float 1 < 0.001456 [
   set number-dead-other number-dead-other + 1
   die
  ]
 ]
  if (age >= 36 and age <= 45) and gender = "Male" [
  if random-float 1 < 0.002677 [
   set number-dead-other number-dead-other + 1
   die
  ]
 ]
 if (age >= 46 and age <= 55) and gender = "Male" [
  if random-float 1 < 0.005320 [
   set number-dead-other number-dead-other + 1
   die
  ]
 ]
 if (age >= 56 and age <= 65) and gender = "Male" [
  if random-float 1 < 0.012868 [
   set number-dead-other number-dead-other + 1
   die
  ]
 ]
 if (age >= 66 and age <= 75) and gender = "Male" [
  if random-float 1 < 0.031500 [
   set number-dead-other number-dead-other + 1
   die
  ]
 ]
 if (age > 75) and gender = "Male" [
  if random-float 1 < 0.082420 [
   set number-dead-other number-dead-other + 1
   die
  ]
 ]

 if (age >= 16 and age <= 25) and gender = "Female" [
  if random-float 1 < 0.000338 [
   set number-dead-other number-dead-other + 1
   die
  ]
 ]
 if (age >= 26 and age <= 35) and gender = "Female" [
```

```
    if random-float 1 < 0.000567 [
      set number-dead-other number-dead-other + 1
      die
    ]
  ]
  if (age >= 36 and age <= 45) and gender = "Female" [
    if random-float 1 < 0.001447 [
      set number-dead-other number-dead-other + 1
      die
    ]
  ]
  if (age >= 46 and age <= 55) and gender = "Female" [
    if random-float 1 < 0.003260 [
      set number-dead-other number-dead-other + 1
      die
    ]
  ]
  if (age >= 56 and age <= 65) and gender = "Female" [
    if random-float 1 < 0.008018 [
      set number-dead-other number-dead-other + 1
      die
    ]
  ]
  if (age >= 66 and age <= 75) and gender = "Female" [
    if random-float 1 < 0.020539 [
      set number-dead-other number-dead-other + 1
      die
    ]
  ]
  if (age > 75) and gender = "Female" [
    if random-float 1 < 0.059150 [
      set number-dead-other number-dead-other + 1
      die
    ]
  ]
end

;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;
;;;;;;;;;;;;;;;;;; REPORTERS ;;;;;;;;;;;;;;;;;;;;;;;;
;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;

to reportDeaths
if year = 2001 [
   set IMD1deaths2001 IMD1-deaths
   set IMD2deaths2001 IMD2-deaths
   set IMD3deaths2001 IMD3-deaths
   set IMD4deaths2001 IMD4-deaths
   set IMD5deaths2001 IMD5-deaths

   set IMD1-deaths 0
   set IMD2-deaths 0
   set IMD3-deaths 0
   set IMD4-deaths 0
   set IMD5-deaths 0
  ]
```

```
if year = 2002 [
   set IMD1deaths2002 IMD1-deaths
   set IMD2deaths2002 IMD2-deaths
   set IMD3deaths2002 IMD3-deaths
   set IMD4deaths2002 IMD4-deaths
   set IMD5deaths2002 IMD5-deaths

   set IMD1-deaths 0
   set IMD2-deaths 0
   set IMD3-deaths 0
   set IMD4-deaths 0
   set IMD5-deaths 0
  ]

if year = 2003 [
   set IMD1deaths2003 IMD1-deaths
   set IMD2deaths2003 IMD2-deaths
   set IMD3deaths2003 IMD3-deaths
   set IMD4deaths2003 IMD4-deaths
   set IMD5deaths2003 IMD5-deaths

   set IMD1-deaths 0
   set IMD2-deaths 0
   set IMD3-deaths 0
   set IMD4-deaths 0
   set IMD5-deaths 0
  ]
```

….repeated for each year of the simulation until 2019…

```
if year = 2019 [
   set IMD1deaths2019 IMD1-deaths
   set IMD2deaths2019 IMD2-deaths
   set IMD3deaths2019 IMD3-deaths
   set IMD4deaths2019 IMD4-deaths
   set IMD5deaths2019 IMD5-deaths

   set IMD1-deaths 0
   set IMD2-deaths 0
   set IMD3-deaths 0
   set IMD4-deaths 0
   set IMD5-deaths 0
  ]

end
;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;
;;;;;;;;;;;;;;;; VISUALISATION ;;;;;;;;;;;;;;;;;
;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;

to patch-updateview
  set pcolor (5 + 10 * subareaNumber)
end
```