Essays on Inequalities in Health

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Abstract

This thesis comprises four empirical essays on the different aspects of health inequality. These four chapters are written in the form of self-contained articles.

Chapter 3 explores the effects of inherited socioeconomic characteristics on markers of unhealthy body weight. Taking Australian microdata from 2007 to 2013, we show that approximately 4% of the variation in outcomes is determined by factors beyond an individual's control, such as their race, gender and social class. Paternal socioeconomic status is the primary explanatory factor, with those born to more affluent fathers slightly less likely to be overweight in adulthood. Decompositions reveal that only 20%–25% of this effect is attributable to advantaged families exhibiting better health behaviours, implying that unobserved factors also play an important role. Since diseases associated with unhealthy weight significantly strain public healthcare systems, our results have implications for the provision of treatment when resources are constrained.

Chapter 4 specifies a multigeneration inequality-of-opportunity (IOP) model to study multigenerational health transmission mechanisms in Australian panel data. By applying IOP models, we demonstrate that grandparental socioeconomic status (SES) is an important determinant of personal health, even after controlling for health and SES at the parental level. Our findings hold over a range of mental and physical health outcomes and appear to be especially sensitive to educational outcomes on the father's side. Since ingrained socioeconomic (dis)advantages that persist over multiple generations may be indicative of "social class," our results suggest that subtle attitudinal and behavioural characteristics associated with this variable may be a key driver of health disparities.

Chapter 5 examines the effects of knowledge of HIV/AIDS (human immunodeficiency virus/ acquired immune deficiency syndrome) on HIV prevalence, instrumenting individuals' knowledge of HIV using the level of maternal education. We use pooled Demographic and Health Surveys data from 21 countries in sub-Saharan Africa to show that knowledge about HIV transmission effectively reduces infection rates. Our results persist across a variety of indicators and are plausibly causal. Since educational attainment is passed across generations, the empirical findings of this chapter suggest that individuals born to educated mothers are safer from HIV than those born to less educated mothers.

Chapter 6 presents new evidence of the causal effect of air pollution on Australian health outcomes, using the Black Saturday bushfires in 2009 as a natural experiment. This event was one of the largest bushfires in Australian history and emitted approximately four million tonnes of carbon dioxide into the atmosphere. We use data from the Household, Income and Labour Dynamics in Australia panel and compare the health status of individuals living in affected and unaffected regions before and after the event. Using a triple differences procedure, we further examine whether there is a difference in vulnerability to bushfire smoke by comparing people living in urban and regional areas. The findings of this chapter demonstrate that ambient air pollution had significant negative effects on health and that the magnitudes were higher for individuals residing in urban areas.

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Abbreviations

AIDS	Acquired Immunodeficiency Syndrome
BMI	Body Mass Index
BSB	Black Saturday Bushfires
CI	Concentration Index
CO	Carbon Monoxide
CO_2	Carbon Dioxide
DDD	Triple Differences
DHS	Demographic and Health Surveys
DiD	Difference-in-Difference
DV	Dependant Variable
EC	Elemental Carbon
ELISA	Enzyme-Linked Immunosorbent Assay
EOP	Equality of Opportunity
EQ	Equation
FE	Fixed Effects
GDP	Gross Domestic Product
GFE	Grandfather Education
GFO	Grandfather's Occupation
GFPE	Grandfather in Paid Employment
GFU	Grandfather Unemployed
GH	General Health
GME	Grandmother Education
GMO	Grandmother's Occupation

GMPE	Grandmother in Paid Employment
GP	Grandparents
GPD	Grandparent Divorced
HILDA	Household, Income and Labour Dynamics in Australia
HIV	Human Immunodeficiency Virus
HS	Health Satisfaction
IOP	Inequality of Opportunity
IV	Instrumental Variable
MH	Mental Health
MLD	Mean Log Deviation
MOS	Medical Outcome Study
OC	Organic Carbon
OLS	Ordinary Least Squares
PAH	Polycyclic Aromatic Hydrocarbons
PH	Physical Health
PM	Particulate Matter
RII	Relative Index of Inequality
SAH	Self-Assessed Health
SES	Socioeconomic Status
SII	Slope Index of Inequality
STI	Symmetrized Theil Index
TI	Theil Index
USAID	United States Agency for International Development

Statement of Originality

This work has not previously been submitted for a degree or diploma in any university. To the best of my knowledge and belief, the thesis contains no material previously published or written by another person except where due reference is made in this thesis itself.

(Signed)___  _____

Namal Nishantha BALASOORIYA MUDIYANSELAGE

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Chapter 1

Introduction

1.1 Background and Motivation

In recent years, increasing attention has been paid to inequality in health, largely because it has become a global challenge (Cairns et al., 2017; Gravelle et al., 2002; Lago et al., 2018; Subramanian and Kawachi, 2004; Lordan, Soto, Brown, & Correa-Valez, 2012). Although the summary statistics (World Health Organisation, 2019) demonstrate that developed countries tend to have better health than developing countries, recent empirical studies suggest that the association between gross domestic product (GDP) and life expectancy is not consistently positive and significant (Hu & Lenthe, 2015). Nonetheless, among poor people, a higher mortality rate (Michelozzi et al., 1999; Shishehbor et al., 2006) and lower life expectancy (Wagstaff & Doorslaer, 2000) have significantly prevailed. In addition, a relatively lower socioeconomic status (SES) has a significant correlation with poor health (Cairns et al., 2017; Lago et al., 2018), such that a higher level of income, education and occupational status are associated with better health. For instance, the prevalence of diseases is relatively higher among people from poor socioeconomic backgrounds (Fotso & Kuate-Defo, 2005). Tobacco use, unhealthy diet, physical inactivity and the harmful use of alcohol are the major risk factors in this group (Alwan & Maclean, 2009).

Health inequality is generally explained as the differences in health between individuals and social groups.[1] In developing this inequality explanation, health disparity has been measured at both group and individual levels (i.e., group level and overall health distribution) (Arcaya et al., 2015). Researchers have focused on group-level inequality measures to understand socioeconomic inequality in health, such as health disparities by gender, income level, race, occupational level, educational level and social class. In contrast, individual-level inequality is measured by considering all people in one distribution. However, evaluating group-level health inequality is the most commonly used approach because it allows policymakers to recognise vulnerable groups and provide resources to minimise group-level disparities.

[1] According to the World Health Organization (2013), the stratification of social groups is based on individuals' race, gender, religion, place of living, occupation, education, SES and social capital.

The intergenerational transmission mechanism plays an important role in socioeconomic or social class disparities. The evidence on socioeconomic mobility shows that parental SES positively impacts offspring SES (Andersen & Jæger, 2015; Beller, 2009; Hout, 1988). However, the duration of individuals in specific social classes matters for transferring the advantages or disadvantages from one generation to the next (Solon, 2014). For instance, the evidence suggests that families in higher social classes have held higher socioeconomic status for generations and vice versa (Chan & Boliver, 2013; Erikson & Rudolphi, 2010; Erola & Moisio, 2007; Hertel & Groh-Samberg, 2014; Paulsen, 1991; Solon, 2014). These elements of the social class create differences in individuals' material lifestyles between upper and lower classes (such as different neighbourhoods, different educational institutions, belonging to different social clubs, engaging in various recreational events and eating different foods). These differences may influence individuals' childhood outcomes and their later life outcomes.

Childhood experiences (including *in utero* and infanthood) are significant determinants of the health of individuals. Those who grew up with disadvantaged backgrounds face greater health risks than those with advantaged backgrounds. For example, children born to well-off parents are healthier than children of less-educated, poorer parents (Currie & Goodman, 2020; Huebener, 2019). Several potential channels can direct this effect on the children. Firstly, educated mothers can provide nutritious food and better healthcare and encourage their children to have a healthy lifestyle. Secondly, an impact on the parents' health passes through to children's education and employment. In addition, since educated parents tend to have better health and behaviour, their children may also have this health benefit in, for example, the intergenerational transmission of health and behaviours (Thompson, 2014; Wickrama et al., 1999).

Correlations between parental characteristics and child outcomes are often interpreted under the umbrella of *inequality of opportunity* (IOP) (Roemer, 1998), which comprises harmful disparities that lie beyond personal control. IOP justifies policies that focus on childhood circumstances to break the links in a child's family's SES (e.g., parental and grandparental SES) and may partially reduce health inequality. For example, since equality of health care is no longer practical due to scarce medical resources, prioritising people for health care based on their circumstances is a legitimate policy option (see Siciliani, 2016).

The normative framework of IOP offers a partial solution to this problem. Roemer (1998) posits two sources of inequality: *circumstances* and *effort*. Factors of *circumstances* are

predetermined and inherited in nature (such as genetics, ethnicity, gender and parental characteristics), are considered beyond individual control and are therefore treated as unfair sources of disparity in outcomes. On the other hand, inequalities in individuals' outcomes due to effort factors are considered fair or legitimate disparities because individuals can at least partially control these factors (such as behaviours and attitudes).

Following Roemer (1998), researchers have studied inequality in health by using different health outcomes for the last two decades. Most of these empirical investigations (Bricard et al., 2013; Jusot et al., 2013; Rosa Dias, 2009; Trannoy et al., 2010) have modelled self-assessed health as the outcome variable. Other studies (Carrieri & Jones, 2018; Rosa Dias, 2010) have used various biomarkers such as chronic illness and disability (including mental health conditions) to address heterogeneous patterns in health outcomes. As with the research presented here, these studies have focussed mainly on parental SES and family background as measures of circumstances and have used lifestyle behaviours as proxies for effort (Bricard et al., 2013; Carrieri and Jones, 2018; Jusot et al., 2013; Rosa Dias, 2010). Nonetheless, in the empirical literature on IOP in health, most have focused on health markers related to non-communicable diseases or aggregate measures for overall physical and general health.

As well as social groups, people's environmental and geographic contexts influence differences in outcomes because exposure to health risk factors or protective factors may depend on an individual's precise location. For example, exposure to air pollution and natural hazards may vary according to an individual's place of residence and working environment. There are volumes of empirical studies that have examined the effect of air pollution on respiratory health, cardiovascular disease, chronic obstructive pulmonary disease, asthma and lung cancer (e.g., Dennekamp et al., 2015; Franzi et al., 2011; Hamon et al., 2018; Kim et al., 2018; Navarro et al., 2019; Pavagadhi et al., 2013) and the effect of psychological stress on birth outcomes (e.g., Holstius et al., 2012). Bryant et al. (2020) considered the incidence of bushfires: using longitudinal data before and after the wildfires, they examined bushfire's long-term effects on mental health outcomes. Gallagher et al. (2016) used a cross-sectional survey to demonstrate the mental health impacts due to separation from family during bushfires. Furthermore, Johnston et al. (2021) have also identified a reduction in life satisfaction for people living in bushfire-affected areas.

1.2 Research Objectives

Against this background, the main aim of this thesis is to undertake four detailed, self-contained empirical studies on four different issues of health inequality linked with individuals' socio-economic and geo-environmental disparities. The objective of each study is:

1. To model and measure the inequality of opportunity in body weight by determining: (i) What are the most harmful circumstances of disparity in the distribution of Australian bodyweight? (ii) Whether the correlations between circumstances and body weight emerge through differences in health behaviours? (iii) How do correlations between circumstances and behaviours affect IOP estimates?

2. To model the effect of grandparental characteristics on grandchildren's health and measure grandparents' contribution to IOP in health. While fulfilling these objectives, the following questions are considered: (i) Is grandparental SES a source of unequal opportunity in Australian health? (ii) What is the contribution of grandparents on IOP in Australian health outcomes?

3. To estimate the causal effect of HIV/AIDS knowledge on the prevalence of the disease. While reaching this objective, the study deals with the endogeneity issue due to reverse causality. Since HIV is a human infectious virus that mainly transmits through risky sexual behaviours, people's awareness or knowledge are important determinants of HIV prevalence. It is most likely that educated individuals are knowledgeable about HIV transmission before they attend HIV test clinics. Nonetheless, some lower-educated, disadvantaged individuals may become aware after they are tested for HIV (see Hutchinson et al., 2006). Because of this causality in the relationship, the coefficient of basic regression estimates of HIV awareness on HIV prevalence cannot be defined as the causal effect.

4. To quantify the effects of air pollution on health by employing a natural experimental approach, considering the Black Saturday bushfires that occurred in the Australian state of Victoria in 2009. Since these fires provide an exogenous shock to air particulates, the study focuses on the following questions: (i) Does air

pollution have a causal impact on human health? (ii) Which groups of people are primarily influenced by air pollution?

1.3 Data and Methodology

The objectives are achieved using appropriate econometric applications and widely- used cross-sectional and panel data. Three of four empirical research exercises (Chapters Three, Five and Six) used data from the HILDA (see below) panel. However, the first study limits its analysis to the 2007, 2009 and 2013 survey years, and the second study's data consists of 17 waves (from 2001 to 2017), whereas the fourth study the nine waves of the restricted version of the HILDA longitudinal survey from 2004 to 2012. Data for the third study are drawn from demographic and health cross-sectional household surveys (DHSs) from 21 counties in sub-Saharan Africa from 2006 to 2019.

HILDA Longitudinal Survey

The Household, Income and Labour Dynamics in Australia survey (HILDA) is an approximately nationally representative panel. Since 2001, HILDA has collected data annually on individual health status and demographic and socioeconomic background through face-to-face interviews and self-completion questionnaires. The data cover more than 9,000 Australian households and follow approximately 200,000 individuals. In accordance with the changes in household composition, new household members were added to the sample. In Wave 11, the sample size was increased by an additional 2,153 households.

From Wave 1, HILDA has included four different instruments: the household form, the household questionnaire, the person questionnaire and the self-completion questionnaire. In subsequent waves, the person questionnaire continues for those individuals interviewed in the last wave, while newly added individuals complete the new person questionnaire. Since additional households were included from Wave 11, the household form and new person questionnaire were top-up. The household form is the master document, used to record basic household information and administrated by interviewers. The household questionnaire collects the household information from one member. Every member of the household aged 15 or above is considered in the personal questionnaire.

The Australian Government's Department of Social Services funds the survey, while the Melbourne Institute of Applied Economic and Social Research at the University of

Melbourne conducts and manages HILDA. Information about HILDA's methodology is published in the HILDA user manual (see Summerfield et al., 2020).

DHS Data

A Demographic and Health Survey (DHS) is designed to collect a range of information about an individual's socioeconomics, demographics, health & wellbeing, behaviours and family life from a nationally representative cross-sectional sample. Currently, the DHS program covers over 90 countries in Asia, Africa, Latin America and the Caribbean using funds from the United States Agency for International Development (USAID) and participating countries. The surveys are managed by the authorised government institutions of each country.

The standard DHS consists of a large sample (varying from 5,000 to 30,000) and is conducted within a five-year range. The survey uses the stratified two-stage cluster sampling to draw a representative sample at the national, residential, and regional levels (e.g., states). Enumeration areas are selected from the county's census files in the first stage. Next, a sample of households is selected from each enumeration area chosen in the second stage. The sample size is estimated based on the urban and rural population proportion and gender ratio. The data collection is conducted using separate standardised questionnaires for household- and individual-level data for women and men separately. In addition, individuals' HIV test results are included in most surveys.

Econometric Applications

As noted above, this thesis consists of four empirical essays (as chapters). The empirical methodology used in each essay will be presented in detail. Therefore, this section briefly describes the econometric applications used for the empirical analysis.

In Chapter 3 (the first empirical essay), regression models are specified based on the concept of IOP. Therefore, the estimations are derived in three steps. In the first step, we define a reduced-form IOP model that measures the impact of only the observable circumstances on health outcomes (e.g., body mass index (BMI) and measure of over-weightiness). The second step includes some demographic control variables such as age, marital status, employment status and area of residence. Finally, since individuals' health behaviours are correlated with their circumstances, we introduce additional health behaviour variables into the model to determine if they mediate the empirical links with outcomes. This approach allows us to test pathway and latency hypotheses on the

intergenerational transmission channels. In addition, in order to study correlations between circumstances and behaviours, we estimate regressions of each behaviour factor on circumstances. By exploiting an econometric framework introduced by Jusot et al. (2013), we evaluate the consequences of the correlation between circumstances and behaviours to IOP estimates. All the models are fitted using ordinary least squares (OLS).

Chapter 4 (the second empirical chapter) again applies IOP concepts to Australian health data. Here, based on the assumption that individuals' *grandparents* may play a role in IOP, the standard IOP model is extended to consider these effects. Therefore, we model the impacts of grandparental SES on individuals' health while also controlling for similar parental traits. This approach allows our models to capture a direct effect of grandparental status rather than an effect that flows through the intermediate (i.e., parental) generation. We then specify two econometric models. In the first, we estimate the baseline IOP model, including parental characteristics, while controlling some socioeconomic and demographic confounders. In the second model, we test the effect of grandparents while controlling for all the covariates in the first model. Furthermore, we quantify the grandparents' contribution to IOP using regression-based econometric decompositions. All the regressions in this chapter are estimated using OLS.

Chapter 5 (the third empirical chapter) models the impact of HIV knowledge on HIV/AIDS prevalence. However, in this relationship, we assume that the regression coefficient of interest cannot be interpretable as a causal effect of HIV knowledge on the risk of HIV infection because of the potential reverse causality between these two factors. Therefore, we estimate this causal effect using instrumental variable (IV) regression. We exploit both OLS and probit regression estimates as a robustness check.

In Chapter 6 (the fourth empirical chapter), a quasi-experimental approach is used to analyse the causal effect of bushfire pollution on health outcomes. To quantify the causal health effect of air pollution, we estimate the standard difference-in-difference (DiD) regression model using OLS. We then extend the DiD model to a triple differences (DDD) model to analyse whether heterogeneity in exposure to ambience differs in response to less health status after bushfires.

1.4 Structure of the Thesis

This thesis consists of seven chapters. This introductory chapter provides an overview of the background and motivation of the thesis, its objectives, data sources and econometric applications. The next chapter reviews concepts and measurements of health inequalities

to provide the context for four empirical studies in the thesis. Following this, Chapters Three to Six present four separate empirical but related to health inequalities.

Chapter 3 applies IOP concepts to identify harmful sources of disparity in bodyweight. Obesity (and its variants) make an interesting case study for IOP analysis because it is highly reflective of lifestyle choices that can be plausibly classed as "effort" relative to other health conditions. This chapter examines the effects of various circumstance variables on Australian body weight. The chapter then extends the analysis to examine how correlations between an individual's circumstances and weight arise by using additional data on health behaviours, including diet, exercise, and alcohol and tobacco consumption. Finally, the chapter examines the consequences of the correlation between circumstances and behaviours to IOP.

Chapter 4 extends the standard IOP model to consider the effect of grandparents on individual health. The chapter thus studies the role of grandparental characteristics in affecting the health outcomes of their grandchildren. It extends the analysis to measure grandparents' contribution to IOP based on regression-based decomposition.

Chapter 5 presents the third empirical study on the effect of HIV knowledge on HIV/AIDS prevalence inequality using pooled DHS data from 21 countries in sub-Saharan Africa. The chapter focuses on the potential identification issue arising from the reverse causality between variables of interest. Based on the results, this chapter highlights the importance of maternal education in controlling HIV by improving individuals' HIV education.

Chapter 6 presents the final empirical study, investigating the causal effect of air pollution on health outcomes by considering Australia's 2009 BSB as a natural experiment. The data source is the restricted version of HILDA. This chapter presents new evidence of the causal effect of air pollution on Australian health outcomes. It then investigates whether the heterogeneity of exposure to ambient air pollution plays a role in health inequality.

Chapter 7 concludes the whole thesis. It summarises the four empirical studies' main research findings and discusses policy implications and recommendations. The chapter also highlights the limitations of the thesis and future research paths.

Chapter 2

Health Inequality: Concepts and Measurement

2.1 Introduction

Since the 1980s, there has been significant attention to the problem of health inequalities among policymakers, researchers and health professionals; nevertheless, health inequality is still a global issue. For example, World Health Organisation (2022) reported that a healthy life expectancy in low-income countries is 57 years, while people in high-income countries expect 70 years of healthy life. Moreover, there are substantial health differences between social groups within countries (Bollen et al., 2001; Feinstein, 1993; Lago et al., 2018; Pickett & Wilkinson, 2015; Shiels et al., 2017). Therefore, researchers focus on developing new concepts and applications and using them to reach a feasible solution for improving overall population health and minimising or irradicating the health inequities between social groups.

The Black Report, which examined health inequality in the United Kingdom, has been an influential document in the UK and elsewhere in the health inequality literature. Most theoretical and empirical developments in the health inequality literature in the last four decades have adopted the concepts, questions and recommendations discussed in the Black Report (Brocklehurst & Costello, 2003; Macintyre, 1997; Vågerö & Illsley, 1995). Its recommendations underlined the importance of improving disadvantaged groups' physical and social environments. Consequently, new developments in health inequality literature focus on inequality related to sociocultural, economic, environmental, behavioural, societal or biological factors.

This chapter describes some theories and measurements related to health inequality. The following section provides a brief overview that clarifies some vocabulary used to explain health inequality. The following section explains theories that have been commonly employed to understand health disparities. After that, some widely used health measures and inequality indices are explained. The last section briefly explains the theoretical and empirical development of the inequality of opportunity.

2.2 Health Inequality: An Overview

Recent evidence reveals that health inequality due to disparities in the distribution of resources is widening (Elgar et al., 2015; Olshansky et al., 2012). Similarly, the economic

burden of health inequity has been staggering (Laveist et al., 2011). There is concern about such health disparities because they offend against fundamental beliefs about equity and justice (Sen, 2002) and can be interpreted as objectionable to human rights (Sen, 2008). Consequently, World Health Organisation and the United Nations have prioritised health equity (Marmot et al., 2008) and have intensified the measurement and analysis of health distribution. Economists, in particular, have paid substantial attention to such issues and have made many empirical and theoretical contributions to health inequality literature.

Even though there have been substantial empirical and theoretical developments in health disparity, it is hard to find a general definition of "health inequality". Nonetheless, two concepts have been operationalised in the literature to examine health inequality within and between populations (e.g., Wagstaff & van Doorslaer, 2000). One approach considers the social group differences in health (Braveman et al., 2000; Wagstaff et al., 1991) by, for example, comparing the mean BMI of high-income and low-income groups. This approach examines the distribution of health outcomes across social and economic groups using joint distribution measurements such as concentration curves and the concentration index (Bommier & Stecklov, 2002). Measuring health inequality by the group is widely adopted because group-level health inequality considers the health differences between advantaged and disadvantaged groups and has implications for reducing group-level inequalities (e.g. World Health Organisation, 2013).

Alternatively, the second approach deals with health inequality across individuals—for example, variations in nutrition levels across a whole population. Although this approach has relatively less weight in the health inequality literature, it can be used to compare the health status of individuals from similar backgrounds (Arcaya et al., 2015). The pure inequality approach considers the distribution of health outcomes for the entire population (see Murray et al., 1999) and uses standard inequality measures such as the Gini index and Lorenz curve to measure overall health inequality.

Defining social groups is crucial for evaluating group-level inequality because cultural, historical, political and religious characteristics shape social class divisions. For example, caste in South Asia is a better indicator of social demarcation. While the distinction between black and white Americans is meaningful in the United States, individuals' educational achievement and occupation define social class divisions in the United Kingdom. Moreover, according to Gillespie et al. (2012), researchers must be aware that

social categories are perspectival, historical or cultural, that change with peoples' movements and, therefore, can interfere with the phenomenon.

In addition, the health inequality literature defines groups according to individuals' absolute and relative social position. This is mainly based on individuals' income.[2] Absolute income measures are based on fixed income thresholds, which mainly use the poverty line, while relative income is defined based on the income distribution of the corresponding population. Therefore, according to the absolute income hypothesis, individuals' health depends on their own income and does not vary by neighbourhood earnings. Therefore, this hypothesis does not accept that the effect of private income varies between societies (Kawachi et al., 2002). However, a great deal of empirical evidence reveals that relative income has a role in health inequality (see Wagstaff & van Doorslaer, 2000). This literature claims that, in general, humans compare themselves with those around them. Therefore, people may suffer physiological distress and stress related to health if they cannot reach the level of their neighbours. Since the relative income hypothesis considers the subjective measure of wealth, the effect of income on health is psychologically linked.

Social groups are usually measured using nominal and ordinal categories (Dressler et al., 2005). Alternatively, some are based on continuous measures such as separating economic groups based on income. Researchers generally use some theory or *a priori* contextual knowledge to construct clearly defined membership categories. When using ordered or continuous variables, researchers consider whether the effect of socioeconomic resources on health outcomes can be concluded according to the *threshold model* and whether socioeconomic gradients in health show a *dose-response relationship*. The socioeconomic gradient in health occurs if an increase in socioeconomic status corresponds with increasing health in a dose-response relationship. The socioeconomic gradient in health is evident in every socioeconomic spectrum (see Marmot et al., 1991; Marmot & Mcdowall, 1986). For example, an increase in the level of health in response to increasing socioeconomic resources does not mean that individuals in higher social classes will always be healthier than those in the middle class (i.e., a threshold effect of social class on health).

[2] Some instances use multiple ordered stratification variables (e.g., employment, education, diet, clothing, housing) or multidimensional indices (e.g., multidimensional poverty index) to measure social position.

Researchers have focused on fundamental concepts to understand and measure group differences in health. Firstly, causal pathways and conditional health effects are useful concepts focusing on third-party involvement between outcome and exposure. The causal pathway explains how the third variable mediates outcome and exposure, while the framework of conditional health effects explains the conditions that flow from the exposure to the outcome (Baron & Kenny, 1986). In this case, the third variable is called a "mediator" and "moderator" in the concept of causal pathways and conditional health effects, respectively.

Secondly, researchers attend to the correlation between individual experiences across lifespan and health, known as the "life course perspective" on health inequality (Wadsworth, 1997). This framework studies the persistence of health inequality due to childhood or *in utero* circumstances. Since family characteristics (mainly parental characteristics) drive childhood experiences that link with health determinants, the life course perspective of health disparities can be interpreted as inter(multi)generational social class disparities in health. In the economic literature, the impact of predetermined factors on individual outcomes is explained by the concept of inequality of opportunity that has been used to examine health inequality for the last two decades (see Jusot & Tubeuf, 2019).

In addition, the evidence using longitudinal data explores the lag or cumulative effect of neighbourhoods on driving health disparities (e.g., Arcaya et al., 2014; Ludwig et al., 2011). Furthermore, the choices of individuals throughout life (such as choosing a residence, friends or workplace) may influence their health—that is, the *selection* effect of health inequality. For example, according to people's lifestyle requirements, someone may select a crowded neighbourhood while others prefer living in calmer environments. These neighbourhood differences may matter for health disparities (see Alcock et al., 2014; Ineichen & Hooper, 1974; Schulz et al., 2000).

Besides social group inequality, researchers such as Jones and Moon (1993) and Kearns and Joseph (1993) have discussed the role of geographical context in health inequality. These authors have developed the concept of geographical differences in health considering the concepts of *space* and *place.* S*pace* connects with health inequality due to the disparities of exposure or proximity to health risk and protective factors according to individuals' specific location. Examples are the health risk of air pollution that spreads across space and proximity to crime clusters, natural disaster zones or hospitals. *Place*

involves the inequality related to government healthcare programs or policies because the impact of these public or private institutional involvements depends on how individuals are members of different political or administrative units, such as countries, districts, states or cities. The well-known paper "Is there a place for geography in the analysis of health inequality?" by Curtis and Jones (1998) shows how people's geographical differences are significant in describing health inequality.

2.3 Theories of Health Inequality

Since the publication of *The Black Report: Inequalities in Health* in the United Kingdom, several theoretical developments have sought to explain how individual SES drives health outcomes (Brocklehurst & Costello, 2003). The Black Report distinguished four root causes of health inequality: artefact, selection, behavioural/cultural and structural (McCartney et al., 2013). Most theoretical developments after the Back Report focus on the conclusion that life expectancy is strongly related to a social and economic position—social class. However, current theoretical explanations of health inequality consist of the social determinants of health, fundamental causal theory, the eco-social model and the political economy of health.

2.3.1 Social Determinants of Health

The social determinants approach describes the role of social and economic factors in shaping the health status of individuals and groups (Marmot, 2005; Marmot & Mcdowall, 1986). According to this idea, socioeconomic position and health status follow a social gradient; it concludes that the lower the socioeconomic status, the worse the health. Thus, action is required to reduce socioeconomic inequalities for reducing health inequality. Even though many factors influence health inequalities among individuals and groups, socioeconomic factors play a key role.

Overall, this approach shifts the explanation of health disparity from biological mechanisms to social structures to specify the role of cultural, behavioural, material, psychological and life-course factors in health. (Bartley, 2004; Skalická et al., 2009).

Cultural and Behavioural Model

Under this model, the differences in the behaviours and cultures of social groups or individuals are basic drivers of health disparities. Thus, even though health behaviours and cultural practices are considered to be causes of health inequality, socioeconomic

characteristics act as a modifier in the relationship. For example, behavioural factors contribute to health inequality due to differences in lifestyles such as eating, smoking and physical activities (e.g., Bickel et al., 2014).

Materialist Model

The materialist model describes health inequality as due to disparities in absolute resources: health inequality arises from variations in the availability of basic requirements. The role of economic and other associated social characteristics in the distribution of health and wellbeing are thus explained (Townsend et al., 1992): differing social positions are linked to different physical and material life conditions which drive health status.

Psychological Model

The psychological viewpoint views health inequality as a consequence of psychological stress and tries to reveal the psychological pathway between socioeconomic disparities and health (Marmot et al., 1998). For example, individuals in lower social classes suffer stressful life events such as less social support, less autonomy at the workplace, less job security and less income; these can lead to many health issues.

Life-course Model

The life-course model had initially been applied to understanding human life from a sociological and psychological perspective in the 1920s (Elder, 1998). However, this term has been used in health inequality literature since a pioneering study that examined the effect of childhood health on life expectancy (G. D. Smith & Kuh, 2001). This approach explains the long-term effect of being exposed to physical, social and economic conditions from gestation to later adulthood on health. The idea is that while individuals' current socioeconomic conditions or lifestyles shape health inequality, the past conditions of life also matter. Life-course approaches are important tools that have been used to examine the persistence and transmission of health inequality across generations (Braveman & Barclay, 2009; Kuh et al., 2003). Therefore, social scientists, as well as epidemiologists, widely accept this model.

2.3.2 Fundamental Causal Theory

The fundamental causal theory claims that individuals in higher socioeconomic clusters experience better health due to higher resource availability than those of lower SES. Therefore, a lengthy advantage acts as a safeguard against existing health threats (Willson, 2009). Although earlier sociological viewpoints held that SES does not have a causal link to health (Phelan et al., 2004), individuals in higher social classes have greater resources availability (such as education, money, social connections and social power) to eliminate health risks in their environment (Link & Phelan, 1995). Using these resources leads decisively to the persistence of a strong relationship between SES and health outcomes, regardless of current SES. The basic idea of this theory is that removing the proximal risk factors of health does not mitigate the impact of SES on health (Link & Phelan, 1995). Instead, on the one hand, the resources linked to higher SES—such as a higher level of education—direct individuals to protect themselves from new health risk factors that mediate the correlation between SES and health. On the other hand, the same advantage helps individuals to access better health care services (Lutfey & Freese, 2005).

2.3.3 Social-Ecological Model

The basic idea of the social-ecological model in health was discussed in the 1947 World Health Organisation's constitution and, as a theory, was propounded in the 1970s (e.g., Bronfenbrenner, 1979; Garbarino & Crouter, 1978). This model considers the multiple factors that may affect health status. For example, a social-ecological approach tries to understand health disparities as a consequence of the interconnection between individuals, groups or communities and the proximal and social environment (physical, behavioural, sociocultural, political, etc.) (Green et al., 1996; McCloskey et al., 2011). Stokols (1996) has proposed four core principles to understand the complex relationship between socio-ecological factors and health:

(1) Individuals' physical, social and cultural characteristics affect health status, well-being and social cohesion.

(2) Similar social settings may have a different health impact, but this depends on the individuals' experiences of controlling financial and environmental resources.

(3) There is a spillover impact with multiple health as individual and group engagements (such as workplace and neighbourhood).

(4) Individual and environmental leverage points where resource availability, the physical environment and social customs exert a significant impact on health.

Krug & Dahlberg (2002) explained four levels of social-ecological theory (see Figure 2.1). The first level includes individual characteristics such as age, gender, education, income and past health conditions. The second level considers individuals' closest relationships, such as friends, family members or partners who significantly influence personal behaviours, attitudes and experiences. In addition, the health impact of exposure to communities such as schools, workplaces and neighbourhoods comprises the third level. The fourth level considers societal factors such as cultural and social standards, policies, and socio-economic and educational backgrounds that influence health and well-being.

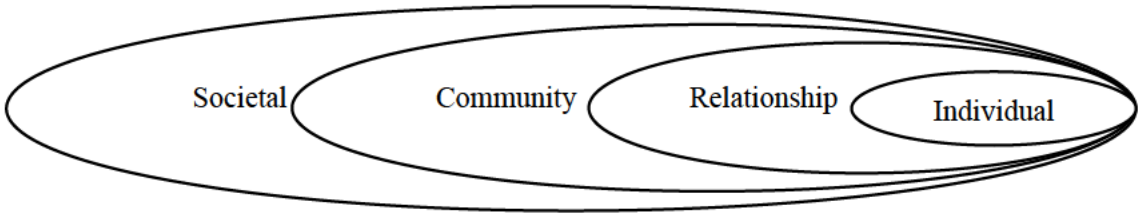


Figure 2.1 The Social-Ecological Model

2.3.4 Political Economy of Health

The political economy of health focuses on how individuals or the population's health status varies due to differences in the political and economic domains. The idea is that material, cultural-behavioural and psychosocial explanations of health inequalities are rooted in structures according to the political and macroeconomic environment that determine the distribution of the social determinants of health outcomes and population health (Bambra et al., 2005, 2019). The political economy application to health was raised by the significant work of Doyal and Pennell (1979). This approach is environmentally and socially focused. It has, for example, been recently applied in health sociology and social epidemiology to study the health differences between countries (e.g., Navarro & Muntaner, 2004) and social groups (Bambra et al., 2005).

2.4 Measures of Health Inequality

The measures of health variables can be cardinal, ordinal or nominal. For example, BMI is a cardinal health variable, while self-assessed health (SAH) is an ordinal health variable. Similarly, socioeconomic variables can be cardinal or categorical. Health and

socioeconomic variables measurements should match the inequality indices' properties when calculating inequality.

The measurement of health and the health inequality index has gained greater attention in recent decades. While most of the literature details the statistical properties of health inequality measures, there is disagreement among researchers about how health inequality should be measured (Nesson & Robinson, 2019). This issue has arisen because most mean-based summary inequality indices require cardinal health measures; however, most available health measures are categorical or ordinal. Therefore, researchers deal with two possible ways to measure health inequality: design indices to match ordinal or nominal variables or transform categorical data into scale data (Madden, 2010). Another issue is that some health variables suffer from reporting heterogeneity. Nevertheless, the literature does provide some solutions to this problem (e.g., Lindeboom & Van Doorslaer, 2004; Van Doorslaer & Jones, 2003).

The attention to inequality measurements has been raised in the health inequality literature after the influential paper of Wagstaff et al. (1991). They critically reviewed six different inequality measures (the range, Gini coefficient, pseudo-Gini coefficient, index of dissimilarity, slope index of inequality (SII) and the concentration index (CI)) and suggested the SII and CI as accurate measures of health inequality. This conclusion is primarily based on whether existing inequality measures consider socioeconomic and health distributions. For example, the range, Gini coefficient and pseudo-Gini coefficient are univariate measures that only consider the distribution of health outcomes, whereas SII and CI consider the joint distribution of health and socioeconomic statuses.

There are, in addition, methodological issues related to the classification of social position (e.g., occupationally based classification, educational classification, occupational together with educational, and a combination of income and education) and the quantification of health inequality (Manor et al., 1997). The different methods for quantifying health inequality are discussed below.

2.4.1 Quantitative Measures of Health

Existing quantitative health status measurements fall into two main groups: population health and individual health. Population health measures describe the health status of a group of individuals that may be grouped as a country, region or any geographical or administrative division by, for example, mortality rate, life expectancy, healthy life years,

neonatal mortality rate, maternal mortality ratio and percentage of newborns with low birth weight. Individual health measures indicate an individual's health status and can be a single dimension or aggregate health measure. A single dimension health measure indicates the conditions of specific diseases, like biological markers such as blood pressure, cholesterol level and blood sugar level. The aggregate health measures indicate the overall health condition of an individual and may use multiple dimensions such as self-rated/assessed health, physical and mental health indicators based on the SF-36 health survey, or BMI. This section explains some of these individual health status indicators that are most frequently used in health inequality literature.

Self-Assessed Health (SAH)

This is a widely used measure in empirical studies of an individual's health status**.** SAH is ordered into five categories, from very good to very poor health. Although SAH has been identified as a measurement with individual reporting heterogeneity (Bago D'Uva et al., 2008), it has been recognised as a better indicator of disease burden and a better predictor of mortality (Lorem et al., 2020; Thong et al., 2008). Moreover the continuous measure of SAH, which is constructed from the ordinal SAH using the method of Wagstaff & Van Doorslaer (1994), correlates with other continuous health variables (Gerdtham et al., 1999).

SF-36 Health Measures

This is the short form of the Medical Outcomes Study (MOS). Nevertheless, SF-36 is a widely employed, self-completion health assessment tool (Ware, 2000) which was developed according to psychometric standards to assess an individual's functional health and well-being and to measure the burden of disease (Ware & Gandek, 1998) among groups. This includes the 36 items (except one) used to measure eight concepts of health selected from 40 concepts contained in MOS. The eight concepts are mental health, role-emotional, physical functioning, social functioning, general health, role-physical, bodily pain and vitality (Butterworth & Crosier, 2004). In addition, summary scales for physical and mental functioning and well-being are constructed using these eight scales. Recent literature (McCallum, 1995; Sanson-Fisher & Perkins, 1998; Yarlas et al., 2018) concludes that SF-36 is a valid, reliable and responsive tool for measuring health status.

The improved form of SF-36 was available from 1998. To quantify health outcomes, this developed version (i.e., Version 2) applies norm-based scoring algorithms (i.e., T-score transformation with mean, 50 ±10 [SD]) (Ware, 2000, p3131).

The Body Mass Index (BMI)

BMI is considered a proxy for individual health status because body weight indicates the health risks of non-communicable diseases like blood pressure, strokes, heart disease and cancers.

The use of BMI as a health measure carries some standard caveats. It is, for instance, not sensitive to the difference between body fat and muscle and does not consider types of fat that have a different metabolic effect and which part of the body contains more fat (Stevens et al., 2008). Moreover, since there is a possibility of measurement error due to self-reported weight and height, the calculation all also a bias associated with the respondent's characteristics (Rothman, 2008). However, these biases can be minimised by using continuous BMI instead of the categorical version (Stommel & Schoenborn, 2009). Although it has some significant limitations, BMI has been a widely used health measure in health-related research, showing a robust association with non-communicable diseases (Nuttall, 2015; Stommel & Schoenborn, 2009).

BMI is a ratio that is weight in kilograms over the square of height in meters. According to the standard classification, the normal weight category (18.5–24.9) is healthy, while the others (underweight = < 18.5; pre-obesity = 25.0–29.9; obesity class I = 30.0–34.9; obesity class II = 35.0–39.9; obesity class III = > 40) indicate risk categories (World Health Organization, 2000).

2.4.2 Single Distribution Measures

Here, we consider univariate measures based only on the distribution of health variables for the entire population. These measures are employed to determine health inequality across individuals and compare the health status of individuals from similar backgrounds (Arcaya et al., 2015).

Gini Index

This index is a univariate measure of health inequality, originally developed to measure income inequality (Atkinson, 1970). When estimating health inequality, the Gini index

can be defined as follows. The index is based on the commutative health of the poorer individuals. Let *f(y)* be a probability density function of a continuous random variable *Y* that is the health variable, and *F(y*) is the health variable's commutative distribution function. The index calculation is based on the Lorenz curve, where the sickest individuals indicate the cover's beginning and the healthiest are located at the end of the curve. The value of the index ranges from 0 to 1. The Lorenz curve (Langel & Tillé, 2013) is given by

$$L(\alpha) = \frac{\int_0^{F^{-1}(\alpha)} yf(y)dy}{\int_0^{\infty} yf(y)dy} = \frac{1}{\mu}\int_0^{\alpha} F^{-1}(u)du, \qquad (2.1)$$

where F^{-1} (.) is the inverse function of *F*(.) and

$$\mu = \int_0^{\infty} yf(y)dy.$$

Then the Gini index can be defined as:

$$G = 2\int_0^1 \{\alpha - L(\alpha)\}d\alpha = 1 - 2\int_0^1 L(\alpha)d\alpha = \frac{2}{\mu}\int_0^{\infty} yF(y)f(y)dy - 1. \qquad (2.2)$$

Moreover, if y_i, (i = 1…….n) is a series of positive random variables with the *f*(.) probability density function, the Gini index (*G*) can be estimated by

$$\hat{G} = \frac{2\sum_{i=1}^{n} iy_{(i)}}{n\sum_{i=1}^{n} y_{(i)}} - \frac{n+1}{n}, \qquad (2.3)$$

where $y_{(i)}$ is y_i arranged in ascending order (Ceriani & Verme, 2012; Langel & Tillé, 2013).

The Gini index was first used to measure health inequality by Le Grand & Rabin (1986). Subsequently, Leclerc et al. (1990) compared health inequality in four European countries. Moreover, Wagstaff et al. (1991) estimated health inequality using group data instead of individual data. Because they used group data, they referred to the Lorenz curve as a pseudo-Lorenz curve. Following these studies, the Gini index has been widely used for the last three decades (Yi Tao et al., 2014).

Entropy-Based Health Inequality Measures

The Theil index (*TI*) and mean log deviation (*MLD*) are widely used entropy-based inequality measures between two probability distributions (Borrell & Talih, 2011). These measures were initially proposed to estimate income inequality (Firebaugh, 1999; Theil, 1967). However, they have been commonly used to measure inequality in health (Borrell & Talih, 2011; Harper et al., 2008; Levy et al., 2006; Pearcy & Keppel, 2002). These indices range from 0 to 1, where a smaller value indicates more inequality and has been used to measure disparities in individual outcomes, including health. Since these indices are decomposable by groups and can be applied to group-level data, they have been employed to estimate inequality in health resources allocation (e.g., Harper et al., 2008; Harper & Lynch, 2006). *TI* and *MLD* are specified as follows.

$$TI = \sum_{i=1}^{n} (y_i/y) \times ln\left(\frac{y_i/y}{1/n}\right) = \frac{1}{n}\sum_{i=1}^{n} (y_i/\bar{y}) \times ln(y_i/\bar{y}) \qquad (2.4)$$

$$MLD = -\sum_{i=1}^{n} (1/n) \times ln\left(\frac{y_i/y}{1/n}\right) = -\frac{1}{n}\sum_{i=1}^{n} ln(y_i/\bar{y}) \qquad (2.5)$$

Here, in both Eq. (2.4) and (2.5), y_i represents a cardinal health measure for the *i*th individual in a sample of *n* and (y_i/y) is each individual's contribution relative share to the sample population aggravate (y). *TI* and *MLD* consider that each individual's leaving from fairness is weighted by (y_i/y) and ($1/n$).

Due to the inherent asymmetry of both *TI* and *MLD*, these measures require a value adjustment concerning higher disease frequency in *TI* and groups with a large population share in *MLD* (Borrell & Talih, 2011; Levy et al., 2006). Nevertheless, the symmetrised Theil index (*STI*) is the average of *TI* and *MLD,* a symmetric which, therefore, overcomes a value judgement issue in *TI* and *MLD*. *STI* is also a semi-metric measure, whereas *TI* and *MLD* are pre-metric. *STI* can be therefore defined as follows. Interpretation of the components is similar to *TI* and *MLD*.

$$STI = \frac{1}{2n}\sum_{i=1}^{n} (y_i/\bar{y} - 1) \times \ln(y_i/\bar{y}) \qquad (2.6)$$

Atkinson Index

This index is used to measure the contribution of observed inequality of outcome and was developed to measure income inequality by Atkinson (1970). However, it has been applied to measure inequality in health and access to health care (e.g., Silber, 1982; Waters, 2000). According to the Atkinson index, the maximum value of 1 indicates the highest inequality, while the minimum value of 0 indicates the highest equality. The formula of the Atkinson index is:

$$I_R = 1 - \left[\sum_{i=1}^{n} \left(\frac{Y_i}{\bar{Y}}\right)^{1-\varepsilon} f_i\right]^{\frac{1}{1-\varepsilon}}, \text{if } \varepsilon \neq 1 \qquad (2.7)$$

$$I_R = 1 - exp\left[\sum_{i=1}^{n} f_i \, log_e \frac{Y_i}{\bar{Y}}\right], \text{if } \varepsilon = 1 \qquad (2.8)$$

Here, in Eq. 2.7 and 2.8, Y_i indicates the level of the outcome (e.g., health or health resources) of the i^{th} individual or i^{th} health resources, and $\bar{Y}$ is the mean of the outcome variable. The Atkinson index calculates inequality for different scenarios' social preferences for equality indicated by the value of epsilon (ε) that integrates with Rawl's idea of social justice (Levy et al., 2006). ε is ordinally equal to $1 - \theta$ where $\theta < 1$. If $\varepsilon > 0$, then there is a higher weight for a social preference for equality. The age of the index value is 0 to 1, where value 0 implies the equal distribution of outcome.

Index of Dissimilarity

The dissimilarity index can be considered an adaptation index of the Lorenz curve and Gini coefficient and has been used to measure health inequality between social groups (Wagstaff et al., 1991). This indicator can particularly be employed to measure the inequality of health resource variables and determine whether there are disparities in the provision of health resources among regions (Yi Tao et al., 2014). The index is defined as

$$ID = \frac{1}{2} \sum_{j=1}^{k} \left|S_{jh} - S_{jp}\right| \qquad (2.9)$$

where, S_{jh} indicates the proportion of outcome variable reflecting the equality in the provision of health resources and S_{jp} is the proportion of the population in the j^{th} region or social class. There is higher inequality in resource allocation when the difference between S_{jh} and S_{jp} is large—that is, if the majority of the population belongs to low and

high socioeconomic groups and there is a small proportion of the population in the middle group, the value of the index of dissimilarity is high.

2.4.3 Joint distribution Inequality Measures

Concentration Index (CI)

CI (Kakwani, 1980; Kakwani et al., 1977) is a bivariate index that has been applied to measure socioeconomic inequalities in health (Wagstaff et al., 1989). In the health inequality literature, CI has been used for different health outcomes such as health care utilisation (Van Doorslaer et al., 2006), child health (Wagstaff, 2000; Wagstaff et al., 2003) and adult health (Van Doorslaer et al., 1997).

CI is defined based on the concentration curve that plots the cumulative population proportion of each socioeconomic group beginning from the most underprivileged against the cumulative proportion of health (for the calculation, people are ranked according to their socioeconomic status from most disadvantaged to most advantaged). The curve overlaps the equality line when the health outcome distributes equally across socioeconomic groups. The CI is twice the concentration curve area that defines the magnitude of health disparities associated with socioeconomic status. Therefore, the formula of the index is:

$$CI = \frac{2 \times Cov(X,H)}{M} \qquad (2.10)$$

$$Cov(X,H) = E(XH) - E(X)E(H) \qquad (2.11)$$

where X and H denote social class with corresponding rank and health variable, respectively, M is the mean of the health variable and $Cov(X, H)$ is the covariance of X and H. The value of the index ranges from −1 to 1. A negative CI value indicates that health resources are disproportionately concentrated on disadvantaged groups. On the contrary, a positive CI value reveals that health resources are unfairly concentrated on well-off social classes.

Although CI is a popular measurement of health inequality related to individuals' socioeconomic position, recent attention has focused on some of its shortcomings. Consequently, researchers have proposed alternatives for CI. For example, Wagstaff (2005) suggested a normalisation formula that deals with the bounds issue of CI. Since the bounds of CI vary with the mean of the health variable, comparing the mean value of

the health outcome between populations is problematic. Clarke et al. (2002) developed a generalised CI to address the issue when inequality in ill health is considered. Moreover, if the health variable is qualitative, the CI value becomes unfairly large (Erreygers, 2006). Considering these drawbacks of the CI, Erreygers (2009) proposed a corrected concentration index.

Regression-Based Inequality Measures

1. Relative and Slope Inequality Indices (RII & SII)

These two indices are based on a regression between health outcomes and the relative position of social groups and are used to monitor health policies and compare the health status of individuals who experience different socioeconomic, environmental, and geographic conditions.

SII represents the linear regression coefficient that shows the relationships between the health status of a class and its relative rank in the socioeconomic distribution. Therefore, in this case, the regression slope is interpreted as the absolute effect of moving the lowest class through to the highest on health (Wagstaff et al., 1991). In this case, a socioeconomic variable is constructed with ordinal categories. This SII is specified as

$$SII_1 = h(1) - h(0) \qquad (2.12)$$

where *h(1)* and *h(0)* denote the health status in the lowest and the highest socioeconomic categories, respectively. In this case, however, the regression equation is heteroskedastic due to the use of categorical variables. There is also some variation in the index estimation with different regressions (Moreno-Betancur et al., 2015). Therefore, ordinary least squares (OLS) estimation is insufficient but not biased. An econometric solution, in this case, is to use weighted OLS (Cheng et al., 2008; Renard et al., 2019; Wagstaff et al., 1991). The SII based on weighted OLS is given by:

$$SII = \frac{\sum_{i=1}^{n} w_i(y_i - \bar{y}_w)(x_i - \bar{x}_w)}{\sum_{i=1}^{n} w_i(x_i - \bar{x}_w)^2} \qquad (2.13)$$

where x_i and y_i denote the midpoint of the class range and health outcome, w_i is the frequency of each class, and *i,,* $\bar{x}_w$ and $\bar{y}_w$ are the frequency-weighted averages of x_i and y_i.

RII is estimated by extrapolating the regression on the extreme position of the x-axis: 0 and 1. Therefore, RII is the proportion of the lowest class *h(1)* value from the value of the highest social class *h(0)*. RII is defined by:

$$RII = \frac{h(1)}{h(0)} = \frac{\hat{\alpha}}{\hat{\alpha} + \hat{\beta}} \qquad (2.14)$$

where $\hat{\alpha}$ and $\hat{\beta}$ are the intercept and the slope of the estimated regression (see Cheng et al., 2008; Moreno-Betancur et al., 2015; Renard et al., 2019; Wagstaff et al., 1991).

2. Additively Decomposable Index

This index is calculated based on the variance of outcomes once the models are fitted. This measure is proportional to the squared coefficient of variation, which is a member of the additively decomposable index below.

$$I_\alpha(y) = \frac{1}{N\alpha(\alpha - 1)} \sum_{i=1}^{N} \left[\left(\frac{y_i}{\hat{y}} \right)^\alpha - 1 \right] \qquad (2.15)$$

Here $I_\alpha(y)$ is the health inequality index and α a weighting parameter, which sets the index equal to half the squared coefficient of variation when $\alpha = 2$. We can model the fraction of total inequality explained by our model covariates using the ratio $\frac{I_\alpha(\hat{y})}{I_\alpha(y)}$. The advantage of this measure is that this ratio is equal to the R^2 term from a regression model used to estimate $\hat{y}$.

Ordinal Measures

The frequency ratio has been used to measure health inequality when a health event is binary. When socioeconomic variables are categorical—for example, polytomous or dichotomous—the frequency of health events in each category is compared to the reference category. The ratios are calculated using a contingency table or log-linear regression model (Krieger, 2002; Mackenbach et al., 1997). However, if the socioeconomic variable is continuous, log-linear regression is most probably be used and, therefore, the estimated inequality measure is the frequency ratio associated with one unit change in the independent variable (Regidor et al., 2003). Moreover, when the frequencies of the events are very small, the odds ratio is used as an alternative to the frequency ratio. The odds ratio can be calculated using a contingency table or logit

regression model. Although there are no significant differences between the odds ratio and frequency ratio, the odds ratio overestimates the association between outcome and independent variable when the frequency of the outcome is higher than 20% (Clayton & Hills, 1993).

2.5 Inequality of Opportunity (IOP)

Based on recent development in the social choice and political philosophy literature (Arneson, 1889; Cohen, 1989; Rawls, 1971; Roemer, 1998, 2002), the economic view of inequality conceives responsibility through the lens of distributive justice. Rawls' (1999) pioneering idea of equality concludes that if resources and responsibilities distribute equally across social classes or individuals, existing inequality is considered a legitimate consequence of individuals' selections. By initiating the concept of IOP, Roemer (1998) divided the inequality of individual outcomes (e.g., income, health) into two sources. First, inequalities related to predetermined factors beyond an individual's control are acknowledged as IOP, which is considered unacceptable. These inherited factors of unacceptable inequalities are known as *circumstances.* In contrast, inequalities due to an individual's choices are considered acceptable or legitimate because individuals should be responsible for their selections; these are called *efforts.* Although circumstances and effort are distinguished as different sources of inequality, the efforts can be a function of circumstances. Roemer's normative framework of IOP developed following Dworkin's (1981a, 1981b) explanations regarding the individual's preference and available resources. Dworkin (1981a, 1981b) claimed that ethically acceptable equality can be achieved by distributing resource quality across individuals, but those disparities due to personal choice following differential preferences should be allowed. Therefore, he held that individuals should be responsible for their preferences but not for the availability of resources.

Researchers disagree about which factors are considered to be *circumstances* and *effort*. However, most of the empirical studies on IOP in health include social and family background characteristics as circumstances because individuals cannot control the chance of birth. For example, children cannot be held responsible for parents' SES factors such as level of education, income, occupation, lifestyle, attitudes and genetic inheritance. On the contrary, effort factors are challenging to observe and measure, but it is possible that, for health outcomes, individuals' lifestyle behaviours (such as smoking or alcohol consumption, diet, and physical activities) can be used as effort. However, in this respect,

Arneson (1989) argues the importance of considering individual ages because they cannot have independent lifestyle preferences before a certain age.

According to the theory, two basic principles should be maintained to obtain equal opportunity for individual outcomes (Fleurbaey, 2008). Firstly, the compensation principle suggests eliminating IOP or compensating individuals who suffer from inherited circumstances. Secondly, liberal-rewards demand rewards for individual efforts. However, there is disagreement about separating the contributions of circumstances and efforts in practice. For example, Barry (2005) and Roemer (1998, 2002) initially disputed the correlation between efforts and circumstances regarding rewording education. In considering Asian students, Roemer (1998) claimed that, since education achievements depend on parental influences beyond students' control, extra education efforts should not be reworded. He argues that students perform well in the classroom because, in the Asian family culture, parents push children to do educational activities.

In contrast, Barry (2005) supported the idea of Yellen (1984), which justified rewarding the extra effort of a student to achieve a high level of education. Recently, by examining the alternative specifications of legitimate and illegitimate disparities, the influential work of Jusot et al. (2013) suggests an approach to identifying the partial contribution of circumstances and effort. We explain this method of decomposition in Chapter Three.

2.5.1 Measure IOP

For formulating the compensating mechanism, the *ex-ante* and the *ex-post* approaches were proposed (Fleurbaey & Peragine, 2013; Li Donni et al., 2014; Ramos & Van de gaer, 2016; Roemer & Trannoy, 2015). The *ex-post* approach considers the disparities in the outcome variable between individuals who experience similar responsibilities; IOP can be achieved if those individuals obtain the same outcome. On the other hand, the *ex-ante* approach holds that if people have an equal chance to experience an available set of opportunities, there is no IOP apart from their circumstances.

Applying the *ex-post* approach to practical scenarios is challenging because it requires observable effort factors that are considered unobservable by nature. Therefore, researchers should provide a valid justification for selecting proxies for effort. In contrast, the *ex-ante* approach needs observations only for circumstances and allows the measurement of IOP using a limited set of these. According to (Fleurbaey & Peragine, 2013), the *ex-post* and *ex-ante* approaches are incompatible with the compensation

principle. For example, the *ex-post* approach is inconsistent with the time individuals expend on effort, but the *ex-ante* approach is consistent with this. However, data availability and ethical justification are the main concerns in selecting these two approaches.

According to the IOP framework, the standard production function of individual outcomes (Y) was specified as $Y\{C, E(C)\}$, where C represents circumstances and E represents effort. The empirical literature has used both parametric and nonparametric methods to measure IOP. The studies based on Parametric methods follow the *ex-ante* approach to estimate the reduced-form IOP model to measure the variation in outcomes (e.g., health) associated with exogenous circumstances (Ferreira & Gignoux, 2011; Trannoy et al., 2010). In contrast, the IOP model based on the *ex-post* approach considers normative position explanations about the correlation between circumstances and effort (e.g., Asada et al., 2014, 2015; Bricard et al., 2013; Carrieri & Jones, 2018; Deutsch et al., 2018; Li Donni et al., 2014; Rosa Dias, 2010). Furthermore, the outcome variable is regressed using the observed circumstance and effort variables. Although observing effort is problematic, in this case, individuals' lifestyle behaviours such as diet, physical activities, smoking and alcohol consumption have been used as proxies for effort, particularly for moderating health outcomes (Bricard et al., 2013; Carrieri and Jones, 2018; Jusot et al., 2013; Rosa Dias, 2010).

Lefranc et al. (2009) introduced a nonparametric methodology that considers the commutative distribution of outcome variables conditional to types of circumstances and groups with the same efforts to understand IOP in income. Using this approach, some empirical studies (Carrieri & Jones, 2018; Gigliarano & D'Ambrosio, 2013; A. M. Jones et al., 2012, 2014; Rosa Dias, 2009) have estimated IOP in health.

Recent studies (Brunori et al., 2013b; Fleurbaey & Schokkaert, 2011; Ramos & Van de gaer, 2016) have proposed direct and indirect measures of IOP. The direct measures consider IOP that is due to circumstances residues. For example, based on the *ex-ante* compensation approach, Fleurbaey & Schokkaert (2009) have proposed a direct IOP measure that assesses IOP under the condition that "all people utilise the same level of effort". Nonetheless, indirect methods measure the inequality that remains after opportunities are utilised. In this case, there is a condition that all individuals experience the same set of circumstances and estimate IOP to compare disparity in actual outcome distribution and counterfactual outcome distribution. The indirect approach quantifies the

fairness gap (Fleurbaey & Schokkaert, 2009) between observed outcome and outcome, consisting of the assumptions of the *ex-post* approach regarding the circumstance. Moreover, considering Roemer's (1998), Barry's (2005), and Swift's (2005) alternative normative viewpoints on the correlation between circumstances and effort, Jusot et al. (2013) have provided an econometric framework for evaluating the importance of attributions to circumstances. More information about these measures can be found in Chapter Three.

Chapter 3

The Intergenerational Effects of Socioeconomic Inequality on Unhealthy Bodyweight

3.1 Introduction

Large socioeconomic gradients in health outcomes represent a significant social issue in developed countries. For example, richer and better-educated individuals routinely report better health outcomes than those who are poorer, a result that persists over a wide variety of indicators, including longevity (Clarkwest, 2008; Kaplan et al., 2015), mortality (Petrie et al., 2011), mental and physical health aggregates (Rohde et al., 2017; Wang & Geng, 2019; Watson & Osberg, 2017) and other measures such as subjective self-assessments (Brunello et al., 2016; Ichida et al., 2009). Similarly, empirical evidence suggests that poorer individuals are more likely to smoke (Pisinger et al., 2011), be diagnosed with cancer (Gallaway et al., 2019), suffer from depression (Phongsavan et al., 2006) and even commit suicide (Hajizadeh et al., 2019; Vandoros et al., 2019). Findings such as these are highly robust—they tend to hold in both micro-data and at the macro level, and show no sign of diminishing over time (see Cairns et al., 2017; Gravelle et al., 2002; Lago et al., 2018; Subramanian and Kawachi, 2004).

Correlations between economic wellbeing and health exist for a wide variety of reasons. Most explanations can be traced back to variations in the underlying drivers of health, which include an individual's genetics, family background, diet, lifestyle choices, attitudes, behaviour and educational level (Fleurbaey & Schokkaert, 2009; Roemer & Trannoy, 2016). The sheer heterogeneity of such determinants and the complex web of causal flows that map from determinants to outcomes means that researchers often struggle to identify the true underlying drivers of economic disparities in health.

Despite this apparent complexity, recent innovations in the study of economic inequality offer a path forward for disentangling various sources of disparity. Indeed, not all factors driving inequality are equally objectionable, and for policy making, it is desirable to identify and combat the most harmful underlying forms. Following the work of Dworkin (1981a, 1981b), Roemer (1998) formulated the inequality of opportunity (IOP) conceptual framework, which identifies harmful inequalities by stratifying variations in outcomes into two forms: circumstances (factors that lie beyond personal control) and efforts (factors for which individuals are responsible). Circumstances are typically

inherited—occurring at birth or during childhood, and therefore predetermined with respect to adult health—and include factors such as race, gender and social class. In the context of health, the effort is proxied by personal behaviours, such as dietary choices, drug and alcohol consumption, and physical activity.

Decomposing inequality into contributions from circumstances and effort is conceptually difficult. For instance, some important circumstances (such as genetic endowment) are not normally directly observable, while in other cases, judgment is required in classifying variables as legitimate or illegitimate sources of variation.[3] While factors such as parental education are commonly treated as circumstances, others such as childhood experience occupy a grey area.[4]

Furthermore, the effort is not easily established due to the complex nature of identifying freely made a personal choice. For this reason, it is more common for IOP studies to focus on inequalities induced by background characteristics, which leave the residual (unexplained) component to reflect both unobserved circumstances and efforts. The fact that circumstances themselves are only partially observed means that this approach provides a lower representation of true IOP.

In this study, we apply IOP concepts to identify harmful sources of disparity in the bodyweight of Australians. Obesity (and its variants) makes an interesting case study for IOP analysis as, relative to other health conditions, it is highly reflective of lifestyle choices that can be plausibly classed as effort. As such, we expect to see much lower estimates than those produced for summary variables, such as self-rated health (Li Donni et al., 2014; Rosa Dias, 2009, 2010). Furthermore, bodyweight itself is an input into a spectrum of health conditions, including some (such as accidents and injuries) that are not commonly linked with health behaviours (Koepp et al., 2015). IOP in unhealthy weight is therefore likely to produce IOP for a broad range of other outcomes (such as heart disease, cancer and diabetes; see Pi-Sunyer, 2009), including those directly related to well-being such as self-assessed happiness (Habibov et al., 2019).

Using regression models applied to microdata obtained from the Household, Income and Labour Dynamics in Australia (HILDA) panel, we determine the effects of inherited

[3] Roemer (2018) recommends that this choice be guided by prevailing social norms.

[4] Environmental factors experienced during childhood can be regarded as circumstances using a subjectively-defined age-of-responsibility concept (Arneson, 1990) where children are not held responsible for events experienced before some age threshold, such as 18 or 21.

factors on (i) body mass index (BMI) and (ii) a measure of overweightness (defined as the excess positive distance an individual is from the 18.5–25 healthy range). Our results suggest that an individual's observable set of circumstances plays a relatively small role in determining body composition in adulthood, with estimates indicating that only around 4% of the inequality in BMI and unhealthy bodyweight can be attributed to a standard battery of inherited socioeconomic characteristics. Nonetheless, we do uncover a tendency for persons born into favourable economic conditions to have significantly healthier weight, with the prime determinant being the socioeconomic status (SES) of the individual's father. Results such as these are important for allocating healthcare resources because they provide a basis for identifying individuals who (i) may suffer from lifestyle-related illnesses but (ii) are relatively disadvantaged by circumstances and, *ceteris paribus*, can be considered less responsible for their health conditions.

After estimating our IOP models, we then turn our attention to determining how the correlations between an individual's circumstances and their weight arise. By sourcing extra data on health behaviours—including diet, exercise, and alcohol and tobacco consumption—we study whether those born into more advantageous backgrounds are healthier because they develop better habits or, conversely, if other factors omitted from our models account for the results. Our models show mixed evidence that an individual's background prompts healthy behaviours. Individuals with higher SES parents are more likely to eat vegetables and engage in physical exercise but are also more likely to consume alcohol. Accounting for these factors only partially attenuates our results, which implies that other omitted factors (such as unobserved circumstances or behaviours, or genetic factors correlated with birth characteristics) also play a role. We also present a decomposition using the approach of Jusot et al. (2013), which examines the effect of the correlation between circumstances and behaviours in determining inequality. Like these authors, we find that the tendency for individuals from advantaged backgrounds to engage in healthier behaviours has relatively small quantitative effects.

Our study is one in a recently developing literature that examines IOP in health. Out of these empirical investigations, most (Bricard et al., 2013; Jusot et al., 2013; Rosa Dias, 2009b; Trannoy et al., 2010) have modelled self-assessed health as the outcome variable. These studies often produce estimates of IOP in the range of 20%–30% of explained inequality. Other studies (Carrieri & Jones, 2018; Rosa Dias, 2010) use various biomarkers of diseases, chronic illness and disability (including mental health conditions) to tackle heterogeneous patterns in health outcomes and again produce relatively high

values for IOP. As with our study, these studies focus mainly on parental SES and family background as measures of circumstances and use lifestyle behaviours as proxies for effort (Bricard et al., 2013; Carrieri and Jones, 2018; Jusot et al., 2013; Rosa Dias, 2010). Nevertheless, despite the literature on the impact of SES on an individual's BMI (Ball & Crawford, 2005; Devaux & Sassi, 2013; McLaren & Godley, 2009; Rauschenbach et al., 1995; van Lenthe et al., 2000; Watson, 2018) and the effect of lifestyle behaviours on BMI (Kontogianni et al., 2010; Sundquist & Johansson, 1998), we are unaware of any studies that use BMI or unhealthy bodyweight as outcome variables to study IOP in health.

The structure of the chapter is as follows. The following section outlines the data set and describes the key variables. Section 3.3 estimates some baseline models that allow us to examine IOP in BMI and unhealthy bodyweight, while Section 3.4 presents a decomposition showing the effect of correlations between circumstances and behaviours. Section 3.5 considers the implications of our results for prioritizing treatment for lifestyle-related diseases when resources for healthcare are limited. A summary and conclusion are presented in the last section.

3.2 Data

Data come from the HILDA survey, which is an approximately nationally representative panel, similar in design to the US-based Panel Study of Income Dynamics and the German Socioeconomic Panel (*Sozio-oekonomisches Panel*). HILDA contains an extensive catalogue of both ancestral variables (which we will use as circumstances) and data on a wide variety of health behaviours. The survey comprises more than 7,000 Australian households (more than 9,000 are included in Wave 11) and follows approximately 200,000 individuals. We limit our analysis to the 2007, 2009 and 2013 survey years due to certain questions regarding Australians' health behaviours being included only for those waves.

3.2.1 Health Variables

We employ two biometric measures of physical health. The first is the standard individual-level BMI score, defined as the ratio of weight in kilograms divided by the square of height in meters. According to the standard classification, the normal weight category (18.5–24.9) is healthy, while the others (underweight = < 18.5; pre-obesity = 25–29.9; obesity class I = 30–34.9; obesity class II = 35–39.9; obesity class III = > 40)

indicate risk categories (World Health Organization, 2000). BMI has some well-known drawbacks as a health indicator—for example, it does not differentiate between fat and muscle mass, it is sensitive to subcutaneous fat (rather than more harmful visceral fat), and it differs across gender and ethnicity in its relationship with diseases such as diabetes (Bhurosy & Jeewon, 2013). Nonetheless, it is a suitable indicator over a large sample (as most idiosyncrasies can be averaged out) and has the advantages of being systematically measured and widely understood (Nuttall, 2015; Stommel & Schoenborn, 2009).

The second variable is derived from the underlying BMI score and is designed to deal with the nonmonotonicities associated with BMI and general health (Apablaza et al., 2016). The primary health concerns associated with BMI occur in the very high range; therefore, changes in weight become less relevant closer to the healthy weight range (Berrington de Gonzalez et al., 2010; Flegal et al., 2013). We, therefore, define a second measure for overweightness (*BMI**), which is the positive distance from the upper bound (25) of the healthy BMI level.

$$BMI^* = \begin{cases} 0 & if\ BMI > 25 \\ BMI - 25 & If\ BMI \leq 25 \end{cases} \qquad (3.1)$$

3.2.2 Circumstance Variables

We source a range of socioeconomic variables designed to capture the economic conditions experienced by individuals at birth or in early life. While it is not possible to completely measure every aspect of a child's environment, HILDA contains a wide variety of suitable indicators. Larger and richer data sets have a greater capacity for identifying inequalities, so we selected a total of 27 variables to capture individuals' initial conditions.[5] Given our focus on socioeconomics, we do not use parental biometric data. Parental SES is measured using the schooling completion of both mother and father, indicators of higher educational attainments, and synthetic markers of occupational prestige (see McMillan et al., 2009). These variables are 0–100 scales based on an aggregation of underlying socioeconomic variables (such as education, income, and job type) where higher numbers indicate greater status.[6] Other markers capture parental divorce during childhood, loss of a parent and non-biological parentage. Following

[5] Data quality is important as the partial observability of circumstances results in smaller estimates for IOP. Some strategies for ameliorating this involve defining inequalities (the so-called *ex post* approach) or assuming that all time-invariant phenomena are circumstances, which can be used in panel data to bound IOP estimates from the opposite direction (Niehues & Peichl, 2014).

[6] See Figure A3.1 in the appendix for detail on these variables.

Bourguignon et al. (2007), Ferreira & Gignoux (2011) and Checchi et al. (2010), we also use race and gender as circumstances, although we note that some authors prefer to employ these as intermediate controls. Other circumstances include whether English was the first language learned, whether Australia was the country of birth, and immigration and refugee status. We also control for birth order using a dummy for being the oldest child when growing up.

3.2.3 Health Behaviours

To measure the lifestyle choices that drive body fat content, we draw on exercise data, dietary habits and smoking and drinking frequencies. We include the frequency of eating vegetables, foods with more carbohydrates, snack foods, pub foods (fried potatoes, French fries, hot chips or wedges), red meat and processed meat products. Those food frequencies are split into ordinal categories that refer to the number of times the food is consumed per week, where the highest category is more than seven times. Similar scales represent physical activity, and we have dummies for smoking (where the reference group had either never smoked or no longer smoked) and drinking alcohol (where the reference never drank alcohol). As with all self-assessed data, there is likely to be some heterogeneity in response, although this should not affect our estimates provided it is uncorrelated with our explanatory variables.

3.2.4 Other Control Variables

We also employ some demographic characteristics of respondents that may directly or indirectly correlate with their body weight, including age, age squared, marital status and employment status. Since there are small differences between BMI scores in regions of different population densities, we also employ dummies for those residing in cities or other urban areas. In some instances, these variables themselves may be seen as health behaviours and may be partially determined by circumstances, although auxiliary regressions reveal that their empirical links with the background characteristics are generally very weak. The main exception here is age, which we include to adjust for lifecycle inequalities and do not interpret as a driver of illegitimate inequality.

3.2.5 Descriptive Statistics

Table A3.1 (in the appendix) presents some descriptive statistics of our sample. We limited our sample to individuals over 18 to remove idiosyncrasies associated with weight while maturing, and we exclude women who gave birth in the subsequent year to avoid

distortions due to pregnancy. The mean BMI of our sample is 26.71, which is notably above the overweight threshold of 25. Approximately 59% of respondents lie outside the healthy bodyweight range of 18.5–25, with 2% below 18.5 and 57% above 25. In terms of demographics, just over half of our sample are female (52%). Furthermore, a higher proportion of the respondents were born in Australia (80%) and learned English as their first language (92%), whereas only 2% are refugees and indigenous. In addition, most of the sample are legally married or in de facto relationships (68%). These figures concur approximately with Australian national averages (e.g., according to the Australian Bureau of Statistics (2018a, 2018b): the sex ratio is 98.4 males per 100 females, 67% of those aged 18 years or over are overweight or obese, while 1.3% are underweight).

3.3 Inequality of Opportunity in Lifestyle Health Diseases

3.3.1 Empirical Approach

We begin by specifying three models that link bodyweight with inherited socioeconomic characteristics. We firstly employ a reduced-form IOP model to measure the impact of only the observable circumstances on BMI and BMI*. Secondly, we include some demographic control variables such as age, marital status, employment status and living area. Finally, since individuals' health behaviours are correlated with their circumstances, we introduce additional health behaviour variables to determine if they mediate the empirical links with outcomes. This approach allows us to test pathway and latency hypotheses on the intergenerational transmission channels[7].

The models are given below and are fitted using ordinary least squares (OLS). We use heteroskedasticity-robust standard errors throughout.

$$H_i = \alpha + \sum_{j=1}^{k} \beta_{1j} C_{ij} + \varepsilon_i \qquad (3.2)$$

$$H_i = \alpha + \sum_{j=1}^{k} \beta_{2j} C_{ij} + \sum_{l=1}^{m} \gamma_{2j} D_{il} + \varepsilon_i \qquad (3.3)$$

[7] Modeling the conditional mean of these variables implies that very low BMI scores (associated with 2.3% of underweight individuals in Table A3.1) may plausibly offset very high values; however, we observe that excluding these values (or defining variables that are U-shaped transformations of BMI to capture non-monotonicity) produce very similar results.

$$H_i = \alpha + \sum_{j=1}^{k} \beta_{3j} C_{ij} + \sum_{l=1}^{m} \gamma_{3l} D_{il} + \sum_{p=1}^{q} \theta_{3p} B_{ip} + \varepsilon_i \qquad (3.4)$$

Here, $H_i = BMI_i;\ BMI_i^*$ is the health outcome of the individual $i; C_j;\ \forall j \in (1, \dots, k)\ i$; represents individual specific circumstances; $D_i;\ \forall l \in (1, \dots, m)$ represents demographic characteristics; and $B_p;\ \forall p \in (1, \dots, q)$ represents health behaviours. β, γ and θ are parameters to be estimated, where subscripts 1 Equations (3.2–3.4) 3 denote Models 1–3, and *j*, land p index the specific parameter. Here, α represents an intercept and ε_i represents the error term. Estimates of Eq. (3.2–3.4) are run for each year (i.e., over the waves for the 2007, 2009 and 2013 surveys); the results are presented in Table 3.1 (for BMI) and Table 3.2 (for BMI*) below.

3.3.2 Do Circumstances Matter?

To examine the role of circumstances in determining adult health, we look at estimates for Eq. (3.2) and (3.3) across our two indicators and our three waves of data. Table 3.1 shows that our observed circumstances explain approximately 3%–4% of the total inequality in BMI (e.g., Table 3.1 shows that R^2 values of Eq. (3.2) for 2007, 2009 and 2013 data are 0.030, 0.032 and 0.043, respectively), and similarly small values for the variation in BMI* (see the R^2 values of Eq. (3.2) in Table 3.2). These proportions of variation captured by the circumstances do not vary substantially over time (indicating that our estimates are robust to the choice of the year), and there are only slight differences between the two markers of individual health. Moreover, F-statistics show that our estimates of Model (1) over all three waves are highly significant overall. Thus, we can reject a null hypothesis of no links between an individual's circumstances and their body weight at all levels in all years.

Nonetheless, the estimates presented are fairly small, indicating that there are only limited intergenerational effects, as captured by our parental socioeconomic variables. This suggests that predetermined inequalities in bodyweight may be much lower than those reported for other health outcomes, such as the 20%–30% often found for self-rated health (Rosa Dias, 2009b) or the 50%+ found in cholesterol and glycated haemoglobin by Carrieri & Jone (2018). Nonetheless, our smaller estimates are still quantitatively meaningful and are in line with theory for several reasons. As outlined above, bodyweight is likely to be unusually responsive to behaviours and, hence, the share explained by circumstances will be relatively small. If we replace our dependent variables with {1–10}

self-assessed health scores, we get higher estimates ranging from 0.04 to 0.22 (Table A3.4 in the appendix) which, in some instances, are almost double those reported in Tables 3.1 and A3.2.[8]

Furthermore, our covariates capture predetermined socioeconomic inequalities but do not measure direct physiological transmission, such as via parent-level biometric circumstances. This is appropriate for understanding the links between economic inequality and health but will result in smaller estimates than models that include detailed biological data.

The methodology also plays a role. Our decomposition approach is based upon the variance, which is desirable because the total inequality can be written as the sum of explained and unexplained terms. This produces much lower estimates than the Gini coefficient (Brunori, 2016), where explained and unexplained inequalities will not equal the total. However, if the ratio of the Gini coefficient of fitted values to the overall Gini is calculated, we obtain estimates of approximately 20%–22% of inequality attributable only to circumstances.[9]

The unexplained component in these models (which will reflect unobserved efforts and circumstances) is of substantial interest and is unlikely to be attributable purely to unobserved efforts. For example, BMI is known to be strongly comprised of genetic factors, which have been shown to account for approximately 40% of the variation (Classen & Thompson, 2016; Moll et al., 1991). Similarly, Evans et al. (2016) show that social environments (which are also unobserved) contribute almost half of the variation of adolescents' BMI. Other authors have shown generally weak links between individuals' SES and bodyweight in other contexts (Rauschenbach et al., 1995; van Lenthe et al., 2000), which may also be consistent with our small intergenerational effects.

[8] The values are {0.040, 0.111, 0.208; 0.040, 0.108, 0.208; 0.050, 0.116, 0.220} for Models 1–3 across our three waves (Table A3.4).
[9] Note that these estimates are only available for BMI. Variable BMI* has negative fitted values which are incompatible with Gini coefficients.

Table 3.1 Association between Circumstances, Health Behaviour and Body Mass Index

Variable		2007			2009			2013		
		M(1)	M(2)	M(3)	M(1)	M(2)	M(3)	M(1)	M(2)	M(3)
Circ	Female	-0.518***	0.564***	-0.420***	-0.236*	-0.327***	-0.155	-0.396***	-0.487***	-0.295***
	Non-biol mother	0.825	0.56	0.529	1.325**	0.962*	0.847	0.974**	0.541	0.465
	Non-biol father	0.119	0.25	0.272	-0.026	0.039	0.087	0.129	0.163	0.132
	Indigenous	1.929	2.309	1.995	2.612	2.649*	2.506	2.570**	2.831***	2.524**
	Parents divorced	0.409*	0.253	0.327	0.552**	0.368	0.410*	0.330*	0.096	0.086
	Refugee	0.249	0.122	-0.104	0.5	0.42	0.45	0.496	0.283	0.307
	Oldest child	0.001	0.063	0.046	-0.028	0.063	0.078	0.178	0.218*	0.264**
	Father immigrated	-0.123	-0.129	-0.103	-0.181	-0.152	-0.122	-0.109	-0.031	0.014
	Mother immigrated	-0.211	-0.085	-0.02	-0.416**	-0.337	-0.266	-0.601***	-0.527***	-0.481***
	Born overseas	-0.001	-0.542**	-0.447**	0.425	-0.124	-0.014	0.562***	-0.047	0.027
	Non-native English	-0.471	-0.387	-0.351	-0.778**	-0.710**	-0.728**	-0.913***	-0.762***	-0.774***
	Father employ	-0.627	-0.025	-0.056	-0.078	0.008	-0.002	0.278	0.058	0.078
	Mother employ	-0.015	0.218	0.235*	-0.122	0.121	0.166	-0.156	0.087	0.131
	Father died	0.157	0.29	0.184	0.247	0.336	0.526	0.226	0.25	0.412
	Mother died	0.157	0.29	0.184	0.247	0.336	0.526	0.226	0.25	0.412
	Father prim ED	-0.019	-0.217	-0.061	-0.957	-1.005	-1.155	1.106*	0.812	0.984*
	Father some HS	-2.460**	-2.240*	-1.937	-2.342***	-2.016**	-2.079***	-0.599	-0.37	-0.256
	Father Non-U T	-0.211	-0.002	0.077	-0.116	0.076	0.167	-0.510***	-0.221	-0.205
	Father university	-0.677***	-0.309	-0.215	-0.523**	-0.161	-0.015	-0.722***	-0.337*	-0.198
	Mother prim ED	3.570*	3.206	2.962	0.87	0.823	0.886	-0.367	-0.383	-0.71
	Mother some HS	1.035	1.225	1.076	1.122	1.163	1.274*	-0.215	-0.192	-0.34
	Mother full HS	1.676	1.951	1.845	2.016**	2.204***	2.303***	-0.173	0.158	0.016
	Mother non-U T	-0.633***	-0.349**	-0.251	-0.660***	-0.306	-0.213	-0.484***	-0.071	-0.004
	Mother university	-0.51**	0.12	0.226	-0.860***	-0.152	-0.096	-0.573***	0.225	0.285
	Father occ stat	-0.010***	-0.013***	-0.009***	-0.013***	-0.015***	-0.011***	-0.013***	-0.016***	-0.012***
	Mother occ stat	-0.010***	-0.007**	-0.005	-0.011***	-0.009**	-0.006*	-0.014***	-0.012***	-0.008***

Table 3.1 (Continued)

	Variable	2007			2009			2013		
		M(1)	M(2)	M(3)	M(1)	M(2)	M(3)	M(1)	M(2)	M(3)
Contr	Age	-	0.0297***	0.320***	-	0.308***	0.330***	-	0.346***	0.365***
	Age squared	-	-0.003***	-0.003***	-	-0.003***	-0.003***	-	-0.003***	-0.003***
	Employed	-	-0.357*	-0.289	-	-0.461**	-0.358*	-	-0.535***	-0.362**
	Unemployed	-	0.121	0.219	-	-0.203	-0.09	-	0.359	0.514
	Marred/ De-facto	-	0.183	0.069	-	0.022	-0.071	-	-0.121	-0.264**
	City	-	-0.049	-0.068	-	0.203	0.143	-	0.327**	0.262**
	Regional area	-	0.29	0.232	-	0.265	0.176	-	0.407**	0.266
Behav	Smoker	-	-	-0.930***	-	-	-0.668***	-	-	-0.716***
	Alcohol	-	-	-0.274	-	-	-0.509**	-	-	-0.365**
	Vegetables	-	-	-0.109**	-	-	-0.095**	-	-	-0.150***
	Carbohydrate	-	-	-0.305***	-	-	-0.400***	-	-	-0.278***
	Snack food	-	-	0.233***	-	-	0.137**	-	-	0.238***
	Fried food	-	-	0.233***	-	-	0.137**	-	-	0.238***
	Red meat	-	-	0.313***	-	-	0.246***	-	-	0.450***
	Processed food	-	-	0.183***	-	-	0.277***	-	-	0.157***
	Exercise freq	-	-	-0.412***	-	-	-0.422***	-	-	-0.508***
Aux	Constant	28.026***	20.832***	21.568***	28.358***	21.074***	22.069***	28.739***	20.632***	20.821***
	R^2	0.030	0.065	0.101	0.032	0.068	0.105	0.043	0.087	0.131
	F	8.627	16.036	18.917	9.816	17.118	19.812	18.339	31.114	35.366
	N	6701	6701	6701	6962	6962	6962	9687	9687	9687

Notes: The table presents estimates of Models 1–3 from Equations (3.2–3.4) with BMI as the dependent variable. Model 1 contains only circumstance variables while Model 2 includes demographic controls. Model 3 further adds behavioural variables. Parameters are estimated using OLS with heteroskedasticity-robust standard errors throughout. Dummies are defined with respect to a reference individual who is an unmarried male, non-smoker and non-drinker and lives in a remote area. Parental educational attainments are none/unknown. *, ** and *** define significance at 10%, 5% and 1%, respectively.

Table 3.2 Association between Circumstances, Health Behaviour, and Overweightness

	Variable	2007			2009			2013		
		M(1)	M(2)	M(3)	M(1)	M(2)	M(3)	M(1)	M(2)	M(3)
Circ	Female	0.078	0.025	0.119	0.348***	0.265***	0.383***	0.272***	0.178**	0.315***
	Non-biol mother	0.616	0.459	0.428	1.091**	0.869*	0.768*	0.680*	0.41	0.349
	Non-biol father	0.129	0.154	0.159	-0.01	-0.032	-0.007	0.229	0.187	0.152
	Indigenous	2.010*	2.157*	1.945	2.427*	2.362*	2.240*	2.385**	2.456***	2.229**
	Parents divorced	0.371**	0.26	0.308*	0.453**	0.330*	0.268**	0.313*	0.161	0.151
	Refugee	-0.035	-0.08	-0.229	0.041	0.029	0.062	0.333	0.239	0.27
	Oldest child	-0.031	0.009	0.001	-0.061	0	0.017	0.058	0.095	0.136
	Father immigrated	-0.054	0.065	-0.051	-0.118	-0.114	-0.103	-0.06	-0.023	0.005
	Mother immigrated	-0.113	-0.042	0.002	-0.311**	-0.266*	-0.208	-0.446***	-0.415***	-0.379***
	Born overseas	-0.104	-0.384**	-0.314*	0.245	-0.03	0.047	0.257	-0.056	-0.009
	Non-native English	-0.19	-0.17	-0.173	-0.373	-0.369	-0.416*	-0.500**	-0.435**	-0.502**
	Father employ	-0.079	0.067	0.034	-0.041	-0.005	-0.021	0.267	0.136	0.142
	Mother employ	0.049	0.155	0.166	-0.05	0.057	0.088	-0.064	0.051	0.085
	Father died	0.211	0.11	0.083	0.074	0.007	-0.02	0.308	0.202	0.121
	Mother died	0.002	0.111	0.055	0.033	0.13	0.286	0.186	0.204	0.329
	Father prim ed	0.547	0.418	0.532	-0.734	-0.719	-0.845	0.815	0.628	0.769
	Father some HS	-0.681	-0.512	-0.354	-0.456	-0.205	-0.147	-0.018	0.089	0.194
	Father Non-U T	-0.254**	-0.145	-0.085	-0.165	-0.072	-0.003	-0.401***	-0.424**	-0.227**
	Father university	-0.424**	-0.219	-0.144	-0.352**	-0.167	-0.043	-0.499***	-0.305**	-0.187
	Mother prim ed	2.37	2.143	1.989	0.562	0.522	0.569	-0.307	-0.292	-0.556
	Mother some HS	0.467	0.613	0.518	0.599	0.624	0.681	-0.055	-0.075	-0.197
	Mother full HS	0.897	1.078	1.012	1.398**	1.492**	1.528**	0.038	0.203	0.085
	Mother Non-U T	-0.455***	-0.304**	-0.224*	-0.408***	-0.225	-0.15	-0.329***	-0.089	-0.032
	Mother university	-0.342*	0.021	0.094	-0.530***	-0.168	-0.124	-0.350**	0.101	0.147
	Father occ stat	-0.007***	-0.009***	-0.006**	-0.010***	-0.011***	-0.008***	-0.010***	-0.011***	-0.008***
	Mother occ stat	-0.008***	-0.006**	-0.004	-0.008***	-0.006**	-0.004	-0.010***	-0.009***	-0.006***

Table 3.2 (Continued)

	Variable	2007			2009			2013		
		M(1)	M(2)	M(3)	M(1)	M(2)	M(3)	M(1)	M(2)	M(3)
Cont	Age	-	0.194***	0.210***	-	0.202***	0.218***	-	0.236***	0.249***
	Age squared	-	-0.002***	-0.002***	-	-0.002***	-0.002***	-	-0.002***	-0.002***
	Employed	-	-0.414***	-0.257**	-	-0.581***	-0.481***	-	-0.650***	-0.496***
	Unemployed	-	0.27	0.328	-	-0.069	0.01	-	0.321	0.436
	Marred/ De-facto	-	-0.072	-0.136	-	-0.144	-0.196	-	-0.267**	-0.360***
	City	-	-0.038	-0.043	-	0.15	0.117	-	0.183	0.149
	Regional area	-	0.229	0.208	-	0.203	1.158	-	0.360***	0.277*
Behav	Smoker	-	-	-00585***	-	-	-0.104***	-	-	-0.464***
	Alcohol	-	-	-0.276*	-	-	-0.585***	-	-	-0.368**
	Vegetables	-	-	-0.095***	-	-	-0.075**	-	-	-0.135***
	Carbohydrate	-	-	-0.194***	-	-	-0.277***	-	-	-0.158***
	Snack food	-	-	-0.071*	-	-	0.011	-	-	-0.03
	Fried food	-	-	0.174***	-	-	0.100*	-	-	0.194***
	Red meat	-	-	0.210***	-	-	0.133***	-	-	0.306***
	Processed food	-	-	0.111***	-	-	0.217***	-	-	0.108***
	Exercise freq	-	-	-0.345***	-	-	-0.357***	-	-	-0.442***
Aux	Constant	3.542***	-0.646*	0.111	3.798***	-0.354	0.664	4.133***	-0.771**	-0.335
	R^2	0.023	0.043	0.076	0.027	0.047	0.082	0.036	0.064	0.108
	F	6.868	11.162	13.548	8.25	11.955	14.211	15.168	21.82	25.767
	N	6701	6701	6701	6962	6962	6962	9687	9687	9687

Note. The table presents estimates of Models 1–3 from Equations (3.2–3.4) with BMI* as the dependent variable. Model 1 contains only circumstance variables while Model 2 includes demographic controls. Model 3 further adds behavioural variables. Parameters are estimated using OLS with heteroskedasticity-robust standard errors throughout. Dummies are defined with respect to a reference individual who is an unmarried male, non-smoker and non-drinker and lives in a remote area. Parental educational attainments are none/unknown. *, ** and *** define significance at 10%, 5% and 1%, respectively.

Table 3.3 Circumstances and Health Behaviours

Variable	2007			2009			2013		
	Vegetables	Carbs	Snack food	Fried	Red meat	Proc food	Smoking	Alcohol	Exercise
Female	0.330***	-0.013	-0.364***	-0.472***	-0.275***	-0.603***	-0.052***	-0.054***	-0.229***
Non-biol mother	0.123	0.090	0.159*	0.011	0.192***	0.227***	0.075***	-0.008	-0.086
Non-biol father	-0.319***	-0.070*	0.075	0.177***	-0.062	0.075	0.134***	-0.005	0.017
Indigenous	-0.05	-0.136	0.207	0.389***	0.292*	0.420**	0.130**	0.020	-0.153
Parents divorced	-0.099**	-0.027	0.01	0.052*	-0.047	0.011	0.061***	0.00	0.042
Refugee	0.067	-0.342***	-0.257**	-0.088	-0.118	0.131	-0.047*	-0.023	0.126
Oldest child	0.085***	0.045**	-0.026	-0.024	0.01	-0.014	-0.032***	0.016***	0.011
Father immigrated	-0.071*	0.097***	0.043	-0.019	-0.063**	-0.064*	0.001	-0.005	-0.043
Mother immigrated	0.066*	0.109***	0.083**	0.046	-0.036	-0.023	0.016	0.005	0.019
Born overseas	0.235***	-0.101***	-0.298***	-0.102***	-0.147***	-0.167***	-0.037***	0.001	0.013
Non-native English	-0.045	0.719***	0.075	-0.117**	0.137***	-0.083	-0.030**	-0.126***	-0.35***
Father employ	-0.034	0.046	0.085*	0.066*	-0.010	0.017	0.045***	0.006	-0.088*
Mother employ	-0.149***	0.127****	0.198***	0.147***	-0058***	0.030	0.033***	0.022***	0.040
Father died	0.03	-0.101-	-0.116*	-0.120**	0.037	-0.102*	-0.043***	-0.02	-0.099*
Mother died	0.027	-0.117	-0.230**	-0.047	0.049	-0.106	0.027	0.003	0.168*
Father prim ed	-0.027	0.035	0.029	-0.118	-0.082	-0.091	-0.059	-0.034	0.201
Father some HS	-0.096	0.092	0.133	0.075	-0.041	-0.019	-0.055	0.032	0.251
Father Non-U T	-0.002	0.108***	0.110***	0.076***	-0.118***	-0.020	-0.018**	0.015**	0.031
Father university	0.060	0.129***	0.107**	0.05*	0.119***	-0.019	0.018	0.030***	0.127***

Table 3.3 (Continued)

Variable	2007			2009				2013	
	Vegetables	Carbs	Snack food	Fried	Red meat	Proc food	Smoking	Alcohol	Exercise
Mother prim ed	-0.014	-0.233*	-0.237*	-0.018	0.112	0.000	0.024	-0.006	-0.311*
Mother some HS	-0.074	-0.148	-0.086	0.028	0.001	0.027	0.070*	-0.023	-0.130
Mother full HS	-0.057	-0.002	0.113	0.154*	0.034	0.078	0.051	-0.024	-0.095
Mother Non-U T	0.051	0.168***	0.202***	0.079***	0.014	0.046	-0.002	0.019**	0.111***
Mother university	-0.019	0.245***	0.287***	0.178***	0.026	0.069*	0.014	0.007	0.064
Father occ stat	0.006***	0.004***	-0.001	-0.004***	-0.002***	-0.003***	-0.001***	0.001**	0.001
Mother occ stat	0.004***	0.003***	0.001*	-0.001**	-0.001*	-0.001**	-0.001**	0.001	0.003***
R^2	0.041	0.079	0.041	0.057	0.025	0.062	0.032	0.025	0.018
F	39.116	62.41	36.564	53.331	20.535	56.925	26.429	17.793	15.722
N	23,350	23,350	23,350	23,350	23,350	23,350	23,350	23,350	23,350

Notes: The table presents estimates from Equation (3.5) with health behaviours as the dependent variables and circumstances as covariates. All estimates are performed on the pooled sample and obtained using OLS with heteroskedasticity-robust standard errors. *, ** and *** define significance at 5%, 1% and 0.1%, respectively.

3.3.3 Which Circumstances Matter Most?

A range of factors related to paternal SES significantly predicts the BMI of Australians. Firstly, Table 3.1 shows that the effect of the father's occupational status on BMI is negative and significant in every model in each wave. According to Equation (3.2), a unit rise in the father's occupation level correlates with a decrease in BMI and BMI* by 0.009–0.016 units and 0.006–0.011, respectively. Since the standard deviation in this variable is 23.07, we conclude that a *ceteris paribus* standard deviation increase yields a 0.20–0.37-unit decline in BMI and a 0.14–0.25 decline in our unhealthy bodyweight measure. In isolation, these effect sizes are fairly small—for a 170 cm tall individual, a 0.1 unit reduction in BMI corresponds to a 290gm reduction in bodyweight. However, as markers of parental SES are positively correlated (e.g., other paternal and maternal educations, incomes), these effects will aggregate across dimensions.

Considering maternal SES, the results for Eq (3.2) in Table 3.1 show that occupational status and education level also have significant and negative links with individual BMI. However, compared to paternal SES, the influence of maternal SES on an individual's BMI is relatively minor. Jusot et al. (2010) uncover similar results for an IOP study of self-assessed health and suggest that this is due to the low participation of women in the labour market. If this is the case, it appears that we might be observing a resource effect along the lines found by Mulligan (1997). Since maternal and paternal education and occupational status might yield comparable cultural or social effects, the latter is much more likely to also bring monetary benefits, suggesting that access to economic resources plays an important role.

The other covariates presented in Tables 3.1 and 3.2 also give relatively straightforward results. For example, indigenous Australians have consistently higher outcomes for both dependent variables. Similarly, an individual who has grown up in a family with divorced or separated parents has a robustly (but only occasionally significantly) higher BMI than an individual living with two parents (e.g., according to Eq (3.2), on average, the BMI of single-parent individuals is 0.2–0.4 units higher than two-parent individuals). Stress and disruption of the family structure are significant barriers to the mental and physical development of children (Escarce, 2003; Perales et al., 2017). Furthermore, economic conditions are typically poorer in a single-parent family compared to a two-parent family (McLanahan & Sandefur, 1994; Mueller & Cooper, 1986). Due to this disadvantage, unsupervised children and adolescents of single parents are more likely to engage in

unhealthy lifestyle behaviours, such as smoking, drinking alcohol and drug use (Richardson et al., 1993).

The results presented in Table 3.1 show that women have significantly lower BMIs than men, although the strength of this effect seems to be lessening with time. Across the models, the strongest gender effects were in 2007, with a steady decline noted until 2013. However, if we turn to the results for BMI in Table 3.2, we do not see significant effects associated with gender. Thus, women are more likely to have low body weight but are not more likely to be in the unhealthy range. This suggests that problems associated with being underweight are largely a female phenomenon in our data. Such a result aligns with most other empirical work (e.g., Kenardy et al., 2001; van Lenthe et al., 2000).

3.3.4 Transmitting Effects

We now consider whether the correlations between circumstances and body weight emerge through differences in health behaviours: do persons born into higher SES families have healthier bodyweights because they develop better health behaviours? In order for such a mediation effect to be present, we require our health behaviour variables to be determinants of BMI or BMI* in Eq (4) and empirically linked to background characteristics (see Table A3.2 in the appendix). The results presented in Tables 3.1 and 3.2 highlights the first part of this mechanism, where some lifestyle health behaviours highly correlate with BMI and BMI*. Strong results hold for vegetable consumption and exercise (which are negatively correlated with body weight) and for red meat and processed foods (which have positive associations). It is notable that smoking is predictive of lower body weight, which likely reflects an appetite suppressant effect (Audrain-McGovern & Benowitz, 2011).[10] A similar result is found for alcohol, which may reflect a substitution between alcohol consumption and that of other foods or drinks. It is also plausible that the true results of these variables may also be stronger than our estimates suggest due to downward biases in reporting negative behaviours. This may be picked up by our socioeconomic markers if correlated with parental status (e.g., Graham & Owen, 2003).

The expected signs on the other behavioural variables are more ambiguous and depend upon (i) variations of healthy/ unhealthy food types within these categories and, as above,

[10] Clearly, smoking will predict negative outcomes for other dependent variables such as cardiovascular health.

(ii) the degrees to which consumption of one food type offsets consumption of another. For example, red meat and carbohydrate-rich foods predict positive and negative impacts on body weight, respectively, which may not be intuitive when considered in isolation. Nonetheless, in general, our behaviours mostly conform to expectations associated with a healthy weight (Kontogianni et al., 2010; Sundquist & Johansson, 1998).

The beta coefficients of the circumstances in Eq (3.4) also indicate that health behaviours partially mediate some circumstances of an individual's health outcomes. According to the results shown in Tables 3.1 and 3.2, point estimates of paternal occupation level and education—compared to Eq (3.3)—are particularly mediated substantially by the inclusion of health behaviours. These variables are also strongly associated with certain behaviours, such as exercise and the consumption of processed foods (Table A3.2), in children born to higher SES fathers who inherit different behavioural traits. Past empirical evidence (Currie & Hyson, 1999; Trannoy et al., 2010) has found that the effects of fathers' SES on health are transmitted primarily through access to resources. Such a result would hold if poorer individuals consume inexpensive high-energy foods or are more likely to use eating to diffuse stress (Watson, 2018). Cultural dietary norms associated with SES may also be relevant (Pestoni et al., 2019).

3.4 How Do Correlations between Circumstances and Behaviours Affect IOP Estimates?

The estimations presented above make use of the correlations that appear between circumstances variables and markers of behaviour.[11] We now analyse these links in detail and model the two-part mechanism flowing from background characteristics to health inputs like diet and exercise. Our baseline model is of the form

$$B_{pi} = \delta + \sum_{j=1}^{k} \xi_j C_{ij} + \epsilon_i \quad p = 1, \dots, q \qquad (3.5)$$

and is estimated by OLS. As above, we have made a cardinality assumption for the health behaviour measures B_p. While this may be relaxed (e.g., by using an ordered probit model), linearity is desirable as it later allows us to decompose the inequality estimates into contributions accounted for by the correlations between B and C. Contrasting

[11] Note that the correlations between health behaviors themselves inform the models estimated in Eq (3.2–3.4). Table A3.3 in the appendix shows these correlations. A key result here is that beneficial behaviors tend to be positively associated with each other. For example, vegetable consumption is positively linked with exercise frequency, and both are negatively associated with tobacco and alcohol consumption.

estimates from ordered regression models with linear specifications (Ferrer-i-Carbonell & Frijters, 2004) suggests that this assumption is not too unreasonable.

Table 3.3 presents estimates of $\xi_1, \ldots \xi_k$ for health behaviours $p = 1, \ldots, q$ and shows that parental characteristics significantly predict a variety of health behaviours. An individual born to a father with a higher occupational status has a higher likelihood of eating vegetables and undertaking physical activity, whereas the same individual has a lower probability of eating red meat, processed meat and smoking. Similar (albeit less strong) results also hold for maternal status indicators. Again, our results concur with previous empirical evidence (Gidlow et al., 2006; Hulshof et al., 2003), showing that individuals born into relatively favourable socioeconomic conditions inherit better behavioural patterns. Unhealthy behaviour among low SES groups occurs across low education levels (Fransen et al., 2016; Hulshof et al., 2003) and may be due to limited access to healthy foods (W. P. James et al., 1997).

Although these results have implications for the pathways that transmit circumstances into health outcomes, the contributions of these inherited factors are also very low (the R^2 terms are around 1%–4%, see Table 3.3). Thus, like bodyweight, health behaviours are not strongly linked with an individual's observable inherited circumstances. Other factors outside the model (genetics, unobserved circumstances, personality, etc.) are therefore likely to be the primary drivers of behaviour and therefore account for our unexplained variations in body weight.

3.4.1 Regression-Based Decomposition

In this section, we investigate more deeply the correlations between circumstances and health behaviours examined above and consider their consequences for the interpretation of IOP estimates. If an individual inherits circumstances that promote unhealthy behaviours, should the consequences of these behaviours form part of the illegitimate inequality that characterizes IOP (since they are predetermined)? Alternatively, as these behaviours still lie within personal control, should they be classified as effort and therefore represent legitimate sources of inequality? Jusot et al. (2013) provide a framework for examining these issues. They note that attributing all predetermined drivers of inequality to circumstances coincides with a Roemerian definition of IOP, while allowing individuals to benefit from efforts correlated with circumstances; this coincides with the philosophical view of Barry (2005).

Jusot et al. (2013) also provide an econometric framework for evaluating the importance of circumstances-attributed behaviours. Using Eq (3.5), we define an additional measure of health behaviour $-B_{ip}^{\dagger} = B_p + \epsilon_{ip}$. This is the behaviour of individual i with any component attributable to circumstances removed. $B_{ip}^{\dagger}$ is sometimes interpreted as the pure effort exerted by the individual. We also define summaries of the aggregate effects of our variables $\hat{C} = \sum_{j=1}^{k} \hat{\beta}_{3j} C_j$, $\hat{D} = \sum_{l=1}^{m} \hat{\beta}_{3l} D_l$, $\hat{B} = \sum_{p=1}^{q} \hat{\gamma}_{3p} B_p$, and $\hat{B}^{\dagger} = \sum_{p=1}^{q} \hat{\gamma}_{3p} B_p^{\dagger}$. The following formulae provide generic algebraic decompositions of the R^2 terms (equal to the ratio of explained variance to total $-\sigma^2_{\hat{H}}/\sigma^2_H$) from our models. For brevity, we estimate these on the pooled sample such that we obtain summary estimates over the complete data set, rather than wave-by-wave as above.

$$\frac{\sigma^2_{\hat{H}}}{\sigma^2_H} = \frac{1}{\sigma^2_H}\left[Cov\left(\hat{H};\, \hat{C}\right) + Cov\left(\hat{H};\, \hat{D}\right) + Cov\left(\hat{H};\, \hat{B}\right)\right] \qquad (3.6)$$

$$\frac{\sigma^2_{\hat{H}}}{\sigma^2_H} = \frac{1}{\sigma^2_H}\left[Cov\left(\hat{H};\, \hat{C}\right) + Cov\left(\hat{H};\, \hat{D}\right) + Cov\left(\hat{H};\, \hat{B}^{\dagger}\right)\right] \qquad (3.7)$$

Here, Eq (3.6) breaks down the explained inequality (the explained percentages from Tables 3.1 and 3.2) into additive contributions from each set of variables *C*, *D* and *B*. Eq (3.7) presents the same decomposition but, in this instance, behaviours that may be attributed to circumstances are reallocated from *B* to *C*. The difference between $Cov\left(\hat{H};\, \hat{B}\right)$ and $Cov\left(\hat{H};\, \hat{B}^{\dagger}\right)$ therefore, captures this effect and, hence, the empirical consequences of Barryian versus Roemerian philosophical treatments. The results are presented in Table 3.4, where raw estimates are given in the top two panels and percentage contributions in the lower panels.

The first three rows give the covariances that make up the elements in Eq (3.6)—jointly, this sum gives the explained variance $\sigma^2_{\hat{H}}$. Estimates for BMI are given in the left-most three columns, while those for overweightness (BMI*) appear on the right. The lower panel presents the same results where $\hat{B}^{\dagger}$ replaces $\hat{B}$; these estimates hence correspond to the Roemerian case where correlations between circumstances and behaviour are attributed to circumstances.

Across both health measures, we see that health behaviours account for the largest shares of explained variation. In the unattributed cases, behaviours account for approximately 40% of explained inequality in BMI and about 45% of the inequality in BMI*. The

remainder is attributed to demographic controls—around 40% for BMI and 30%–35% for BMI*—and circumstances—20%–25% for both variables. Thus, approximately, this fraction of explained inequality from Model 3 (Eq 3.4) comes from circumstances—estimates that are not too dissimilar from those obtained from Eq (3.2).[12]

The effects of re-attributing correlated behavioural characteristics to circumstances appear in the bottom panel. If we replace $\hat{B}$ with $\hat{B}^{\dagger}$, the effects of circumstances increase by 6%–7% across our three years for BMI and 5%–6% for BMI*.

Table 3.4: Decompositions of Explained Inequality—Circumstances, Demographic Controls, and Behaviours

Contributing Factor	Body mass index—BMI			Overweightness—BMI*		
	2007	2009	2013	2007	2009	2013
Circumstances Cov $(\widehat{\boldsymbol{H}};\widehat{\boldsymbol{C}})$	0.649	0.591	0.609	0.372	0.377	0.374
Demographics Cov $(\widehat{\boldsymbol{H}};\widehat{\boldsymbol{D}})$	1.224	1.300	1.702	0.460	0.498	0.705
Behaviours Cov $(\widehat{\boldsymbol{H}};\widehat{\boldsymbol{B}})$	1.201	1.286	1.597	0.646	0.741	0.964
Total	*3.074*	*3.178*	*3.908*	*1.478*	*1.616*	*2.042*
Circumstances attributed Cov $(\widehat{\boldsymbol{H}};\widehat{\boldsymbol{C}})$	0.843	0.809	0.860	0.460	0.475	0.488
Demographics Cov $(\widehat{\boldsymbol{H}};\widehat{\boldsymbol{D}})$	1.224	1.300	1.702	0.460	0.498	0.705
Attributed behaviours Cov $(\widehat{\boldsymbol{H}};\widehat{\boldsymbol{B}})$	1.007	1.069	1.346	0.557	0.643	0.849
Cov $(\widehat{\boldsymbol{H}};\widehat{\boldsymbol{B}})$ - Cov $(\widehat{\boldsymbol{H}};\widehat{\boldsymbol{B}}^{\dagger})$	0.194	0.217	0.251	0.089	0.098	0.115
Total	*3.074*	*3.178*	*3.908*	*1.478*	*1.616*	*2.042*
Circumstances Cov $(\widehat{\boldsymbol{H}};\widehat{\boldsymbol{C}})$%	21.12	18.61	15.59	25.14	23.32	18.29
Demographics Cov $(\widehat{\boldsymbol{H}};\widehat{\boldsymbol{D}})$%	39.82	40.91	43.56	31.14	30.81	34.53
Behaviours Cov $(\widehat{\boldsymbol{H}};\widehat{\boldsymbol{B}})$%	39.06	40.48	40.86	43.71	45.87	47.18
Total	*100%*	*100%*	*100%*	*100%*	*100%*	*100%*
Circumstances attributed Cov $(\widehat{\boldsymbol{H}};\widehat{\boldsymbol{C}})$%	27.43	25.24	22.00	31.14	29.41	23.88
Demographics Cov $(\widehat{\boldsymbol{H}};\widehat{\boldsymbol{D}})$%	39.82	40.91	43.56	31.14	30.81	34.53
Attributed behaviours Cov $(\widehat{\boldsymbol{H}};\widehat{\boldsymbol{B}})$	32.75	33.63	34.44	37.71	39.78	41.59
Cov $(\widehat{\boldsymbol{H}};\widehat{\boldsymbol{B}})$ - Cov $(\widehat{\boldsymbol{H}};\widehat{\boldsymbol{B}}^{\dagger})$%	6.31	6.85	6.42	6.00	6.09	5.59
Total	*100%*	*100%*	*100%*	*100%*	*100%*	*100%*

Notes: The table presents decompositions from Eq (3.6–3.7) based upon Jusot et al. (2013). The first panel gives an additive decomposition of explained inequality where correlations between behaviours and circumstances are not attributed to circumstances as per Barry (2005). The second panel presents the same decomposition based on pure efforts defined via Eq (3.5) as per Roemer (1998). The third and fourth panels express these contributions as percentages, while the row Cov $\widehat{\boldsymbol{H}};\widehat{\boldsymbol{B}})$ - Cov$(\widehat{\boldsymbol{H}};\widehat{\boldsymbol{B}}^{\dagger})$ gives the percentage of explained inequality re-attributed to circumstance.

[12] Note that the estimates in Table 3.4 handle potential confounding effects while those in Tables 3.1 and 3.2 do not.

Correspondingly, the inequality attributed to behaviours falls by these amounts. These estimates here are remarkably stable and can be further expressed by examining the composition of the aggregate share explained by circumstances. Taking the ratio $[\mathrm{Cov}\left(\widehat{H};\widehat{B}\right) - \mathrm{Cov}\left(\widehat{H};\widehat{B}^{\dagger}\right)]/[\mathrm{Cov}\left(\widehat{H};\widehat{C}\right)]$, where the denominator comes from Eq (3.7), reveals that the total inequality attributed to *C* is comprised of a direct effect which makes up 70%–80%, and a correlation with *B*, which explains the other 20%–30%. If background characteristics cause variations in behaviour, such an effect accounts for only around a quarter of the inequality they explain.

3.5 Implications for the Provision of Healthcare

Despite many interventions to reduce the disparity in health between groups of differing SES, ethnicity or race, and other social factors, inequality in health due to unequal opportunity remains a significant issue. It is apparent that the policy for expanded access to healthcare services has implications for overcoming health inequality. However, the demand for publicly funded free health services exceeds the supply (Sharma et al., 2013). Thus, some form of compensating mechanism is often required (Bricard et al., 2013).

According to our results, individuals' inherited circumstances affect their body weight directly as well as indirectly through health behaviours. We consider the direct effect first. If the correlation between health behaviours and the hereditary factors that are beyond an individual's control affects intergenerational inequality in health, then the policy of prioritizing the provision of healthcare or other compensation options based on a Roemerian approach (Roemer, 1998) would reduce unfair disparities in health. For instance, even after allowing for behaviours, individuals who suffer from unhealthy body weight disproportionately originate from families from less-advantaged backgrounds (see Table 3.1 and Table 3.2). These individuals did not control their parents' decisions nor their parents' education or occupation, so it is hard to hold them fully responsible for health problems associated with body weight. Proposals designed to bolster equity in health (such as extending access for lifestyle-related illnesses to disadvantaged areas) would be beneficial. Since the covariates that predict unhealthy body weight in our models are also predictors of lower income (e. g. Checchi et al. 2010), policies that direct resources toward poorer individuals will also disproportionately benefit those with less opportunity for a healthy BMI.

The implications of the indirect effects that we study are less clear. Individuals who inherit favourable circumstances tend to consume healthier diets and engage in more

physical activity, which would have beneficial flow-on effects on their health. How we treat these path effects depends upon Roemarian or Barryian value judgments concerning responsibility over inherited behavioural traits. On the one hand, some fraction of health behaviour is attributable to circumstances and is, therefore, seemingly beyond the control of the individual. On the other hand, even if the discipline to lead a healthy lifestyle is predetermined, the effort to do so (and subsequent disutility) is still experienced. Determining how to interpret health behaviours attributable to circumstances remains an issue of practical importance for the healthcare sector. Nonetheless, as we show, the quantitative implication of this correlation is relatively small.

3.6 Conclusion

By using regression models applied to Australian microdata, this study examined the effects of various circumstance variables on bodyweight. We interpreted the explained component from these models as a measure of IOP; that is, the fraction of variation that is accounted for by factors beyond an individual's control, such as their race, gender and inherited social class. Our results show that IOP with respect to bodyweight is relatively small, with predetermined factors explaining only around 3%–4% of the inequality in BMI and a BMI-derived indicator of overweightness. The estimates were contrasted with those found by other authors for indicators such as ordinal self-assessed health scores, which attribute a much greater fraction of variation to circumstances. A likely explanation is that bodyweight is much more responsive to health behaviours than more general health indicators and, hence, the proportion explained by background variables is smaller.

Despite our relatively small estimates, there is some evidence that some parental characteristics do have intergenerational effects. Specifically, the SES of an individual's father appears to be a key determinant, with persons born to more advantaged fathers having a small health advantage in later life when considering weight-related health. However, since (i) markers of paternal SES tend to be positively correlated, and (ii) body composition is predictive of a spectrum of negative health outcomes, the aggregate effects of these socially determined inequalities can still be substantial.

Our results also show that empirical links between circumstances and outcomes are only partially reflective of correlations between background and health behaviours, such as the consumption of vegetables and engagement in physical activity. Decompositions revealed that these correlations only explain around 6% of explained health inequality; statistically attributing health behaviours to circumstances when correlated only increases IOP

estimates by 20%–25%. Therefore, desirable background characteristics seem to produce health benefits that cannot be fully accounted for by improved lifestyle choices.

Lastly, we argued that these results have implications for the provision of healthcare when resources are constrained. Certain factors entirely beyond an individual's control (such as the educational attainments of their parents) and additional factors that are potentially uncontrollable (i.e., behaviours that are predicted by circumstances) both exert influence over body weight. Therefore, it may be desirable to promote those from disadvantaged backgrounds when treating lifestyle-related diseases in order to offset other predetermined inequalities.

Appendix

Table A 3.1 Descriptive Statistics

Variable	Mean	StDev	Min	Max	Variable	Mean	StDev	Min	Max
BMI	26.71	5.453	11.9	85.3	Father University	0.146	0.353	0	1
*BMI_*	2.917	4.353	0	60.3	Mother Primary Education	0.016	0.126	0	1
Underweight	0.023	0.150	0	1	Mother Some H School	0.071	0.257	0	1
Overweight	0.343	0.475	0	1	Mother Complete H School	0.088	0.284	0	1
Obese	0.223	0.416	0	1	Mother Non-Uni Tertiary	0.173	0.378	0	1
Female	0.523	0.499	0	1	Mother University	0.114	0.318	0	1
Non-Biological Mother	0.022	0.147	0	1	Father Occ Status	45.69	23.07	0	100
Non-Biological Father	0.061	0.240	0	1	Mother Occ Status	42.95	23.37	0	100
Indigenous	0.005	0.073	0	1	Age	44.13	16.03	18	80
Parents Divorced	0.103	0.304	0	1	Age Squared	2204	1481	324	6400
Refugee	0.013	0.114	0	1	Employed	0.721	0.448	0	1
Oldest child	0.342	0.475	0	1	Unemployed	0.029	0.168	0	1
Father Immigrant	0.382	0.486	0	1	Married or De facto	0.684	0.465	0	1
Mother Immigrant	0.357	0.479	0	1	City	0.247	0.431	0	1
Born Overseas	0.205	0.404	0	1	Regional Area	0.120	0.325	0	1
Non-Native English	0.079	0.269	0	1	Smoker	0.196	0.397	0	1
Father Employed at 14	0.238	0.426	0	1	Drinks Alcohol	0.873	0.333	0	1
Mother Employed at 14	0.668	0.471	0	1	Vegetable Cons Freq	6.767	1.562	1	8
Father Died in Childhood	0.047	0.211	0	1	Carbohydrate Cons Freq	4.603	1.233	1	8
Mother Died in Childhood	0.014	0.117	0	1	Snack Food Cons Freq	3.726	1.478	1	8
Father Prim. Education	0.021	0.144	0	1	Fried Food Cons Freq	3.378	1.224	1	8
Father Some H School	0.074	0.262	0	1	Red Meat Cons Freq	4.958	1.219	1	8
Father Complete H School	0.076	0.265	0	1	Processed Food Cons Freq	3.956	1.360	1	8
Father Non-Uni Tertiary	0.205	0.404	0	1	Exercise Freq	3.684	1.502	1	6

Note: The table presents means, standard deviations and min/max for all variables used in the chapter. Observations are taken over three years (2007, 2009, 2013), and the pooled sample is 23,350 observations. The reference individual is a non-indigenous male from non-immigrant parents with no/unspecified parental educational attainments. The individual is unmarried, a non-smoker and drinker and lives in a remote area. Note that the behavioural indicators are constructed as ordinal and hence rely on a cardinality assumption to define means and variances.

Table A 3.2 Correlation Coefficients - Circumstance and Behaviours

Variable	Smoking	Alcohol	Vegetable	Carbs	Snacks	Fried	Red Meat	Proc Food	Exercise
Female	-0.068	-0.083	0.107	0.000	-0.122	-0.194	-0.110	-0.221	-0.076
Non-Biol Mother	0.061	-0.005	-0.009	-0.001	0.017	0.014	0.022	0.032	-0.007
Non-Biol Father	0.096	-0.005	-0.051	-0.022	0.016	0.038	0.001	0.026	-0.005
Indigenous	0.035	0.007	-0.014	-0.015	0.016	0.033	0.019	0.028	-0.008
Parents Divorced	0.051	0.006	-0.022	-0.002	0.011	0.021	-0.013	0.007	0.012
Refugee	-0.027	-0.039	0.014	0.017	-0.030	-0.021	-0.018	-0.009	-0.009
Oldest Child	-0.036	0.026	0.030	0.028	-0.001	-0.006	0.000	-0.003	0.009
Father Immigr	-0.012	-0.037	0.020	0.103	-0.006	-0.017	-0.055	-0.058	-0.026
Mother Immigr	-0.011	-0.033	0.036	0.112	-0.003	-0.012	-0.053	-0.057	-0.019
Born Overseas	-0.043	-0.054	0.059	0.110	-0.046	-0.043	-0.062	-0.079	-0.027
Non-Native Eng	-0.045	-0.102	0.034	0.167	-0.021	-0.048	-0.022	-0.062	-0.052
Father Employ	0.048	0.015	-0.029	0.046	0.059	0.055	-0.010	0.013	0.001
Mother Employ	0.039	0.033	-0.036	0.082	0.080	0.062	-0.033	0.005	0.020
Father Died	-0.022	-0.022	-0.002	-0.026	-0.026	-0.022	0.012	-0.014	-0.020
Mother Died	0.007	-0.008	0.002	-0.023	-0.027	-0.009	0.014	-0.005	0.009
Father Prim Ed	0.013	-0.029	-0.019	-0.024	-0.011	-0.003	0.006	-0.001	-0.017
Father Some HS	0.041	0.015	-0.047	-0.012	0.035	0.057	0.001	0.021	0.008
Father Full HS	-0.006	0.018	0.012	0.081	0.048	0.016	-0.018	-0.008	0.019
Father Non-UT	-0.017	0.026	0.003	0.044	0.041	0.029	-0.031	0.001	0.014
Father Uni	-0.046	0.042	0.085	0.142	0.053	-0.022	-0.052	-0.037	0.054
Mother Prim Ed	0.004	-0.019	-0.014	-0.027	-0.023	-0.007	0.010	-0.006	-0.029
Mother Some HS	0.044	0.004	-0.041	-0.028	0.009	0.029	-0.003	0.012	-0.002
Mother Full HS	0.005	0.018	0.001	0.086	0.072	0.045	-0.014	0.005	0.025
Mother Non-UT	-0.015	0.033	0.035	0.077	0.057	0.012	-0.017	-0.004	0.039
Mother Uni	-0.018	0.027	0.049	0.137	0.078	0.019	-0.030	-0.014	0.043
Father Occ Stat	-0.072	0.051	0.128	0.152	0.037	-0.067	-0.063	-0.063	0.054
Mother Occ Stat	-0.043	0.045	0.096	0.152	0.071	-0.016	-0.040	-0.024	0.074

Note: The table present correlation coefficients depicting associations between health behaviours and circumstance variables obtained from the pooled sample Based upon the formula $SE\ (\hat{\rho}) = \sqrt{1-\hat{\rho}}/(n-2)$ where n = 23, 350, estimates will be significant at α= 5% when $|\hat{\rho}|$> 0.0129

Table A 3.3 Correlation Coefficients - Health Behaviours - Pooled Sample

Variable	Smoking	Alcohol	Vegetable	Carbs	Snacks	Fried	Red Meat	Proc Food	Exercise
Smoking	1.000								
Alcohol	0.056	1.000							
Vegetable	-0.189	0.001	1.000						
Carbs	-0.047	0.017	0.096	1.000					
Snacks	0.072	0.073	-0.141	0.262	1.000				
Fried	0.143	0.040	-0.232	0.147	0.463	1.000			
Red Meat	0.044	0.049	0.052	0.030	0.100	0.167	1.000		
Proc Food	0.117	0.096	-0.126	0.080	0.293	0.375	0.423	1.000	
Exercise	-0.037	0.055	0.115	0.029	-0.033	-0.068	-0.006	-0.034	1.000

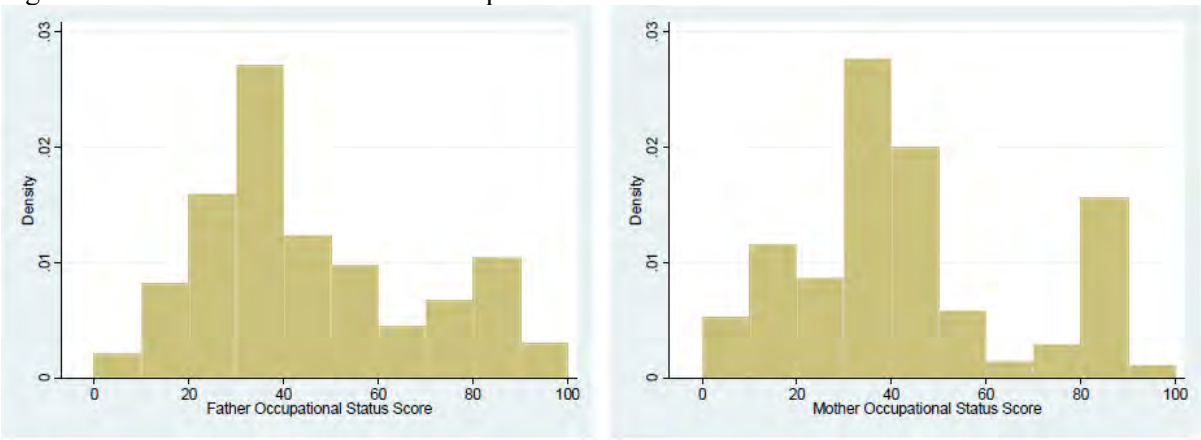
Note: The table present correlation coefficients depicting associations between health behaviours and circumstance variables obtained from the pooled sample Based upon the formula $SE\ (\hat{\rho}) = \sqrt{1-\hat{\rho}}/(n-2)$ where n = 23, 350, estimates will be significant at α= 5% when $|\hat{\rho}|$> 0.0129

Table A 3.4 Associations Between Circumstances, Health Behaviour and Self-Rated Health

	Variable	2007			2009			2013	
		M(1)	M(2)	M(3)	M(1)	M(2)	M(3)	M(1)	M(2)	M(3)	M(1)	M(2)	M(3)
Circ	Female	0 012	-0 047**	-0 036	0 01	-0 049**	-0 038*	-0 022	-0 076***	-0 052***
	Non-Biol Mother	0 144	0 154*	0 132	0 162*	0 145*	0 11	0 048	0 04	0 013
	Non-Biol Father	0 063	0 089*	0 041	0 059	0 081	0 046	0 182***	0 175***	0 122***
	Indigenous	0 349**	0 329*	0 286*	0 386**	0 349**	0 265*	0 177*	0 227**	0 173*
	Parents Divorced	0 034	0 04	0 03	0 04	0 032	0 02	0 055*	0 048	0 03
	Refugee	-0 097	-0 156	-0 172*	-0 021	-0 065	-0 016	0 041	-0 062	-0 024
	Oldest Child	-0 087***	-0 080***	-0 065***	-0 071***	-0 056**	-0 044**	-0 093***	-0 088***	-0 066***
	Father Immigr	-0 003	0 003	-0 005	-0 032	-0 023	-0 033	-0 027	-0 008	-0 005
	Mother Immigr	-0 003	0 038	0 046	-0 02	0 014	0 025	-0 034	-0 001	-0 006
	Born Overseas	0 059	-0 064	-0 034	0 134***	-0 01	-0 003	0 076**	-0 061*	-0 068**
	Non-Native Eng	0 141**	0 142***	0 107**	0 101*	0 104*	0 078	0 117***	0 158***	0 132***
	Father Employ	-0 090**	0 017	-0 025	-0 039	0 048	0 025	0 041	0 048	0 034
	Mother Employ	-0 048**	0 044*	0 039*	-0 052**	0 047*	0 051**	-0 074***	0 012	0 016
	Father Died	0 132**	0 055	0 067	0 187***	0 104*	0 082	0 106**	0 034	0 027
	Mother Died	-0 037	-0 052	-0 032	0 063	0 023	0 034	0 023	-0 003	0 009
	Father Prim Ed	0 162	0 122	0 181	-0 159	-0 152	-0 184	-0 047	-0 101	-0 040
	Father Some H S	0 083	0 112	0 145	-0 269*	-0 164	-0 131	-0 151**	-0 084	-0 023
	Father Full H S	0 018	0 050	0 070	-0 376**	-0 303**	-0 249*	-0 172**	-0 095	-0 031
	Father Non-U T	-0 111***	-0 031	-0 005	-0 098***	-0 031	-0 010	-0 080***	-0 019	-0 009
	Father Uni	-0 134***	-0 054	-0 021	-0 115***	-0 028	0 019	-0 120***	-0 054	-0 008
	Mother Prim Ed	-0 128	-0 097	-0 157	0 133	0 096	0 066	0 084	0 028	-0 056
	Mother Some H S	-0 151	-0 084	-0 124	0 120	0 106	0 065	0 06	0 024	-0 041
	Mother Full H S	-0 100	-0 062	-0 063	0 206	0 205	0 173	-0 035	-0 007	-0 065
	Mother Non-U T	-0 115***	-0 049	-0 007	-0 083***	-0 010	0 017	-0 094***	-0 016	0 006
	Mother Uni	-0 023	0 073	0 090**	-0 080*	0 023	0 037	-0 114***	-0 006	0 008
	Father Occ Stat	-0 003***	-0 003***	-0 002***	-0 002***	-0 002***	-0 001**	-0 003***	-0 003***	-0 002***
	Mother Occ Stat	-0 002***	-0 001	0 000	-0 002***	-0 001**	-0 001	-0 002***	-0 002***	-0 001*
Cont	Age	0 031***	0 026***	0 034***	0 028***	0 031***	0 023***						
	Age Squared	-0 000***	-0 000***	-0 000***	-0 000***	-0 000***	-0 000***						
	Employed	-0 443***	-0 394***	-0 379***	-0 319***	-0 416***	-0 329***						
	Unemployed	-0 191**	-0 216**	-0 098	-0 118*	-0 09	-0 078						
	Married/De-facto	-0 110***	-0 053**	-0 145***	-0 101***	-0 102***	-0 057***						
	City	0 050*	0 053**	0 060**	0 057**	0 058***	0 051**						
	Regional Area	0 130***	0 148***	0 078**	0 084**	0 111***	0 102***						
Behav	Smoke	0 244***	0 274***	0 320***									
	Alcohol	-0 182***	-0 137***	-0 128***									
	Vegetables	-0 059***	-0 037***	-0 044***									
	Carbohydrate	-0 019*	-0 031***	-0 016**									
	Snack Food	-0 003	0 005	0 013*									
	Fried Food	0 029***	0 034***	0 041***									
	Red Meat	0 006	-0 005	0 002									
	Processed Food	-0 003	0 005	0 003									
	Exercise Freq	-0 151***	-0 152***	-0 154***									
Aux	Constant	2 869***	2 267***	3 198***	2 811***	2 097***	2 862***	2 921***	2 308***	3 005***
	R_2	0 040	0 111	0 208	0 040	0 108	0 198	0 050	0 116	0 220
	F	10 206	21 574	37 911	10 251	21 876	36 592	19 373	34 262	62 39
	N	6504	6504	6504	6735	6735	6735	9444	9444	9444

Note: The table presents estimates of Models 1–3 from Eq (2–4) with 1–10 self-rated health as the dependent variable Model 1 contains only circumstance variables while Model 2 includes demographic controls Model 3 further adds behavioural variables Parameters are estimated using OLS with heteroskedasticity-robust standard errors throughout Dummies are defined with respect to a reference individual who is an unmarried male, non-smoker and non-drinker and lives in a remote area Parental educational attainments are none/unknown Asterisks *, ** and *** define significance at 10%, 5% and 1% respectively

Figure A 3.1 Paternal and Maternal Occupational Status Score Distributions



Note: The figure presents histograms of the McMillan *et al.* (2009) ANU sociological (AUSEI06) scales designed to capture the occupational prestige of an individual's parents. The scores represent an amalgamation of dimensions related to education, income and job type. Weights are selected on the basis of regression models used to predict earnings. Note that results for individuals outside the labour force can still be imputed. The variables are usually interpreted as continuous and higher values implying greater socioeconomic status.

Chapter 4

Multigenerational Inequalities of Opportunity in Health Outcomes

4.1 Introduction

Economic inequalities are often passed down from generation to generation, where parents' socioeconomic status (SES) influences their children's distribution of outcomes. For example, children from wealthier or more-educated parents are known to be healthier (Apouey & Geoffard, 2013; Currie & Goodman, 2020; Huebener, 2019), achieve better educational outcomes (Pinquart & Ebeling, 2020) and achieve better labour markets (Erola et al., 2016). Correlations between parental characteristics and child outcomes are often interpreted under the umbrella of inequality of opportunity (IOP), which are harmful disparities that lie beyond personal control.

In this chapter, we apply IOP concepts to Australian health data, but we extend the standard econometric models to consider the effects that individuals' grandparents may play in this process. We model the impacts of grandparental SES on individuals' health while also controlling for analogous parental traits. This allows our models to capture the direct effect of grandparental status rather than the effect that flows through the intermediate (i.e., parental) generation. That is, we consider the idea that health status is not just driven by parental characteristics but by ingrained socioeconomic disparities apparent over multiple generations.

Why might grandparental SES be a source of unequal opportunity in health, even once the effects of parental characteristics are removed? We suggest two key mechanisms that may produce this type of result. On the one hand, attitudes and behaviours related to health might be passed down when grandparents have close contact with grandchildren (Braun & Stuhler, 2018; Neidhöfer & Stockhausen, 2019). Such grandparental caregiving may thus contribute directly to children's health. Family backgrounds (e.g., ethnicity, residential arrangements, number of children, and parental and grandparental SES) may also affect grandparental caregiving practices (see Sadruddin et al., 2019).

On the other hand, economic, social, and cultural factors associated with social class, as proxied by the length of time a family has held a given level of social status, may also matter. Advantages and disadvantages shift from one generation to the next, but the effects also depend on the duration over which individuals experience life within a specific social class (Solon, 2014). For instance, different sets of cultural values exist for

families of high social status for several generations (Chan & Boliver, 2013; Hertel & Groh-Samberg, 2014; Solon, 2014); these values may influence a variety of health and social behaviours.

Our analysis used six different health-related markers for physical and mental wellbeing that identify significant effects associated with grandparental SES in all cases. The estimates are most decisive for body mass index (BMI) and mental and physical health variables, although the breadth of the results across indicators is a key finding. We also found the effect strongest for grandpaternal rather than grandmaternal transmission. Since fathers (and grandfathers) tend to perform breadwinning roles, while mothers and grandmothers are more likely to play caring roles, our correlations suggest that the socioeconomics associated with material wellbeing are likely to be especially important.

To quantify the proportional impacts of different types of variables, we used regression-based econometric decompositions (i.e., Owen values) with health outcomes as the dependent variable. These results show that grandparental characteristics explain a similar proportion of IOP in Australian health compared to parental characteristics. For our physical health indicators, the explained contribution of grandparents' SES to their grandchildren's health ranged from 8% to 29% of explained inequality, while the corresponding figures for our parental SES variables were from 8% to 23%.[13] The surprisingly high proportion of inequality attributed to grandparental characteristics suggests that there may be substantial omitted-variable problems associated with the standard two-generation model used to study inequality in health.

Our work ties into a broader literature on inequality with respect to predetermined SES. The central idea here is that background characteristics, or *circumstances* reflect factors that lie beyond personal control and are, therefore, a source of unfair inequality. This is distinct from *effort*—factors that individuals have control over and which, therefore, lie within the domain of personal responsibility (Roemer, 1998). The existing research on IOP in health (Aizawa, 2019, 2021; Balasooriya et al., 2021; Bricard et al., 2013; Carrieri & Jones, 2018; Deutsch et al., 2018; Jusot et al., 2013; Rosa Dias, 2009, 2010; Trannoy et al., 2010) has considered parental characteristics as proxies for circumstances. However, we are unaware of any study that has measured IOP in health by considering the effect of the grandparents–grandchild relationship on health inequality.

[13] Although we have used a similar set of SES variables for both parents and grandparents, we considered both paternal and maternal grandparents. However, we have not included maternal grandparents' education due to the multicollinearity issue.

The rest of the chapter is structured as follows. The following section, 4.2, describes the data source, variables, and sample characteristics. Section 4.3 examines the role of the multigenerational association in IOP in health by applying regression models to demonstrate that grandparental SES is a significant determinant of IOP. Section 4.4 decomposes total predetermined inequality into contributions from different sets of circumstances, and Section 4.5 discusses some important results. The final section, 4.6, presents a summary and conclusion.

4.2 Methodology

4.2.1 Data

Our data consists of 17 waves (from 2001 to 2017) of the Household, Income and Labour Dynamics in Australia (HILDA) survey, a large, nationally representative, a random sample of more than 17000 individuals from more than 7,000 Australian households. Since 2001, HILDA has collected data on individuals' health status, demographic, and socioeconomic backgrounds using face-to-face interviews and self-completion questionaries.

To conduct our analysis, we required a multigenerational dataset assembled from HILDA by matching observations across three consecutive generations. Children (i.e., the first generation) are matched to their parents using cross-wave identifies assigned separately for both mother and father. We could then link grandparental characteristics with corresponding grandchildren because parents respond to their parents' SES questions. Our sample limits individuals less than 59 years old, while the requirement of having data on parents and grandparents restricted our sample to mostly young individuals (basic characteristics of our subsample are outlined further below).

Moreover, to increase the sample, the missing observations of time-invariant variables were replaced based on the observations in other waves to increase the sample size. Also, missing information of time-variant variables is replaced by the first lag/ or lead value of the missing place.

4.2.2 Health Outcomes

We employ six different characteristics of health as outcome variables. The first two are biological indicators of body composition. BMI is a ratio of weight in kilograms over the square of height in metres and calculated in HILDA using self-reported mass and height. Using BMI as a health measure has some standard caveats, e.g., it is not sensitive to the

deferent between body fat and muscle and does not consider the types of fat, which have a different metabolic effect, and which parts of the body contain a considerable amount of fat. However, BMI has been a widely used health measure in health-related research, showing a robust association with non-communicable diseases (Nuttall, 2015; Stommel & Schoenborn, 2009). We also define another variable to measure overweightness (BMI*), using the following formula to tackle non-monotonicities between bodyweight and health (see Apablaza, Bresson, & Yalonetzky, 2016).

$$BMI^* = \begin{cases} 0 \; if \; BMI \leq 25 \\ BMI - 25 \; if \; BMI > 25 \end{cases} \qquad (4.1)$$

Four other measures are sourced as indicators of individuals' mental health (MH), physical health (PH), general health (GH), and health satisfaction (HS). MH, PH, and GH outcomes are measured using the SF-36 questionnaire, which is a widely employed health assessment tool (see Ware, 2000). Each aggregated health measure uses 36 questions to form a scale ranging from 0 to 100, where higher values indicate healthier outcomes. Lastly, we take data on self-reported HS, which is a standard subjective marker, where respondents can select a number between '0' (no satisfaction in health) and '10' (highest satisfaction in health). This variable is ordinal in structure, although it is common for empirical researchers to impose a cardinality assumption such that econometric decompositions can be performed.

4.2.3 Explanatory Variables

Our main explanatory variables are sets of grandparental SES measures (both from the mother's side and father's side). These include markers of education, occupation, whether the grandparents are divorced, and several other indicators of economic conditions. The level of education is measured using the level of schooling completion of both the grandfathers and grandmothers. Grandparents' occupational prestige is captured with a scale variable ranging from '0' to '100' and is measured according to the Australian and New Zealand standard classifications (McMillan et al., 2009). The other grandparental economic conditions are recorded with dummies indicating whether the grandfather was unemployed for at least six months while the parents were growing up and whether grandfathers or grandmothers were in paid employment when parents were aged 14 years.

Following the standard IOP model, which regresses individuals' outcomes against their parents' characteristics, we take a series of variables related to parental economic

attainments. The set of parental SES includes level of schooling completion, occupation, whether the parents are divorced, whether the father was unemployed for at least six months when the respondent was growing up, and whether the mother or father was in paid employment when the respondent was aged 14 years. The measurements of these variables are similar to those for grandparental SES.

We also include measures of parental health because these outcomes may also be transmitted across generations by non-socioeconomic channels, e.g., each individual inherits a unique set of genes from both paternal and maternal lines (Kardia et al., 2003). Also, these factors may capture the indirect effect of family economic uncertainty on children's health, e.g., transmission via poor mental health and unhealthy behaviours (A. E. Clark, D'Ambrosio, & Barazzetta, 2021; A. E. Clark, D'Ambrosio, & Rohde, 2021; Kong et al., 2021). Parental health status is proxied by both the mothers' and fathers' BMI, MH, PH, GH aggregates, and HS. Scales of these parental health variables are the same as the measures of children's health outcomes. We also control some family background markers and demographic variables: age, gender, ethnicity, living area, country of birth, and first language.

4.2.4 Summary Statistics

Table 4.1 summarises the distribution of all outcome variables and independent variables at baseline. In our study sample, most of the respondents are young (Figure A4.1 in the appendix shows that more than 67% of respondents are aged between 10 and 25 years). As shown in Table 1, the average age of individuals in the sample was approximately 13 years, whereas the respondents' maximum age was 59 years. Therefore, our work measured multigenerational socioeconomic disparities in health inequalities in children and younger individuals. As a result, our inequality estimates are likely to be relatively low as health disparities are known to increase over the lifespan (Galama & van Kippersluis, 2019).

Aside from age distribution and gender ratio, our sample is relatively representative of the Australian population. Our observations include an almost equal proportion of females (49%) and males (51%). However, most respondents' born language is English (98%), and they were born domestically (96%), while approximately 1% of our sample are Indigenous and 0.3% are refugees.

Table 4.1 Descriptive Statistics

	Variable	Mean	Std. Dev.	Min	Max
Demographic	Age	13.478	6.137	0	59
	Female	0.478	0.500	0	1
	Language: English	0.987	0.112	0	1
	Refugee	0.003	0.051	0	1
	Indigenous origin	0.013	0.115	0	1
	Country of birth: Australia	0.969	0.173	0	1
	Major city	0.579	0.494	0	1
	Regional	0.400	0.490	0	1
	Remote	0.021	0.144	0	1
Health	BMI	22.660	4.169	12.8	50.9
	Distance from healthy BMI	0.965	2.329	0	25.9
	Satisfaction: health condition	8.427	1.480	0	10
	Mental health	74.623	15.930	4	100
	General health	75.506	17.874	0	100
	Physical health	92.479	19.466	0	100
GP SES: father's side	Grandfather schooling: not respond	0.985	0.122	0	1
	Primary and secondary	0.008	0.090	0	1
	Year 11 and year 12	0.007	0.084	0	1
	Grandmother schooling: not respond	0.985	0.122	0	1
	Primary and secondary	0.006	0.074	0	1
	Year 11 and year 12	0.010	0.098	0	1
	Grandfather in paid employment	0.038	0.192	0	1
	Grandmother in paid employment	0.633	0.482	0	1
	Grandparent divorced	0.079	0.270	0	1
	Grandfather unemployed	0.078	0.268	0	1
	Grandfather's occupation	43.131	21.693	0	100
	Grandmother's occupation	40.643	23.266	3.4	100
GP SES: mother's side	Grandfather in paid employment	0.003	0.055	0	1
	Grandmother in paid employment	0.623	0.485	0	1
	Grandparent divorced	0.109	0.311	0	1
	Grandfather unemployed	0.103	0.303	0	1
	Grandfather's occupation	46.133	22.549	7.9	100
	Grandmother's occupation	41.114	22.191	3.4	100
Parental SES	Father schooling: no response	0.240	0.427	0	1
	Non	0.001	0.036	0	1
	Primary and secondary	0.267	0.442	0	1
	Year 11 and year 12	0.492	0.500	0	1
	Mother schooling: no response	0.240	0.427	0	1
	Primary and secondary	0.181	0.385	0	1
	Year 11 and year 12	0.579	0.494	0	1
	Father in paid employment	0.915	0.278	0	1
	Mother in paid employment	0.847	0.360	0	1
	Parents divorced	0.025	0.155	0	1
	Father unemployed	0.127	0.333	0	1
	Father's occupation	52.907	24.146	4.9	100
	Mother's occupation	54.236	22.869	3.4	100
Parental health	BMI mother	27.159	5.911	14.8	58.2
	Mother satisfaction: health condition	7.583	1.578	0	10
	Mother physical health	89.830	15.023	0	100
	Mother general health	74.270	17.933	0	100
	Mother mental health	75.832	14.912	0	100
	BMI (father)	27.975	4.347	17.2	49.4
	Father satisfaction: health condition	7.373	1.608	0	10
	Father physical health	88.459	18.504	0	100
	Father general health	69.845	18.144	0	100
	Father mental health	76.828	15.307	0	100
	Age (mother)	43.701	7.074	20	83
	Age(father)	45.927	7.520	20	89

Note: In this table, the second column presents the variables' average values over the estimated sample, which includes 11,704 respondents. The standard deviation, minimum value and maximum value of each variable are presented from the third column to the fifth column, respectively. For binary variables, mean values refer to sample proportion with given characteristics.

4.3 A Multigenerational IOP Model

To measure IOP in health, we employed the standard parametric approach, which uses regression models to attribute variations in outcomes to a set of predetermined circumstance variables (e.g., see Brunori, Ferreira, & Peragine, 2013; Checchi & Peragine, 2010; Ferreira & Peragine, 2016; Roemer & Trannoy, 2015 for some conceptual overviews and empirical applications). Thus, the overall variation in the health indicator represents the total inequality, while the explained component represents inequality due to unequal opportunity. The unexplained (residual) term represents unobserved circumstances and individual efforts. Other demographic variables are sometimes used as controls.

A baseline IOP model for health outcome y_{it} is given below. This baseline model additively partitions inequality into contributions from parental SES (first sigma term), parental health status (second sigma term), demographics (third sigma term) and an unexplained component (captured by u_{it}). The ordinary least squares (OLS) regression estimates of Eq (4.2) are presented in Table A4.1 in the appendix.

$$y_{it} = \alpha_0 + \sum_{p=1}^{q} \gamma_{1p} PSES_{pit} + \sum_{r=1}^{s} \gamma_{2r} PHS_{rit} + \sum_{v=1}^{w} \gamma_{3v} D_{vit} + u_{it} \qquad (4.2)$$

Our augmented model appears below. As in the baseline model, α_0 is an intercept and u_{it} is the error term. Variable y_{it} represents each of our six health outcomes (BMI_{it}, BMI_{it}^{*}, MH_{it}, PH_{it}, GH_{it}, and SH_{it}) of individual i in time t; PGP_j, $\forall_j \in (1 \dots k)$ and MGP_l, $\forall_l \in (1 \dots m)$ represent paternal and maternal grandparents' SES, respectively. $PSES_p$, $\forall_p \in (1 \dots q)$, PHS_r, $\forall_r \in (1 \dots s)$, D_v, $\forall_v \in (1 \dots w)$ represent the control variables: parental SES, parental health status, and individuals' demographic factors and family background. β_1, β_2 , γ_1, γ_2, and γ_3 are parameter vectors to be estimated.

$$y_{it} = \alpha_0 + \sum_{j=1}^{k} \beta_{1j} PGP_{jit} + \sum_{l=1}^{m} \beta_{2l} MGP_{lit} + \sum_{p=1}^{q} \gamma_{1p} PSES_{pit} + \sum_{r=1}^{s} \gamma_{2r} PHS_{rit} + \sum_{v=1}^{w} \gamma_{3v} D_{vit} + u_{it} \qquad (4.3)$$

The model specified in Eq (4.3) is fitted to our multigenerational dataset, and the results are reported in Table 4.2.

Table 4.2 Effect of Circumstances on Individual Health: Regressions Coefficients

Variable	BMI	BMI*	HS	MH	GH	PH
Grandparental SES: father's side						
Grandfather education	0.125**	-0.067*	-0.026	0.151	-0.367	-0.074
Grandmother education	-0.309***	-0.054	0.075**	0.143	0.882***	0.015
Grandfather in paid employment	0.733	0.467	-0.032	1.496	-0.106	5.646***
Grandmother in paid employment	-0.078	-0.071	-0.052	-2.029**	-0.568	-0.783
Grandparent divorced	0.014	0.268	0.105	-0.482	3.234	2.049
Grandfather unemployed	0.792	0.565*	-0.572***	-3.936*	-6.341***	-0.773
Grandfather's occupation	-0.015**	-0.005	0.005**	0.001	0.006	0
Grandmother's occupation	0.009	0.001	0.001	-0.009	-0.008	-0.026
Grandparental SES: mother's side						
Grandfather in paid employment	0.957	-0.497	0.603**	2.833	13.092***	2.493
Grandmother in paid employment	-0.118	-0.028	-0.125	-0.337	-0.819	0.561
Grandparent divorced	0.113	-0.037	0.147	0.35	2.011	-2.093
Grandfather unemployed	-0.105	-0.197	0.122	3.155**	2.849*	3.317*
Grandfather's occupation	-0.004	-0.003	0.002	0.005	0.015	0.012
Grandmother's occupation	0.012*	0.006	0	0.012	-0.033	-0.039
Parental SES						
[(a)]Father schooling: non	-5.074***	-1.503**	0.082	8.509**	20.896***	8.864*
Primary and secondary	-0.663	-0.266	0.456	6.356**	7.573**	4.028
Year 11 and year 12	-0.995	-0.401	0.31	7.914***	7.370**	8.521**
[(a)]Mother schooling: primary and secondary	-0.379	-0.135	-0.093	-2.802	-2.696	-5.83
Year 11 and year 12	0.317	0.112	-0.247	-4.310*	-5.118	-7.266**
Father in paid employment	0.286	-0.019	-0.041	-0.982	-1.865	-3.145*
Mother in paid employment	-0.565	-0.425*	-0.005	-1.176	0.279	-0.199
Parents divorced	1.483*	0.481	-0.622	-7.031*	-9.469**	-1.245
Father unemployed	-0.416	-0.228	-0.159	-3.400**	-1.054	-3.941
Father's occupation	-0.002	-0.005	0.002	-0.008	0.031	0
Mother's occupation	-0.013**	-0.005	0.001	0.011	-0.002	0.029
Parental health						
BMI mother	0.150***	0.059***	-0.004	0.005	-0.112	-0.155
Mother satisfaction: health condition	0.028	-0.015	-0.005	0.082	-0.283	0.147
Mother physical health	-0.008	-0.007**	0.001	0.01	-0.001	0.015
Mother general health	0.011*	0.006*	0.007***	0.054**	0.104***	-0.002
Mother mental health	0.001	0.001	0.009***	0.145***	0.149***	0.011
BMI (father)	0.267***	0.114***	-0.011	-0.004	-0.153	-0.256*
Father satisfaction: health condition	0.09	0.022	0.045**	0.195	0.151	0.627*
Father physical health	-0.007	0	0	0.015	0.007	-0.007
Father general health	0.003	0	-0.001	0.007	0.064**	-0.034
Father mental health	-0.003	-0.002	0	0.065***	-0.019	0.009
Age (mother)	0.086**	0.067***	-0.007	-0.165	-0.122	0.177
Age (father)	-0.079**	-0.074***	0.008	0.172	0.044	-0.216
Demographic factors						
Age	0.019	-0.005	0.004	0.233	0.357*	0.433**
Age2	0.002	0.001	-0.001**	-0.011**	-0.011*	-0.006*
Female	0.27	0.101	-0.440***	-4.026***	-4.629***	-0.65
Refugee	-2.921*	-1.206**	-0.76	-11.71	1.582	-1.791
Indigenous origin	0.241	-0.61	-0.224	-8.298	-10.467**	-3.906
Area of living	0.32	0.126	0.022	0.532	0.931	-0.889
Born in Australia	-0.501	-0.686	-0.346	-0.049	2.338	0.656
English	1.251	1.066	-0.193	-0.9	-5.218	-5.276**
Constant	10.335***	-2.656**	7.827***	51.863***	66.214***	105.144***
R-squared	0.258	0.172	0.124	0.115	0.112	0.051
N	11337	11337	11704	11519	11519	11519

Note: * p<0.10, ** p<0.05, ***p<0.01. Table 4.2 presents regression coefficients of covariates in EQ (4.3), which considers six different health outcomes: BMI, BMI*, HS, MH, PH, and GH. We estimated those models using OLS and used heteroskedasticity-robust standard errors throughout. Reference categories are male, non-refugee, non-indigenous, born in/out of Australia, first language not English, and not responding (a).

Once the models were fitted, we used the variance of outcomes as our inequality metric. This measure is proportional to the squared coefficient of variation, which is a member of the additively decomposable index below.

$$I_\alpha(y) = \frac{1}{N\alpha(\alpha-1)} \sum_{i=1}^{N} \left[\left(\frac{y_i}{\hat{y}} \right)^\alpha - 1 \right] \qquad (4.4)$$

Here $I_\alpha(y)$ is the health inequality index and α a weighting parameter, which sets the index equal to half the squared coefficient of variation when $\alpha = 2$. We can model the fraction of total inequality explained by our model covariates using the ratio $I_\alpha(\hat{y})/I_\alpha(y)$, The advantage of this measure is that this ratio is equal to the R^2 term from a regression model used to estimate $\hat{y}$ (i.e., the fitted values from Eq [4.2] and [4.3]).

We can, therefore, use the coefficient of determination terms reported in Table 4.2 to identify the overall fractions of inequality captured by our parameters. The results show that our models explain 5%–26% of inequality in our health makers. The highest values belong to the BMI (approximately 26%), while the general physical health variable had an explained component of only 5%. The SF-36 indicators of MH and GH had about 11% of their variation captured by our models.

The results from Table 4.2 indicate that grandparental SES is an important predictive variable across a spectrum of health outcomes. Alongside traditional determinants, such as parental health and education, grandparental educational and employment status variables are significant in a number of regressions. This is especially true for grandfathers, which suggests an economic (rather than caregiving) channel may be responsible. There are estimates of offsetting signs for grandmothers and grandfathers in some instances, which is consistent with collinearity between grandparents' SESs (see Table A4.2). For this reason, we recommend a focus on the aggregate effect rather than on individual covariates. Moreover, we test grandparents' effect while controlling parental SES and health status and show that grandparents' impact is not mediated by parental status (see Tables A 4.3, A 4.4, A 4.5, A 4.6, A 4.6, A 4.7 and A 4.8 in the appendix).

4.4 The Contribution of Grandparents to Inequality of Opportunity in Health

In order to boil down aggregate contributions from each set of covariates in Eq (4.3), we employed Owen's (1977) decomposition of the R-squared term. This econometric approach is a relative of the Shapley value decomposition (Chantreuil & Trannoy, 2013;

Shapley, 1953) and is useful for dealing with clusters of related variables within a single model. In our case, the Owen index improved the decomposition because it satisfied several important theoretical properties, including the symmetric treatment of variable subgroups (Khmelnitskaya & Yanovskaya, 2007). Moreover, considering these properties of Owen value decomposition, Huettner & Sunder (2011) suggested that the Owen index is most suitable if at least a subgroup has more than one exogenous variable.

In Eq (4.3), we employed 42 (K) exogenous variables divided into four subgroups (G) representing grandparental SES, parental SES, parental health status and demographic characteristics. A permutation π is compatible with G if variables in each group arrange in the permutation contiguously. So, the Owen index (OW_j) for calculating the decomposition of total explained inequality (R^2) in health outcomes $\left(H_j^m, \forall m \in (1 \dots \dots . m)\right)$ is given by

$$H_j^m = OW_j(K, R^2, G) = \frac{1}{|\Pi(K.G)|} \sum_{\pi \in \Pi(K,G)} R^2\left(P_j^\pi\right) - R^2\left(P_j^\pi \backslash \{j\}\right) \qquad (4.5)$$

Here, $R^2\left(P_j^\pi\right) - R^2\left(P_j^\pi \backslash \{j\}\right)$ is a marginal contribution of *j's* variable when variables appear as $\pi \in \Pi(K, G)$ in the model. Table 4.3 presents the calculated decomposition of predetermined inequality in health by groups of circumstances.

Table 4.3 Decomposition of Explained Inequality in Health

Health outcome	Source of predetermined inequality	Contribution (%)	Confidence interval (95%)	
			Lower	Upper
BMI	Grandparents SES	7.906	5.501	10.962
	Parents SES	8.413	6.232	11.214
	Parents health	62.492	56.830	67.779
	Demographic factors	20.295	16.191	25.126
BMI*	Grandparents SES	8.481	5.481	12.795
	Parents SES	10.664	7.859	14.470
	Parents health	60.094	53.082	66.177
	Demographic factors	19.487	14.756	24.840
HS	Grandparents SES	17.917	11.960	24.072
	Parents SES	11.716	7.595	16.392
	Parents health	35.678	28.313	43.330
	Demographic factors	33.562	26.253	41.038
MH	Grandparents SES	9.960	6.161	14.530
	Parents SES	13.055	8.561	18.382
	Parents health	52.618	44.623	61.300
	Demographic factors	23.395	15.638	32.048
PH	Grandparents SES	29.293	17.380	45.319
	Parents SES	23.137	12.539	35.832
	Parents health	30.924	18.971	45.287
	Demographic factors	13.615	6.226	24.806
GH	Grandparents SES	19.874	13.535	26.469
	Parents SES	9.534	5.607	14.758
	Parents health	49.933	41.897	57.751
	Demographic factors	20.082	14.104	27.423

Note: Column Three in this table presents the percentage of each source of circumstances' contribution to the total predetermined inequality (R-squared), obtained from 2000 bootstrapped samples with a 95% significant level. For the decomposition analysis, we considered the six regression estimates of our health outcomes. These models are the same as those present in Table 2—refer there
for the R-squared value of each estimated model.

Although our regression estimates (see Table 4.2) indicate a small impact of grandparental SES on health, decomposition calculations highlight that ignoring this grandparental effect may lead to an underestimation of the IOP measure. The decomposition results in Table 3 show that grandparental SES is an equally (or more) important factor of health inequality as the effect of parents' SES; for example, grandparental SES contributes 8–29% for IOP in PH measures. In comparison, the contribution of parental SES for the same inequality measure ranges 8%–23%. Moreover, grandparental and parental SES are responsible for 10% and 13% of IOP in MH outcomes, respectively.

Considering parental health status, the decomposition results in Table 4.3 show that the contribution to IOP in health is significantly higher than other sources of circumstances. These values range between approximately 31% and 62% for the different health

outcomes. This result is plausible because, on the one hand, health transmission across generations mostly occurs via genetic interaction (Thompson, 2014). On the other hand, a considerable amount of health inequality is explained by genetic traits (Classen & Thompson, 2016; Moll et al., 1991). The demographic factors and other family background markers account for approximately 20%–34% of the total IOP in health.

4.5 Discussion

Given that we found that grandparental factors account for a substantial fraction of predetermined inequality in health, there is some value in identifying plausible causal mechanisms that account for this result. Here we return to the two potential explanations outlined in the introduction: (a) the effects of grandparental caregiving and (b) the potential effects of cultural attitudes associated with social class.

Caregiving

Correlations between grandparental SES and child health may appear if higher-status grandparents offer better care compared to lower-status grandparents. To create the patterns observed in our data, such an effect needs to be direct and not operate via an intermediate channel, such as parental behaviour. However, considerable evidence exists suggesting that such a direct effect exists. For example, grandparental caregiving is known to positively impact their grandchildren's survival, physical growth and protection against physical injury (Li & Liu, 2019; Rogers et al., 2019). Grandparents can also provide informal medical advice and health-related economic resources that do not flow through parents, such as diagnosing illness (Hillman et al., 2017) and providing money for doctors and treatments (Hillman et al., 2016). Evidence also exists that suggests the influence of grandparental caregiving can be negative, as when unhealthy food habits and behaviours are promoted (Pulgaron et al., 2016; Young et al., 2018). The potential for negative effects to be transmitted through social channels like intrafamilial conflict (between parents and grandparents) also exists and may be a source of stress and, therefore, lead to diminished health.

To be able to explain our results, these tendencies need to be more pronounced (in a positive or beneficial sense) for grandparents with higher educational attainments. Such a link is highly plausible, as when higher SES grandparents are less likely to be absent and, therefore, have a greater capacity to play a caring role (Luo et al., 2012). In addition, education is usually a predictor of better health behaviours (Cowell, 2006), which would likely spill over into better caring skills.

Cultural Factors

Our second proposed explanation is that our results might reflect the latent effects of social class. The key idea here is that families that have held a higher status for longer may take on cultural attitudes that are middle or upper class, while families that have been poorer for multiple generations may exhibit working-class cultural norms. This may be because either individuals' childhood SES is positively associated with a level of education and employment (Lynch et al., 1997) or because lifestyle behaviours and attitudes are transmitted across multiple generations (Arroyo et al., 2017; Chadi et al., 2021; Gauly, 2017). Notably, the class effect is separate from the parental SES effect because ingrained cultural attitudes are distinct from purely economic variables, such as income. Thus, some children may grow up in a relatively affluent environment with cultural attitudes reminiscent of working-class families, while others may be exposed to cultural norms that are predicted by their parents' incomes or education levels (Goldthorpe & Lockwood, 1963; Gugushvili et al., 2019; Manstead, 2018)

Objective resources (such as educational achievements, occupational prestige and wealth) of the social class create cultural identities among the working class and middle or upper social class individuals (Kraus et al., 2011). From childhood, individuals in different social classes experience different material life cultures: different neighbourhoods and peers, belonging to different educational institutions and social clubs, engaging in different recreational events, eating different food, and enjoying fashion with different brands. Individuals from affluent cultural backgrounds also have relatively greater opportunities to make choices, stand up for their rights and live in a secure environment where they are better able to acquire basic needs (Manstead, 2018).

These cultural differences between working class and middle or upper social class individuals may feed through to health behaviours. If decisions to smoke (Baška et al., 2010; Ong et al., 2017), binge drink (Van Wersch & Walker, 2009), overeat (Hulshof et al., 2003) or engage in substance abuse (Lee et al., 2015) are informed by culture, then we would expect to see greater associated health problems in these segments of the population (Dressler et al., 2014; Lynch et al., 1997). Other potential causal channels such as psychological stress or depression due to childhood status (Morrissey & Kinderman, 2020), job uncertainty (Watson & Osberg, 2018), working environment (Lepinteur, 2019) or violent behaviour (Heimer, 1997), may also be present.

Peer Effects

Grandparents may also affect their grandchildren's health by influencing neighbourhood and peer effects. Here, the causal mechanism is that grandparents may provide additional resources that affect the social groups of their grandchildren, which then go on to affect behaviours. For example, high SES grandparents may shape their grandchildren's peer groups by influencing educational choices through their social, cultural and economic capital (Møllegaard & Jæger, 2015). Moreover, since grandparental SES influences educational and employment success (Anderson et al., 2018; Dribe & Helgertz, 2016), relationships built up through individuals' SES may be partially influenced by inherited childhood background.

The distinction between neighbourhoods and peer exposure, indicative of social-class culture during childhood, generates subtle attitudinal and behavioural characteristics associated with individual health outcomes (Chetty & Hendren, 2018; Evans et al., 2016). Differences in behaviours are also associated with peer behaviour; for example, adolescent smoking is motivated by friends' smoking behaviour (Hoffman et al., 2006), and children's food intake is influenced by friends' food intake (Fortin & Yazbeck, 2015). Likewise, some important aspects of the neighbourhood, such as access to common built facilities (recreational or leisure parks, physical activity establishments; Carroll-Scott et al., 2013), neighbourhood socioeconomic background (Jivraj et al., 2020), and unsafe neighbourhood environments (Galaviz et al., 2016) can be responsible for the variation in childhood experiences that drive individuals' health behaviours, attitudes and psychological traits.

4.6 Conclusion

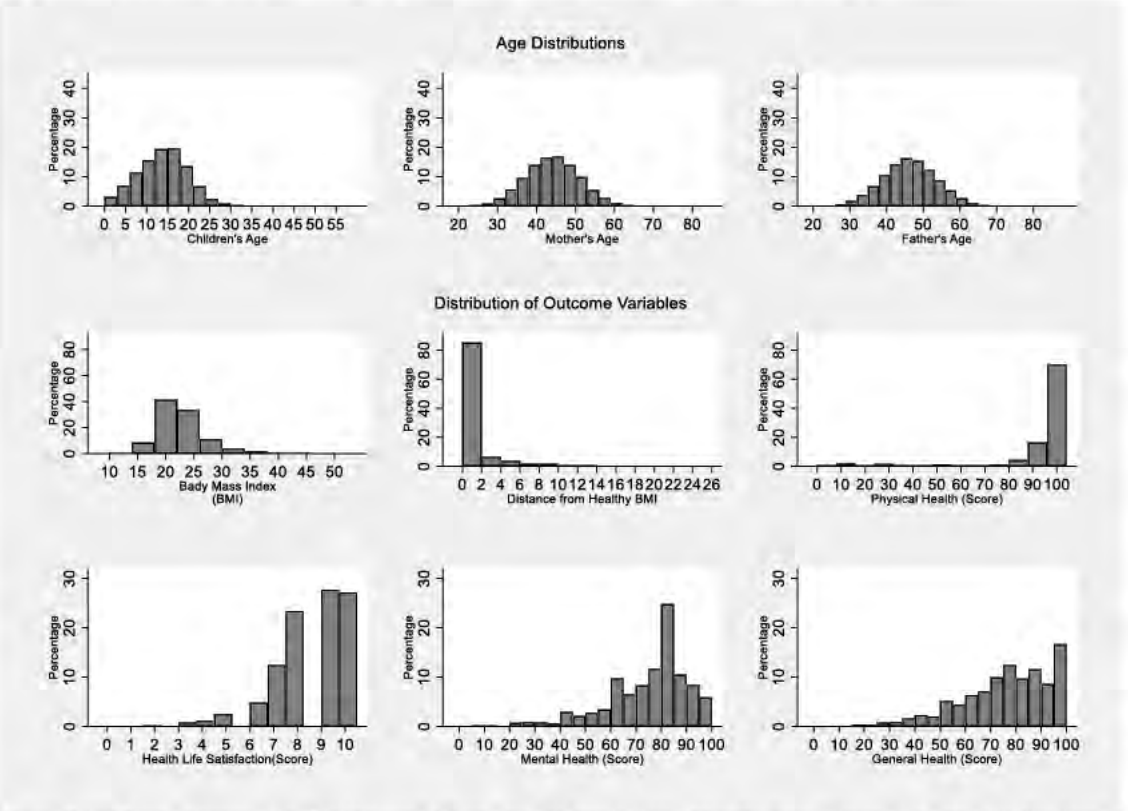
This chapter has studied multigenerational IOP in health using Australian data. We showed that, across a spectrum of MH and PH outcomes, markers of grandparental SES predict grandchildren's health outcomes, even after controlling for parents' equivalent socioeconomic characteristics. We then performed some econometric decompositions that attribute explained inequalities to various clusters of variables related to intergenerational inequalities. Our results are surprising because they place approximately equal weight on the contributions of parents and grandparents, which suggests that more complicated causal flows are present beyond those implicit in standard intergenerational inequality models.

We speculate two possible explanations for this result. Firstly, grandparents may play an important role in caregiving, where more educated grandparents may do a better job raising healthier children. For example, these grandparents may be more likely to be present within the family (and add to the stock of caring resources available) or be better suited to identifying or treating health complaints. They may also provide financial resources to their grandchildren that do not flow directly through the parents. Secondly, we argue that grandparents of higher SES may generate different cultural attitudes in ways reminiscent of social class. These attitudes may feed through to affect various behaviours, such as tobacco or alcohol consumption.

Lastly, our results have some general implications for the measurement of IOP. IOP models, which are typically lower-bound estimates because socioeconomic constraints are only partially observable, commonly produce estimates that seem too low. Our decompositions indicate that neglecting multigenerational factors may explain some of this missing inequality.

Appendix

Figure A 4.1 Distributions of Age and Outcome Variables – HILDA Sample



Note: This figure presents the histograms of two sets of variables. The first part shows the age distributions of respondents and their parents; the second presents the distributions of outcome variables. Data from the estimation sample (n=11,704) has been used throughout.

Table A 4.1 Regression Results of Standard IOP model

	BMI	BMI*	Satisfaction: health condition	Mental health	General health	Physical health
Parental SES						
[(a)]Father schooling: non	-4.902***	-1.288**	0.278	10.973***	23.072***	13.237***
Primary and secondary	-0.818	-0.308	0.474	6.657**	7.679**	4.019
Year 11 and year 12	-1.146	-0.438	0.342	8.105***	7.556**	8.753**
[(a)]Mother schooling:						
Primary and secondary	-0.246	-0.12	-0.088	-2.862	-2.47	-5.991*
Year 11 and year 12	0.431	0.119	-0.258	-4.586*	-5.244	-7.569**
Father in paid empl	0.182	-0.073	0.014	-0.784	-1.65	-3.138*
Mother in paid empl	-0.537	-0.427*	0.004	-1.643	0.302	-0.494
Parents divorced	1.586*	0.508	-0.577	-6.889*	-8.494**	-0.868
Father unemployed	-0.308	-0.167	-0.176	-3.277**	-1.069	-3.174
Father's occupation	-0.004	-0.006*	0.004*	-0.007	0.028	-0.007
Mother's occupation	-0.011*	-0.005	0.002	0.017	0.002	0.023
Parental health						
BMI mother	0.154***	0.060***	-0.006	0.02	-0.104	-0.146
Mother satisfaction: health condition	0.023	-0.016	0.002	0.117	-0.193	0.153
Mother physical health	-0.010*	-0.009**	0.001	0.017	0.007	0.022
Mother general health	0.012**	0.007**	0.006***	0.049**	0.099***	-0.001
Mother mental health	0.002	0.001	0.009***	0.140***	0.146***	0.005
BMI (father)	0.264***	0.112***	-0.01	0.003	-0.129	-0.238
Father satisfaction: health condition	0.076	0.009	0.046**	0.246	0.159	0.621*
Father physical health	-0.009	-0.002	0.001	0.021	0.02	-0.009
Father general health	0.005	0.001	-0.002	0.003	0.058**	-0.039
Father mental health	-0.006	-0.003	0	0.068***	-0.008	0.02
Age (mother)	0.084**	0.065***	-0.002	-0.146	-0.099	0.178
Age (father)	-0.084**	-0.075***	0.005	0.184	0.031	-0.212
Demographic factors						
Age	0.017	-0.006	0.005	0.225	0.383*	0.437**
Age2	0.003*	0.001	-0.001**	-0.011**	-0.012*	-0.007*
Female	0.323	0.13	-0.457***	-4.135***	-4.758***	-0.827
Refugee	-3.305**	-1.508***	-0.419	-8.871	4.913	-0.771
Indigenous origin	0.227	-0.568	-0.287	-8.121	-10.404**	-3.787
Area of living	0.334	0.126	0.008	0.613	0.868	-0.777
Born in Australia	-0.345	-0.667	-0.385	0.008	2.213	-0.727
English	0.896	0.958	-0.008	0.477	-2.732	-3.064
Constant	11.309***	-2.090*	7.491***	46.423***	58.434***	101.887***
R-squared	0.239	0.156	0.102	0.102	0.093	0.041
N	11337	11337	11704	11519	11519	11519

Note: * p<0.10, ** p<0.05, ***p<0.01. Table A 4.1 presents regression coefficients of covariates in Eq (2), which considers six different health outcomes: BMI, BMI*, satisfaction with health, mental health, general health and physical health. We estimated those models using OLS and used heteroskedasticity-robust standard errors throughout. Reference categories are male, non-refugee, non-indigenous, born out of Australia, and first language not English, and not respond (a)

Table A 4.2 Correlation Matrix: Grandparental Characteristics

	GFE	GME	GFPE	GMPE	GPD	GFU	GFO	GMO
Father's side								
GFE	1							
GME	0.613	1						
GFPE	0.018	0.091	1					
GMPE	-0.003	0.01	-0.016	1				
GPD	-0.003	-0.008	-0.048	0.082	1			
GFU	-0.001	0.009	0.02	-0.019	0.109	1		
GFO	0.013	-0.002	-0.003	-0.078	0.022	-0.046	1	
GMO	0.017	0.032	0.008	0.03	0.032	0.017	0.359	1
Mother's side								
GFE	1							
GME	0.105	1						
GFPE	0.022	0.505	1					
GMPE	0.024	0.006	0.018	1				
GPD	0.011	0.012	-0.016	0.061	1			
GFU	0.009	0.002	-0.016	-0.039	0.072	1		
GFO	0.002	-0.018	-0.012	-0.018	-0.003	-0.088	1	
GMO	0.009	-0.004	0.008	0.063	-0.026	-0.084	0.392	1

Note: This table presents the correlation coefficients between grandparental characteristics. Abbreviations: grandfather education (GFE), grandmother education (GME), grandfather in paid employment (GFPE), grandmother in paid employment (GMPE), grandparent divorced (GPD), grandfather unemployed (GFU), grandfather's occupation (GFO) and grandmother's occupation (GMO).

Table A 4.3 Effect of Circumstances on BMI: Regressions Coefficients

	DV: BMI				
Variable	M1	M2	M3	M4	M5
Grandparental SES: father's side					
Grandfather education	0.142***			0.127**	0.125**
Grandmother education	-0.369***			-0.324***	-0.309***
Grandfather in paid employment	0.673			0.701	0.733
Grandmother in paid employment	-0.192			-0.108	-0.078
Grandparent divorced	0.37			0.11	0.014
Grandfather unemployed	0.912			0.785	0.792
Grandfather's occupation	-0.019***			-0.017***	-0.015**
Grandmother's occupation	-0.001			0.009*	0.009
Grandparental SES: mother's side					
Grandfather in paid employment	3.344***			0.871	0.957
Grandmother in paid employment	-0.019			-0.133	-0.118
Grandparent divorced	0.231			0.069	0.113
Grandfather unemployed	0.265			-0.131	-0.105
Grandfather's occupation	-0.017**			-0.004	-0.004
Grandmother's occupation	0.012			0.011*	0.012*
Parental SES					
[(a)]Father schooling: non		-5.578***		-5.238***	-5.074***
Primary and secondary		-0.602		-0.791	-0.663
Year 11 and year 12		-1.378		-1.252*	-0.995
[(a)]Mother schooling: primary and secondary		0		0	0
Year 11 and year 12		-0.532		-0.39	-0.379
Father in paid employment		-0.109		0.33	0.317
Mother in paid employment		-0.126		0.126	0.286
Parents divorced		-0.777*		-0.488	-0.565
Father unemployed		0.837		1.396*	1.483*
Father's occupation		-0.182		-0.6	-0.416
Mother's occupation		-0.009		-0.004	-0.002
Parental health					
BMI mother			0.157***	0.151***	0.150***
Mother satisfaction: health condition			0.066	0.023	0.028
Mother physical health			-0.014**	-0.009	-0.008
Mother general health			0.01	0.011*	0.011*
Mother mental health			0.004	0.002	0.001
BMI (father)			0.281***	0.271***	0.267***
Father satisfaction: health condition			0.074	0.079	0.09
Father physical health			-0.012**	-0.007	-0.007
Father general health			0.007	0.005	0.003
Father mental health			-0.006	-0.002	-0.003
Age (mother)			0.139***	0.133***	0.086**
Age (father)			-0.077**	-0.070*	-0.079**
Demographic factors					
Age					0.019
Age2					0.002
Female					0.27
Refugee					-2.921*
Indigenous origin					0.241
Area of living					0.32
Born in Australia					-0.501
English					1.251
Constant	23.750***	25.956***	8.183***	10.055** *	10.335** *
R-squared	0.038	0.054	0.196	0.245	0.258
N	11337	11337	11337	11337	11337

Note: * p<0.10, ** p<0.05, ***p<0.01. This Table presents coefficients of the regression of BMI on only grandparents' characteristics (M1), only parents' SES (M2), only parents' health outcomes (M3), then grandparents' and parents' characteristics (SES and health status) together (M4), and finally regression coefficients of the complete model with demographic factors are presented (M5). We estimated those models using OLS and used heteroskedasticity-robust standard errors throughout. Reference categories are male, non-refugee, non-indigenous, born in/out of Australia, first language not English, and not responding (a).

Table A 4.4 Effect of Circumstances on BMI*: Regressions Coefficients

	DV: BMI*				
Variable	M1	M2	M3	M4	M5
Grandparental SES: father's side					
Grandfather education	-0.058			-0.064*	-0.067*
Grandmother education	-0.086			-0.062	-0.054
Grandfather in paid employment	0.533			0.486	0.467
Grandmother in paid employment	-0.104			-0.078	-0.071
Grandparent divorced	0.464			0.321	0.268
Grandfather unemployed	0.642*			0.572*	0.565*
Grandfather's occupation	-0.006*			-0.005	-0.005
Grandmother's occupation	-0.002			0.002	0.001
Grandparental SES: mother's side					
Grandfather in paid employment	0.472			-0.562	-0.497
Grandmother in paid employment	0.011			-0.034	-0.028
Grandparent divorced	0.006			-0.076	-0.037
Grandfather unemployed	0.019			-0.191	-0.197
Grandfather's occupation	-0.009**			-0.002	-0.003
Grandmother's occupation	0.006			0.005	0.006
Parental SES					
[(a)]Father schooling: non		-1.479**		-1.601**	-1.503**
Primary and secondary		-0.207		-0.3	-0.266
Year 11 and year 12		-0.51		-0.498	-0.401
[(a)]Mother schooling: primary and secondary		-0.227		-0.146	-0.135
Year 11 and year 12		-0.077		0.117	0.112
Father in paid employment		-0.178		-0.104	-0.019
Mother in paid employment		-0.530*		-0.404*	-0.425*
Parents divorced		0.262		0.46	0.481
Father unemployed		-0.099		-0.307	-0.228
Father's occupation		-0.007**		-0.005*	-0.005
Mother's occupation		-0.009**		-0.006	-0.005
Parental health					
BMI mother			0.062***	0.058***	0.059***
Mother satisfaction: health condition			0.011	-0.016	-0.015
Mother physical health			-0.011***	-0.008**	-0.007**
Mother general health			0.006	0.006*	0.006*
Mother mental health			0.003	0.002	0.001
BMI (father)			0.123***	0.117***	0.114***
Father satisfaction: health condition			0.014	0.019	0.022
Father physical health			-0.004	-0.001	0
Father general health			0.002	0.001	0
Father mental health			-0.003	-0.002	-0.002
Age (mother)			0.085***	0.086***	0.067***
Age (father)			-0.072***	-0.070***	-0.074***
Demographic factors					
Age					-0.005
Age2					0.001
Female					0.101
Refugee					-1.206**
Indigenous origin					-0.61
Area of living					0.126
Born in Australia					-0.686
English					1.066
Constant	1.478***	2.860***	-3.898***	-2.854**	-2.656**
R-squared	0.032	0.044	0.121	0.162	0.172
N	11337	11337	11337	11337	11337

Note: * p<0.10, ** p<0.05, ***p<0.01. This Table presents coefficients of the regression of BMI* on only grandparents' characteristics (M1), only parents' SES (M2), only parents' health outcomes (M3), then grandparents' and parents' characteristics (SES and health status) together (M4), and finally regression coefficients of the complete model with demographic factors are presented (M5). We estimated those models using OLS and used heteroskedasticity-robust standard errors throughout. Reference categories are male, non-refugee, non-indigenous, born in/out of Australia, first language not English, and not responding (a).

Table A 4.5 Effect of Circumstances on HS: Regressions Coefficients

	DV: HS				
Variable	M1	M2	M3	M4	M5
Grandparental SES: father's side					
Grandfather education	-0.022			-0.022	-0.026
Grandmother education	0.100***			0.079***	0.075**
Grandfather in paid employment	-0.073			0.046	-0.032
Grandmother in paid employment	-0.058			-0.044	-0.052
Grandparent divorced	0.035			0.062	0.105
Grandfather unemployed	-0.539***			-0.565***	-0.572***
Grandfather's occupation	0.006***			0.005***	0.005**
Grandmother's occupation	0.002			0.001	0.001
Grandparental SES: mother's side					
Grandfather in paid employment	0.327*			0.585**	0.603**
Grandmother in paid employment	-0.117			-0.103	-0.125
Grandparent divorced	0.144			0.111	0.147
Grandfather unemployed	0.086			0.14	0.122
Grandfather's occupation	0.002			0.002	0.002
Grandmother's occupation	-0.001			0.000	0.000
Parental SES					
[(a)]Father schooling: non		0.870***		0.273	0.082
Primary and secondary		0.656**		0.536*	0.456
Year 11 and year 12		0.600**		0.457	0.31
[(a)]Mother schooling: primary and secondary		-0.175		-0.137	-0.093
Year 11 and year 12		-0.3		-0.299	-0.247
Father in paid employment		0.089		-0.034	-0.041
Mother in paid employment		0.035		-0.038	-0.005
Parents divorced		-0.519		-0.594	-0.622
Father unemployed		-0.232		-0.133	-0.159
Father's occupation		0.003		0.002	0.002
Mother's occupation		0.003		0.002	0.001
Parental health					
BMI mother			-0.003	-0.003	-0.004
Mother satisfaction: health condition			-0.001	-0.001	-0.005
Mother physical health			0.003*	0.002	0.001
Mother general health			0.006***	0.007***	0.007***
Mother mental health			0.009***	0.009***	0.009***
BMI (father)			-0.016*	-0.012	-0.011
Father satisfaction: health condition			0.060***	0.052**	0.045**
Father physical health			0.002	0.001	0.000
Father general health			-0.003	-0.002	-0.001
Father mental health			0.000	-0.001	0.000
Age (mother)			-0.019	-0.025*	-0.007
Age (father)			-0.001	0.003	0.008
Demographic factors					
Age					0.004
Age2					-0.001**
Female					-0.440***
Refugee					-0.76
Indigenous origin					-0.224
Area of living					0.022
Born in Australia					-0.346
English					-0.193
Constant	8.159***	7.774***	7.960***	7.724***	7.827***
R-squared	0.031	0.025	0.047	0.089	0.124
N	11704	11704	11704	11704	11704

Note: * p<0.10, ** p<0.05, ***p<0.01. This Table presents coefficients of the regression of HS on only grandparents' characteristics (M1), only parents' SES (M2), only parents' health outcomes (M3), and then grandparents' and parents' characteristics (SES and health status) together (M4), and finally regression coefficients of the complete model with demographic factors are presented (M5). We estimated those models using OLS and used heteroskedasticity-robust standard errors throughout. Reference categories are male, non-refugee, non-indigenous, born in/out of Australia, first language not English, and not responding (a).

Table A 4.6 Effect of Circumstances on MH: Regressions Coefficients

	DV: MH				
Variable	M1	M2	M3	M4	M5
Grandparental SES: father's side					
Grandfather education	0.23			0.195	0.151
Grandmother education	0.483			0.162	0.143
Grandfather in paid employment	0.941			2.291	1.496
Grandmother in paid employment	-2.413**			-1.869**	-2.029**
Grandparent divorced	-0.728			-0.665	-0.482
Grandfather unemployed	-4.096*			-4.161**	-3.936*
Grandfather's occupation	0.008			0.005	0.001
Grandmother's occupation	-0.002			-0.005	-0.009
Grandparental SES: mother's side					
Grandfather in paid employment	-0.316			2.531	2.833
Grandmother in paid employment	-0.665			-0.123	-0.337
Grandparent divorced	0.497			0.119	0.35
Grandfather unemployed	2.432			3.291**	3.155**
Grandfather's occupation	0.016			0.005	0.005
Grandmother's occupation	-0.005			0.011	0.012
Parental SES					
[(a)]Father schooling: non				10.594**	
		16.245***		*	8.509**
Primary and secondary		8.170***		7.187**	6.356**
Year 11 and year 12		9.785***		9.022***	7.914***
[(a)]Mother schooling: primary and secondary		-4.137		-3.543	-2.802
Year 11 and year 12		-5.650**		-5.271**	-4.310*
Father in paid employment		-0.387		-1.045	-0.982
Mother in paid employment		-1.514		-1.608	-1.176
Parents divorced		-7.344*		-6.925*	-7.031*
Father unemployed		-4.523***		-3.545**	-3.400**
Father's occupation		-0.014		-0.018	-0.008
Mother's occupation		0.027		0.019	0.011
Parental health					
BMI mother			0.047	0.008	0.005
Mother satisfaction: health condition			0.079	0.094	0.082
Mother physical health			0.029	0.018	0.01
Mother general health			0.049*	0.051**	0.054**
Mother mental health			0.140***	0.142***	0.145***
BMI (father)			-0.029	0.002	-0.004
Father satisfaction: health condition			0.402	0.218	0.195
Father physical health			0.032	0.021	0.015
Father general health			-0.009	0.004	0.007
Father mental health			0.064**	0.057**	0.065***
Age (mother)			-0.228	-0.216	-0.165
Age (father)			0.172	0.169	0.172
Demographic factors					
Age					0.233
Age2					-0.011**
Female					-4.026***
Refugee					-11.71
Indigenous origin					-8.298
Area of living					0.532
Born in Australia					-0.049
English					-0.9
				51.625**	51.863**
Constant	75.765***	73.341***	48.655***	*	*
R-squared	0.018	0.032	0.051	0.091	0.115
N	11519	11519	11519	11519	11519

Note: * p<0.10, ** p<0.05, ***p<0.01. This Table presents coefficients of the regression of MH on only grandparents' characteristics (M1), only parents' SES (M2), only parents' health outcomes (M3), and then grandparents' and parents' characteristics (SES and health status) together (M4), and finally regression coefficients of the complete model with demographic factors are presented (M5). We estimated those models using OLS and used heteroskedasticity-robust standard errors throughout. Reference categories are male, non-refugee, non-indigenous, born in/out of Australia, first language not English, and not responding (a).

Table A 4.7 Effect of Circumstances on GH: Regressions Coefficients

	DV: GH				
Variable	M1	M2	M3	M4	M5
Grandparental SES: father's side					
Grandfather education	-0.39			-0.321	-0.367
Grandmother education	1.188***			0.908***	0.882***
Grandfather in paid employment	-0.146			0.656	-0.106
Grandmother in paid employment	-0.636			-0.188	-0.568
Grandparent divorced	2.45			2.932	3.234
Grandfather unemployed	-6.193***			-6.648***	-6.341***
Grandfather's occupation	0.013			0.011	0.006
Grandmother's occupation	0.004			-0.002	-0.008
Grandparental SES: mother's side					
Grandfather in paid employment	8.643***			12.361**	13.092**
Grandmother in paid employment	-1.148			-0.608	-0.819
Grandparent divorced	2.269			1.686	2.011
Grandfather unemployed	2.183			3.296**	2.849*
Grandfather's occupation	0.029			0.016	0.015
Grandmother's occupation	-0.046*			-0.03	-0.033
Parental SES					
[(a)]Father schooling: non		29.296***		23.517**	20.896**
Primary and secondary		9.000**		8.267**	7.573**
Year 11 and year 12		9.201**		8.249**	7.370**
[(a)]Mother schooling: primary and secondary		-4.465		-3.945	-2.696
Year 11 and year 12		-6.560*		-6.643*	-5.118
Father in paid employment		-0.994		-1.801	-1.865
Mother in paid employment		0.68		-0.414	0.279
Parents divorced		-8.642*		-9.386**	-9.469**
Father unemployed		-2.546		-1.214	-1.054
Father's occupation		0.016		0.015	0.031
Mother's occupation		0.011		0.005	-0.002
Parental health					
BMI mother			-0.081	-0.11	-0.112
Mother satisfaction: health condition			-0.214	-0.262	-0.283
Mother physical health			0.015	0.007	-0.001
Mother general health			0.099***	0.102***	0.104***
Mother mental health			0.144***	0.146***	0.149***
BMI (father)			-0.118	-0.142	-0.153
Father satisfaction: health condition			0.316	0.2	0.151
Father physical health			0.028	0.016	0.007
Father general health			0.044	0.058**	0.064**
Father mental health			-0.01	-0.027	-0.019
Age (mother)			-0.113	-0.126	-0.122
Age (father)			0.054	0.071	0.044
Demographic factors					
Age					0.357*
Age2					-0.011*
Female					-4.629***
Refugee					1.582
Indigenous origin					-10.467**
Area of living					0.931
Born in Australia					2.338
English					-5.218
Constant	76.174***	72.594***	58.310***	62.418**	66.214**
R-squared	0.023	0.021	0.052	0.089	0.112
N	11519	11519	11519	11519	11519

Note: * p<0.10, ** p<0.05, ***p<0.01. Table A 4.7 presents coefficients of the regression of GH on only grandparents' characteristics (M1), only parents' SES (M2), only parents' health outcomes (M3), and then grandparents' and parents' characteristics (SES and health status) together (M4), and finally regression coefficients of the complete model with demographic factors are presented (M5). We estimated those models using OLS and used heteroskedasticity-robust standard errors throughout. Reference categories are male, non-refugee, non-indigenous, born in/out of Australia, first language not English, and not responding (a).

Table A 4.8 Effect of Circumstances on PH: Regressions Coefficients

	DV: PH				
Variable	M1	M2	M3	M4	M5
Grandparental SES: father's side					
Grandfather education	-0.041			-0.073	-0.074
Grandmother education	0.184**			0.026	0.015
Grandfather in paid employment	5.199***			5.987***	5.646***
Grandmother in paid employment	-1.004			-0.424	-0.783
Grandparent divorced	1.606			2.045	2.049
Grandfather unemployed	-0.901			-0.491	-0.773
Grandfather's occupation	0.018			0.004	0
Grandmother's occupation	-0.009			-0.026	-0.026
Grandparental SES: mother's side					
Grandfather in paid employment	-0.37			1.679	2.493
Grandmother in paid employment	-0.24			0.551	0.561
Grandparent divorced	-2.444			-1.814	-2.093
Grandfather unemployed	2.218			3.609**	3.317*
Grandfather's occupation	0.043			0.017	0.012
Grandmother's occupation	-0.044			-0.037	-0.039
Parental SES					
[(a)]Father schooling: non		14.937***		9.989**	8.864*
Primary and secondary		3.915		4.122	4.028
Year 11 and year 12		8.910**		8.627**	8.521**
[(a)]Mother schooling: primary and secondary		-7.567**		-6.819*	-5.83
Year 11 and year 12		-8.934**		-8.422**	-7.266**
Father in paid employment		-2.808		-2.962	-3.145*
Mother in paid employment		-0.305		-0.378	-0.199
Parents divorced		-1.259		-1.43	-1.245
Father unemployed		-3.834		-4.006*	-3.941
Father's occupation		0.003		-0.002	0
Mother's occupation		0.028		0.025	0.029
Parental health					
BMI mother			-0.192*	-0.169	-0.155
Mother satisfaction: health condition			0.076	0.123	0.147
Mother physical health			0.025	0.019	0.015
Mother general health			0	-0.003	-0.002
Mother mental health			-0.002	0.007	0.011
BMI (father)			-0.259*	-0.263*	-0.256*
Father satisfaction: health condition			0.685*	0.657**	0.627*
Father physical health			0.001	-0.005	-0.007
Father general health			-0.042	-0.036	-0.034
Father mental health			0.031	0.012	0.009
Age (mother)			0.319*	0.297	0.177
Age (father)			-0.138	-0.167	-0.216
Demographic factors					
Age					0.433**
Age2					-0.006*
Female					-0.65
Refugee					-1.791
Indigenous origin					-3.906
Area of living					-0.889
Born in Australia					0.656
English					-5.276**
Constant	92.499***	95.275***	90.098***	97.421** *	105.144***
R-squared	0.01	0.024	0.018	0.047	0.051
N	11519	11519	11519	11519	11519

Note: * p<0.10, ** p<0.05, ***p<0.01. Table A 4.8 presents coefficients of the regression of PH on only grandparents' characteristics (M1), only parents' SES (M2), only parents' health outcomes (M3), and then grandparents' and parents' characteristics (SES and health status) together (M4). Finally, regression coefficients of the complete model with demographic factors are presented (M5). We estimated those models using OLS and used heteroskedasticity-robust standard errors throughout. Reference categories are male, non-refugee, non-indigenous, born in/out of Australia, first language not English, and not responding (a).

Chapter 5

Maternal Education and HIV Transmission in Sub-Saharan Africa

5.1 Introduction

HIV/AIDS is the second most deadly infectious disease, causing almost one million deaths each year; its prevalence is significantly higher in countries with higher socioeconomic inequalities (Dwyer-Lindgren et al., 2019). For instance, in Sub-Saharan Africa, where more than 70% of people living with HIV dwell (S. L. James et al., 2018), the HIV epidemic poses a significant public health risk. Although, during the 1990s, higher educational attainment and income were associated with higher HIV prevalence (Ainsworth & Semali, 1998; UNAIDS, 1998), the risk has now shifted to lower-educated disadvantaged individuals (Dwyer-Lindgren et al., 2019). One of the potential reasons for this may be a positive relationship between knowledge about HIV and socioeconomic status (see Chirwa, 2020).

Level of education influences health through various mechanistic channels. On the one hand, education improves their health because educated people may make more informed use of medical care and have healthy habits and behaviours. On the other hand, education plays a significant role in the association between childhood socioeconomic circumstances and an individual's health. Although researchers claim a strong correlation between education and health outcomes (Conti et al., 2010; Masters et al., 2012; Raghupathi & Raghupathi, 2020; Zajacova & Lawrence, 2018), they often question whether this relationship is causal; the answer is still inconclusive (Albarrán et al., 2020; Webbink et al., 2010).

This chapter's main purpose is to estimate the causal effect of HIV knowledge on HIV prevalence by using an instrumental variable (IV) regression model that employs high-quality Demographic and Health Surveys (DHSs) data from 21 Sub-Saharan countries. The estimates were derived using variations in HIV transmission awareness associated with maternal education. We find that HIV awareness/ knowledge has a causal effect on mitigating HIV/AIDS prevalence. The instrumental variable (IV) estimates show the negative coefficients for our HIV awareness variables across all models.

For these results, we claim for the intergenerational transmission of educational outcomes, where an individual born to a well-educated mother has a lower risk of contracting HIV than one born to a lower-educated mother. The economic literature has persistently reported the transmission of socioeconomic inequality across generations and

its impact on individuals' outcomes (such as health and income).[14] For instance, the mother's education is substantially associated with children's cognitive skills and development (Harding et al., 2015; Jackson et al., 2017), which may influence lifestyle behaviours, attitudes and even social mobility. Therefore, we claim that maternal education is an important determinant of controlling HIV/AIDS prevalence because individuals raised by educated mothers may be knowledgeable about the risk of unsafe sexual relationships and can easily be educated to avoid HIV.

This chapter makes a threefold contribution to new knowledge in the economic literature. Firstly, it moves from existing observational studies (see Hargreaves & Glynn, 2002) on the impact of education on HIV prevalence by exploring the causal effect of HIV knowledge. Secondly, it further generalises the evidence of the causal effect of education on human health outcomes by using an alternative instrument (e.g., maternal education attainment) for individual education. Most previous literature (Brunello et al., 2016; Gathmann et al., 2015; Kemptner et al., 2011; Kippersluis et al., 2011; Lleras-Muney, 2005; Oreopoulos, 2006; Silles, 2009, Albarrán et al., 2020; Albouy & Lequien, 2009; Clark & Royer, 2013; Courtin et al., 2019; Fletcher, 2015; Jürges et al., 2012) used compulsory schooling laws for instrumenting individuals' education attainment. In addition, schooling is instrumented by higher school availability, birth order (Park & Kang, 2008) and country-specific school reforms (Arendt, 2005; Lindeboom et al., 2009). Finally, the results shed light on the inequality of opportunity (IOP) in human infectious diseases. We reveal that individuals' predetermined factors (e.g., maternal education) are associated with HIV prevalence. Nonetheless, most of the empirical literature of IOP in health has focused on health markers related to non-communicable diseases or aggregate measures for overall physical and general health (Aizawa, 2019, 2021; Balasooriya et al., 2021; Bricard et al., 2013; Carrieri & Jones, 2018; Deutsch et al., 2018; Jusot et al., 2013; Rosa Dias, 2009, 2010; Trannoy et al., 2010).

The rest of this chapter is structured as follows. The next section describes the data sets (including sample selection and data collection procedure), variables and summary statistics. The specification of our empirical models is explained in Section 5.3. Section 5.4 shows the results of the estimated econometrics models. Section 5.5 discusses the main findings and the final section is the conclusion.

[14] The impact of predetermined factors such as parental characteristics on individuals' outcomes is discussed under IOP (see Roemer, 1998).

5.2 Data

5.2.1 DHS Data

Our data are drawn from DHSs of 21 counties in Sub-Saharan Africa from 2006 to 2019. DHSs are cross-sectional household surveys designed to choose a representative sample at the national, residential and regional levels (e.g., states) using a stratified two-stage cluster sampling setting. Enumeration areas are selected from the county's census files in the first stage. Next, a sample of households is selected from each enumeration area chosen in the second stage. The sample size is estimated based on the urban and rural population proportion and gender ratio. Then, using separate standardised questionnaires for household-level data, women's, men's and biomarkers, DHSs collect a range of information about the individual's socioeconomics, demographics, health and well-being, behaviours and family life. In addition, individuals' HIV test results are included in most surveys.

Summary statistics of the HIV-tested sample in each country are given in Table 5.1. We show that the sample proportion of HIV-positive cases approximates national-level HIV-infected percentages in each country. Table 5.1 shows that the HIV prevalence rate is highly unequal among the selected counties. For example, some countries have reported more than a 20% prevalence rate, whereas others have less than a 1% prevenance rate. Because of this variation in HIV prevalence rates across selected countries, the mean HIV prevalence rate is 5.7% in our study sample.

To conduct our analysis, we required a dataset including variables from household level, women and men characteristics for HIV-tested individuals. First, we assembled the data set by matching HIV test results records with corresponding individuals from women's and men's records. Then household member records were merged. Next, we repeated this process for each selected country. We then pooled data sets from the 21 countries listed in Table 5.1.

Table 5.1 Summary Statistics of HIV Tested Sample

Country	Year of Survey	Tested Sample	HIV Prevalence	SD (Standard Deviation)
Lesotho	2015	15640	0.275	0.447
Eswatini	2006/07	26152	0.225	0.417
Namibia	2013	21899	0.163	0.369
Mali	2015/16	69896	0.132	0.339
Zimbabwe	2015	53486	0.126	0.332
Malavi	2015/16	37434	0.110	0.313
Kenya	2008/09	17512	0.073	0.260
Gabon	2012	27619	0.049	0.216
Côte d'Ivoire	2011/12	24683	0.044	0.204
Gambia	2013	21585	0.020	0.141
Ghana	2014	21136	0.020	0.140
Liberia	2013	21602	0.019	0.137
Guinea	2018	23301	0.019	0.138
Sierra Leone	2019	33269	0.019	0.136
São Tomé and Príncipe	2008/09	12480	0.017	0.130
Ethiopia	2008	69369	0.015	0.120
Burkina Faso	2010	40805	0.013	0.113
Burundi	2016/17	46897	0.012	0.108
The Democratic Republic of the Congo	2013/14	50135	0.010	0.101
Senegal	2017	36652	0.008	0.092
Niger	2012	26611	0.005	0.071

Note: The table presents the size of the HIV-tested sample, the proportion of HIV-positive cases and standard deviations for the 21 countries from sub-Saharan Africa from recent DHSs.

5.2.2 Dependent Variable

Our dependent variable is a binary outcome and was recorded as a dummy variable indicating whether an individual was reported positive for HIV. According to the DHS program (see ICF Macro, 2010), HIV testing is structured as follows. The testing sample comprises voluntarily selected individuals aged 15-49. The collected blood samples are tested in the laboratory under the standard testing protocol. The laboratory method calls for an initial enzyme-linked immunosorbent assay (ELISA) test, followed by retesting all positive and 5–10% negative results with a second ELISA. If the two ELISA tests return discordant findings, a new ELISA test or a Western Blot test is conducted.

5.2.3 Independent Variables

Our main explanatory factor is knowledge about HIV/AIDS which includes eight binary variables (yes/no responses) that measure individual knowledge about HIV risk behaviours, prevalence and third-party transmission. Under knowledge about HIV risk behaviours, we consider whether respondents are knowledgeable about reducing the risk of contracting HIV based on (1) always using condoms during sex and (2) having one sex partner only who has no other partner. Knowledge about prevalence is tested from questions that ask (1) can mosquito bites spread HIV/AIDS, (2) can sharing food spread

HIV/AIDS and (3) is it possible for a healthy-looking person to have HIV/AIDS? Under knowledge about third-party transmission, we also consider three questions that examine whether respondents are knowledgeable about HIV transmission during pregnancy, delivery and breastfeeding. Finally, we construct an aggregate variable to measure knowledge about HIV/AIDS by adding all eight binary variables. The scale of our new variable then ranges from 0 (no knowledge) to 8 (highest knowledge).

Since we consider the knowledge of HIV to be an endogenous factor in the function of HIV prevalence (Eq [5.1]), we use maternal education to instrument individuals' knowledge of HIV (instrumental variable assumptions in the context of this instrument have been discussed under the empirical strategy section). Here, the maternal education variable is split into four ordinal categories: no education, primary, secondary and higher education. We use these categories as dummies to estimate the model (Eq [5.3]).

5.2.4 Other Control Variables

We control some socioeconomic, demographic and behavioural variables and some variables about media use in our empirical model because these capture some variation in our outcome variable. The socioeconomic conditions of the respondents are proxied by the family wealth index, occupation and some predetermined family backgrounds, such as whether the parents are alive and whether the respondents have a religion. The wealth index score is calculated using 33 standard questions suited to the context of Sub-Saharan Africa (Rutstein & Staveteig, 2014). Here we use the categorical wealth index variable with five ordinal categories (poorest, poor, middle, richer and richest) that refer to the wealth index score. Individuals' occupation includes seven ordinal categories. To capture the behavioural determinant of HIV prevalence, we include whether the respondents smoked, first had sex before 18 years old, and had sex with only a spouse/partner. As demographic variables, we consider age, gender and living area that may also capture the behavioural determinant of HIV prevalence (Djiadeu et al., 2020; Estébanez et al., 2001).

5.2.5 Summary Statistics

Table 5.2 shows that a higher proportion of people in our sample are knowledgeable about mitigating HIV risk and HIV transmission, ranging from 62% to 87% across our knowledge measures. Considering the mothers' education for respondents, we show that about 72% of mothers have only primary or less education; only 2.3% have higher educational qualifications. Our sample comprises 67% females, with most respondents

around 35 years old. Furthermore, approximately 43% are in the poor or less income bracket.

Table 5.2 Summary Statistics for Estimated Sample

Variable	Obs	Mean	Std. Dev.	Min	Max
HIV Positive	174,702	0.057	0.232	0	1
Reduce Risk: Use Condom	174,702	0.795	0.403	0	1
Reduce Risk: One Sex Partner	174,661	0.874	0.332	0	1
Can't Get HIV: Mosquito Bites	174,622	0.628	0.483	0	1
Can't Get HIV: Sharing Foods	174,567	0.791	0.407	0	1
Can Get HIV: Healthy Looking Person	174,441	0.802	0.399	0	1
HIV Transmitted During Pregnancy	174,694	0.734	0.442	0	1
HIV Transmitted During Delivery	174,689	0.788	0.409	0	1
HIV Transmitted During Breastfeeding	161,524	0.772	0.419	0	1
HIV Prevalence Knowledge Score	161,070	6.166	1.814	0	8
Female	174,702	0.671	0.470	0	1
Age	174,702	35.182	9.053	15	64
Urban	174,702	0.343	0.475	0	1
Has Religion	174,702	0.989	0.104	0	1
Family Wealth: Poorest	174,702	0.225	0.417	0	1
Poorer	174,702	0.208	0.406	0	1
Middle	174,702	0.200	0.400	0	1
Richer	174,702	0.193	0.394	0	1
Richest	174,702	0.175	0.380	0	1
Occupation: Non	174,702	0.234	0.423	0	1
Professional/Technical/Managerial	174,702	0.059	0.236	0	1
Clerical	174,702	0.010	0.097	0	1
Sales	174,702	0.147	0.354	0	1
Agricultural	174,702	0.342	0.475	0	1
Services/Household and Domestic	174,702	0.113	0.316	0	1
Manual - Skilled and Unskilled	174,702	0.095	0.293	0	1
Mother Education: Non	49,966	0.387	0.487	0	1
Primary	49,966	0.337	0.473	0	1
Secondary	49,966	0.254	0.435	0	1
Higher	49,966	0.023	0.148	0	1
Mother Alive	174,702	0.975	0.155	0	1
Father Alive	174,702	0.947	0.223	0	1
Smoke	174,702	0.076	0.265	0	1
First Sex before 18	174,702	0.477	0.499	0	1
Sex With Only Spouse/Partner	174,702	0.880	0.324	0	1
Literate	174,702	0.390	0.488	0	1
Television	174,702	0.305	0.460	0	1
Mobile Phone	174,702	0.697	0.459	0	1
Radio	174,702	0.549	0.498	0	1

Note: The second column presents the size of the estimated sample of Eq (1). The mean, standard deviation, minimum and maximum values of each variable are presented from the third and sixth columns, respectively. The statistics for mother education belong to the estimated sample of Eq (2) and (3). For binary variables, mean values refer to sample proportion with given characteristics.

5.3 Empirical Strategy

Knowledge of HIV transmission reduces its prevalence. Therefore, as a baseline model, we specify the following regression (Eq [5.1]) that links the risk of infecting HIV with knowledge about it. Here, we employ *k* number of regressions for each marker of HIV knowledge, including some demographic and socioeconomic characteristics as control variables. We estimate Eq (5.1) using ordinary least squares (OLS) and apply heteroskedasticity-robust standard errors throughout the models.

$$HIV_{cit} = \alpha_0 + \beta HK_{jcit} + \acute{X_{cit}}\delta + \emptyset_c + \gamma_t + \varepsilon_{cit} \qquad j = 1, \ldots\ldots, k \qquad (5.1)$$

In Eq 5.1, HIV_{cit} indicates whether individual *i* in country *c* in time *t* is positive for HIV. $HK_{jcit} \in \{1,2\ldots.8\}$ is knowledge indicator *j* of individual *i* in country *c* in time *t.* Vector $\acute{X_{cit}}$ represents our controls, $\emptyset_c$ and γ_t represent country and time effects, respectively, and ε_{cit} is the error term. We estimate Eq (5.1) using OLS. Therefore, the parameter β can be interpretable as a causal effect of HIV knowledge on the risk of HIV.

Nevertheless, we cannot verify whether people obtain HIV awareness or knowledge before being tested or contracted. On the one hand, some empirical evidence shows that the proportion of people unaware of their HIV-positive status is considerably high in Sub-Saharan Africa (de Walque et al., 2015; Kharsany & Karim, 2016). For that, higher socioeconomic inequality in HIV education in this region (see Chirwa, 2020) may be one of the main seasons. Further, Table 5.2 shows that about 13%–38% of people in our sample have less knowledge about HIV. These statistics indicate that a significant proportion of people may obtain HIV awareness or knowledge while they attend HIV testing or after contracting HIV (see Hutchinson et al., 2006). On the other hand, more educated individuals are more likely to know about HIV/AIDS before they attend HIV-test clinics. Therefore, we assume that there may be a two-way relationship (i.e., reverse causality) between HIV prevalence and HIV knowledge. However, we do not assume that our model estimates are biased due to omitted variables or measurement errors. Since individual SES can impact both HIV prevalence and HIV knowledge/awareness, we have included respondent SES such as education and occupation in the estimated model. Also, since DSH data is highly accurate and collected from a representative random sample, there is no high probability of having measurement errors. Consequently, we conclude that knowledge of HIV is an endogenous factor in Eq (5.1). Therefore, since

$Cov(HK_{jcit}\epsilon_{ict}) \neq 0$, we cannot define β as the causal effect of knowledge about the risk of HIV (or HIV prevalence).

To deal with the identification issue in Eq (5.1), we consider the existing literature on the causal effect of education on wages that uses parental education as an instrumental variable for respondents' education level. In the same way, we argue that maternal education is linked to the unobservable characteristics that impact individuals' HIV knowledge (i.e., our main RHS variable) and HIV infection (i.e., LHS variable) in Eq (5.1). Therefore, we specify our two-stage least squares regressions using maternal education as an instrumental variable for HIV knowledge:

$$HK_{jcit} = \theta + \pi ME_{cit} + \acute{X_{cit}}\lambda + \emptyset_c + \gamma_t + \nu_{cit} \quad j = 1, \ldots\ldots, k \qquad (5.2)$$

$$HIV_{cit} = \alpha_0 + \beta HK^*_{jcit} + \acute{X_{cit}}\delta + \emptyset_c + \gamma_t + \varepsilon^*_{cit} \qquad j = 1, \ldots\ldots, k \qquad (5.3)$$

$$HK^*_{jcit} = \theta + \pi ME_{cit} + \acute{X_{cit}}\lambda + \emptyset_c + \gamma_t \qquad j = 1, \ldots\ldots, k \qquad (5.4)$$

Here, ME_{cit} represents maternal education of individual *i* in country *c* in time *t*. In the first stage (Eq [5.2]), we estimate individual HIV knowledge on maternal education while controlling for other observable characteristics with country and time fixed effects (FE). We then use the estimated value of Eq (5.2) in the second stage (Eq [5.3]) instead of HK_{jcit} in Eq (5.1).

5.4 Result

The results in Table 5.3 show the estimates for Eq (5.1) and (5.3). Model 1 reports the OLS estimate of our baseline HIV prevalence function (Eq [5.1]), while Model 3 reports the IV regression estimate (EQ [5.3]) using maternal education as the instrument. We run separate regressions for each HIV knowledge variable. The estimated β values in the baseline regressions (Model 1) are positive and significant in all models, excluding the two models. We show similar estimates in the probit regressions (see Table A5.1 in the appendix). Since adequate health literacy about HIV decreases the likelihood of HIV/AIDS risk behaviours (Haile et al., 2007; Kickbusch, 2001; Swenson et al., 2010), the relationship between HIV literacy and prevalence should be negative. In this case, β values may not be positive. This result indicates that HIV knowledge correlates with the error term ($Cov(HK_{jcit}\epsilon_{ict}) \neq 0$) for our OLS estimates, supporting our argument on potential sources of bias in the baseline model.

In contrast to the baseline estimates, β values in the IV regressions (Model 3) are negative and highly significant in all models. This result shows that the causal effect of each HIV knowledge measure on HIV prevalence is negative. For example, if an individual knows that the risk of HIV can be reduced by using a condom while having sex and being satisfied with one sexual partner, the chance of getting HIV/AIDS is less (by 0.153 and 0.529, respectively) than his/her counterpart who is not thus aware. We also show that the magnitude effect of awareness of pregnancy-related HIV transmission is relatively high, whereas the estimates presented are fairly small for other knowledge measures (e.g., mosquito bites and sharing food cannot spread HIV/AIDS, and a healthy-looking person may have HIV/AIDS). Again, interestingly, these two-stage least square IV estimates results are in line with our probit IV regression estimates (see Table A5.1 in the appendix).

Considering the other control variables, we show some interesting results. For instance, as shown in previous literature on the African region (e.g., Sia et al., 2014), our regression estimates (both Models 1 and 3) show that women are more vulnerable to HIV/AIDS. In addition, living in an urban area and age have a positive impact on infection. In contrast, we show that religion may have a negative impact on the prevalence of this disease. Aside from the impact of demographic factors, considering the family's wealth index and individual's occupation, we show that rich people are safer for HIV/AIDS than poor people. This result confirms previous evidence (e.g., Parkhurst, 2010; Santelli et al., 2021) of the relationship between wealth and HIV risk. In addition, we show that smokers and individuals with risky sexual behaviours (begin sex before 18 years old, sex with many partners) are more vulnerable to the disease. Moreover, we show that access to the media (such as television and radio) may reduce the risk of HIV. Nonetheless, having a mobile phone seems to be a risk factor.

In the first stage estimates (Eq [5.2]) (see Table A5.2), we show R-squared ranges of between 5% and 22% across the indicators of HIV knowledge and high F-statistic in all models (ranges 64.399–329.212). Furthermore, we show that all HIV knowledge variables are positively and significantly associated with maternal education, suggesting that this can be a valid instrument for awareness of HIV transmission in the HIV prevalence function.

Table 5.3 Effect of HIV Knowledge on HIV Prevalence: OLS and 2SLS Regressions Results

	Model 1	Model 3	Model 1	Model 3	Model 1	Model 3	Model 1	Model 3	Model 1	Model 3
DV: HIV Positive										
Reduce Risk: Use Condom	0.011***	-0.153**								
Reduce Risk: One Sex Partner			0.003	-0.529***						
Can't Get HIV: Mosquito Bites					0.002	-0.185***				
Can't Get HIV: Sharing Foods							0.009***	-0.084		
Can Get HIV: Healthy Looking Person									0.005***	-0.188
Female	0.015***	0.002	0.014***	-0.009	0.014***	0.019***	0.014***	0.012***	0.014***	0.007*
Age	0.001***	0.001***	0.001***	0.001***	0.001***	0.001***	0.001***	0.001***	0.001***	0.001***
Urban	0.021***	0.021***	0.021***	0.016***	0.021***	0.027***	0.021***	0.021***	0.021***	0.023***
Has Religion	-0.053***	-0.050**	-0.053***	-0.034	-0.053***	-0.038*	-0.052***	-0.049**	-0.053***	-0.044**
Family Wealth: Ref. Poorest										
Poorer	0.001	0.006	0.001	0.017**	0.001	0.010**	0.001	0.004	0.001	0.005
Middle	0.006***	0.008**	0.006***	0.026***	0.006***	0.018***	0.005***	0.01	0.006***	0.014*
Richer	-0.003	0.007	-0.003	0.022**	-0.003	0.018***	-0.004*	0.008	-0.003	0.011
Richest	-0.007**	0.004	-0.006**	0.026**	-0.006**	0.023**	-0.007**	0.003	-0.007**	0.007
Occupation: Ref. Not in Work Force										
Professional/Technical/Managerial	-0.008***	0.004	-0.008**	0.007	-0.008**	0.005	-0.009***	-0.002	-0.008***	0.008
Clerical	-0.01	-0.005	-0.01	0.005	-0.01	0.004	-0.011	-0.005	-0.01	0.005
Sales	0.004**	0.008*	0.005**	0.011**	0.005**	-0.003	0.004**	0.004	0.004**	0.011*
Agricultural	-0.011***	-0.005	-0.010***	-0.003	-0.010***	-0.015***	-0.010***	-0.012***	-0.010***	-0.008*
Services/household & Domestic	0.012***	0.013***	0.012***	0.011**	0.012***	0.001	0.012***	0.010**	0.012***	0.017**
Manual - Skilled and Unskilled	0.014***	0.015***	0.014***	0.018***	0.014***	0.007	0.014***	0.011**	0.014***	0.017**
Mother Alive	-0.015***	-0.012	-0.015***	-0.006	-0.014***	-0.002	-0.015***	-0.005	-0.015***	-0.015
Father Alive	-0.062***	-0.064***	-0.062***	-0.067***	-0.062***	-0.058***	-0.062***	-0.064***	-0.062***	-0.067***
Illiterate	0	-0.012*	-0.001	-0.025**	-0.001	-0.023***	0	-0.007	-0.001	-0.016
Smoker	-0.003	0.003	-0.003	-0.003	-0.003	0.002	-0.003	0.001	-0.003	0.003
First Sex before 18	0.008***	0.009***	0.008***	0.009***	0.008***	0.004*	0.008***	0.007***	0.008***	0.010***
Sex with Only Spouse/Partner	-0.037***	-0.037***	-0.037***	-0.029***	-0.037***	-0.036***	-0.037***	-0.036***	-0.037***	-0.039***
Television	-0.011***	-0.005	-0.011***	-0.002	-0.011***	-0.007*	-0.011***	-0.005	-0.011***	-0.004
Mobile Phone	0.003**	0.008***	0.004**	0.014***	0.004**	0.007***	0.003**	0.007**	0.004**	0.012**
Radio	-0.003**	-0.003	-0.003**	-0.001	-0.003**	-0.004*	-0.003**	-0.004*	-0.003**	-0.003
Year FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Country FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Constant	0.361***	0.523***	0.369***	0.836***	0.370***	0.457***	0.366***	0.437***	0.367***	0.551***
R-squared	0.124	0.036	0.123	.	0.123	.	0.124	0.097	0.123	0.011
N	149390	49966	149476	50005	149459	49992	149395	49975	149267	49920

Note: * p<0.10, ** p<0.05, ***p<0.01. This table presents our baseline OLS estimates (Model 1) and 2SLS IV regression estimates (Model 3). We included year and country fixed effects (FE) and used heteroskedasticity-robust standard errors throughout. In Model 3, we used maternal education for instrumenting HIV knowledge. Maternal education has four categories; therefore, we put this variable into the model as four dummy variables. Since we run a separate regression for each knowledge measure, there are nine regressions for each equation (Eq [1] and [3]). In addition to the dummy control variables, we have two categorical measures for family wealth and individual occupation.

Table 5.3: (Continued)

	Model 1	Model 3	Model 1	Model 3	Model 1	Model 3	Model 1	Model 3
DV: HIV Positive								
HIV Transmitted During Pregnancy	0.004***	-0.656**						
HIV Transmitted During Delivery			0.004***	-0.247***				
HIV Transmitted During Breastfeeding					0.006***	-0.249**		
HIV Awareness Score							0.003***	-0.031***
Female	0.014***	0.039***	0.014***	0.032***	0.014***	0.037***	0.014***	0.016***
Age	0.001***	0.002***	0.001***	0.002***	0.001***	0.002***	0.001***	0.001***
Urban	0.021***	0.033***	0.021***	0.024***	0.021***	0.024***	0.021***	0.024***
Has Religion	-0.053***	-0.071***	-0.053***	-0.053**	-0.055***	-0.059***	-0.054***	-0.049**
Family Wealth: Ref. Poorest								
Poorer	0.001	0.005	0.001	0.002	0.001	0	0	0.006*
Middle	0.006***	0.020**	0.006***	0.010**	0.005***	0.008**	0.005**	0.015***
Richer	-0.003	0.009	-0.003	0.006	-0.005**	0.004	-0.006**	0.011*
Richest	-0.006**	-0.013	-0.006**	0.002	-0.009***	-0.004	-0.010***	0.007
Occupation: Ref. Not in Work Force								
Professional/Technical/Managerial	-0.008**	0.023	-0.008***	0.014	-0.009***	0.011	-0.011***	0.005
Clerical	-0.01	-0.007	-0.01	0.004	-0.004	0.009	-0.006	0.008
Sales	0.005**	0.014*	0.005**	0.004	0.004*	0.006	0.004	0.006
Agricultural	-0.010***	0.002	-0.010***	-0.010***	-0.011***	-0.012***	-0.012***	-0.011***
Services/household and Domestic	0.012***	0.016**	0.012***	0.007	0.014***	0.006	0.013***	0.010**
Manual - Skilled and Unskilled	0.014***	0.019**	0.014***	0.012**	0.015***	0.013**	0.015***	0.014***
Mother Alive	-0.015***	-0.038	-0.015***	-0.01	-0.016***	-0.01	-0.016***	-0.003
Father Alive	-0.062***	-0.032	-0.062***	-0.058***	-0.062***	-0.054***	-0.062***	-0.055***
Can Not Read	-0.001	-0.031**	-0.001	-0.016**	-0.002	-0.013**	0	-0.019**
Smoke	-0.003	0.01	-0.003	0.003	-0.002	0.007	-0.002	0.004
First Sex before 18	0.008***	0.011***	0.008***	0.009***	0.007***	0.010***	0.007***	0.008***
Sex With Only Spouse/Partner	-0.037***	-0.029***	-0.037***	-0.032***	-0.038***	-0.027***	-0.038***	-0.033***
Television	-0.011***	-0.010*	-0.011***	-0.007*	-0.010***	-0.008*	-0.010***	-0.004
Mobile Phone	0.004**	0.005	0.004**	0.012***	0.003**	0.013***	0.003*	0.011***
Radio	-0.003**	0.002	-0.003**	-0.002	-0.002	-0.002	-0.002	-0.003
Year FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Country FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Constant	0.368***	0.814***	0.368***	0.555***	0.373***	0.552***	0.361***	0.555***
R-squared	0.123	.	0.123	.	0.123	.	0.123	0.062
N	149529	50019	149519	50017	137624	46157	137067	45961

Note: * p<0.10, ** p<0.05, ***p<0.01

5.5 Discussion and Conclusion

There is a lack of evidence about the causal effect of HIV knowledge on the prevalence of HIV. To study this, we exploit the relatively large DHS data from 21 Sub-Saharan African counties and HIV test results of surveyed individuals who voluntarily attended testing clinics. For identification, we use maternal education as an instrument. Our sample consists of 174,702 observations for baseline models and 49,966 observations for IV models. Our findings are as follows.

Our OLS estimates are more positive and considerably smaller (i.e., positive estimates indicate that the risk of HIV infection is associated with higher knowledge of HIV) than the causal estimates. The positive sign of the beta coefficient of HIV knowledge variables in our baseline model indicates an identification issue because this is an unrealistic correlation; in fact, comprehensive awareness and knowledge prevent people from HIV risk behaviours (Swenson et al., 2010). We assume this unfair estimate occurs because HIV knowledge is an endogenous factor in this model because of reverse causality. The reverse causality between these two factors may occur due to considerable socioeconomic inequality in HIV education. In this case, we used an instrumental variable approach and exploited maternal education to instrument endogenous HIV knowledge. We identified that the mother's level of education is a valid instrument for modelling HIV prevalence on HIV education or awareness. Our causal estimates are negative and highly significant. Here, we have controlled for country and year fixed effects and for some individual characteristics. The results are consistent for all the markers of HIV knowledge. We identified that the mother's level of education is a valid instrument for modelling HIV prevalence on HIV education or awareness. Our causal estimates are negative and highly significant. Here, we have controlled for country and year fixed effects and for some individual characteristics. The results are consistent for all the markers of HIV knowledge. This result shows persuasive new evidence that peoples' knowledge about HIV has a causal impact on controlling the infection: HIV knowledge thus changes the risk of HIV transmission.

For this casual impact, higher socioeconomic inequality in the comprehensive knowledge of HIV transmission (Ainsworth & Semali, 1998; Chirwa, 2020) may play a significant role. These disparities in HIV knowledge can be driven by individuals' inherited circumstances (such as parental characteristics and gender) and their present socioeconomic backgrounds (such as education and income).

Since educational outcomes transmit across generations (Agüero & Ramachandran, 2018; Daouli et al., 2010), we suggest that maternal education can be transferred into a safeguard for preventing HIV/ AIDS. Individuals with educated mothers are more likely to have better education, which links with better employment, healthy lifestyle behaviours and better health throughout life than those with less-

educated mothers. Thus, for HIV prevention, maternal education may impact in several ways. Firstly, a higher level of offspring education correlates with higher HIV education. On the one hand, an increase in schooling can increase an individual's HIV knowledge. On the other hand, educated individuals can also be aware through better social interaction and duly accessing the media. Secondly, a family with an educated mother may have an environment for sharing knowledge, children's social experiences, and problems. This close family relationship may drive a better mother-child relationship, preventing children from HIV-risk behaviours (Cano et al., 2016; M. K. Hutchinson et al., 2003). For example, mothers can pass their knowledge to children and be aware of their children at home while creating a secure social background. Thirdly, the mother's comprehensive HIV awareness reduces the mother-to-child transmission of the disease.

Previous empirical evidence shows that the relationship between education and HIV knowledge is positive and significant. Higher educational disparities may thus correlate with higher inequalities in HIV knowledge (Chirwa, 2020). While well-educated individuals quickly and accurately understand HIV education, lower-educated individuals may take considerable time to understand the information provided and may sometimes misinterpret it (Kiviniemi et al., 2018). In addition, less educated young people have less confidence in discussing sexually transmitted diseases and accessing health services (such as awareness programs). Therefore, we suggest that increasing educational attainment (particularly for disadvantaged people) can protect individuals from HIV/AIDS because it helps to shift them into better behaviours through information and knowledge.

Moreover, comprehensive knowledge about HIV is higher among wealthy people (Chirwa, 2020; Faust et al., 2017). This wealth-related inequality in HIV knowledge persists because income disparity increases the disparity of accessing health information through the media (such as television, internet and radio) and schooling.

In Sub-Saharan African countries, there is a relatively high HIV/AIDS prevalence among females (Hargreaves et al., 2015; Sia et al., 2014); our regression results also show that the risk of the disease is higher among them. Compared with men, the HIV knowledge of these women is also relatively lower (Chirwa, 2020; Oljira et al., 2013). For this heterogeneity in HIV risk and knowledge, higher gender inequality in various socioeconomic factors (e.g., income, education, choices, social power) may matter. For example, women tend to face sexual violence inside the family and even from outside because of less economic power and lack of autonomy (Gage, 2005). There is also a tendency to engage in prostitution among uneducated and poor women in such contexts (Monroe, 2005; Ulin, 1992).

Despite the fight against the HIV pandemic conducted for more than four decades, poorer individuals are still more likely to catch HIV than those who are better off (Parkhurst, 2010; Sia et al., 2014). In addition, evidence reveals a considerable decline in HIV prevalence among advantaged people where HIV/AIDS education and awareness programs are progressing (Santelli et al., 2021). Consequently, the risk of the disease has shifted from higher social classes to lower ones (S. Gillespie et al., 2007). We contend that socioeconomic disparities in HIV education might be largely responsible for inequality in HIV prevalence. Therefore, it is evident that expanding the opportunities for disadvantaged people to access HIV education and health care relating to HIV/AIDS has clear implications for reducing the disease's prevalence.

Appendix

Table A 5.1 Effect of HIV Knowledge on HIV Prevalence: Probit and Probit IV Regressions Results

	Probit Model	IV Probit Model	Probit Model	IV Probit Model	Probit Model	IV Probit Model	Probit Model	IV Probit Model	Probit Model	IV Probit Model
DV: HIV Positive										
Reduce Risk: Use Condom	0.147***	-1.120*								
Reduce Risk: One Sex Partner			0.047**	-1.999**						
Can't Get HIV: Mosquito Bites					0.043***	-0.761*				
Can't Get HIV: Sharing Foods							0.112***	-1.117*		
Can Get HIV: Health Looking Person									0.073***	-1.532**
Female	0.186***	0.058	0.181***	0.045	0.179***	0.182***	0.177***	0.141***	0.182***	0.089
Age	0.014***	0.013***	0.014***	0.013***	0.014***	0.015***	0.014***	0.014***	0.014***	0.014***
Urban	0.251***	0.201***	0.251***	0.156**	0.249***	0.240***	0.250***	0.218***	0.250***	0.207***
Has Religion	-0.165***	-0.228**	-0.162***	-0.14	-0.164***	-0.195*	-0.163***	-0.215**	-0.163***	-0.16
Family Wealth: Ref. Poorest										
Poorer	-0.013	0.022	-0.012	0.047	-0.012	0.018	-0.016	0.03	-0.013	0.016
Middle	0.040**	0.059*	0.039**	0.100**	0.038**	0.082*	0.035*	0.111*	0.038**	0.105**
Richer	-0.049**	0.029	-0.049**	0.063	-0.052**	0.047	-0.058***	0.077	-0.050**	0.067
Richest	-0.042	0.022	-0.041	0.068	-0.046*	0.053	-0.050*	0.06	-0.044	0.056
Occupation: Ref. Not in Work Force										
Professional/Technical/Managerial	-0.051*	0.036	-0.047*	0.018	-0.050*	0.005	-0.057**	0.015	-0.050*	0.075
Clerical	-0.051	-0.034	-0.048	0.000	-0.051	-0.013	-0.054	-0.011	-0.05	0.056
Sales	0.070***	0.077**	0.073***	0.062**	0.074***	0.016	0.072***	0.057*	0.072***	0.099***
Agricultural	-0.145***	-0.117**	-0.140***	-0.113*	-0.139***	-0.190***	-0.140***	-0.177***	-0.139***	-0.120*
Services/Household and Domestic	0.092***	0.095***	0.094***	0.061*	0.095***	0.034	0.092***	0.077**	0.092***	0.119***
Manual - Skilled and Unskilled	0.100***	0.093**	0.104***	0.082**	0.106***	0.051	0.102***	0.081**	0.103***	0.114***
Mother Alive	-0.091***	-0.012	-0.090***	0.021	-0.089***	0.041	-0.089***	0.081	-0.090***	-0.019
Father Alive	-0.350***	-0.331***	-0.351***	-0.301***	-0.353***	-0.329***	-0.352***	-0.344***	-0.351***	-0.328***
Illiterate	0.004	-0.09	-0.003	-0.088*	0.000	-0.085	0.006	-0.114	0.000	-0.132*
Smoker	-0.027	0.041	-0.024	0.016	-0.023	0.041	-0.022	0.037	-0.023	0.044
First Sex before 18	0.092***	0.088***	0.092***	0.075***	0.093***	0.072***	0.095***	0.073**	0.092***	0.091***
Sex with Only Spouse/Partner	-0.252***	-0.238***	-0.255***	-0.171**	-0.254***	-0.241***	-0.251***	-0.255***	-0.255***	-0.235***
Television	-0.102***	-0.029	-0.098***	-0.021	-0.097***	-0.048	-0.096***	-0.012	-0.098***	-0.012
Mobile Phone	0.033**	0.091***	0.035**	0.091***	0.036**	0.081***	0.034**	0.097***	0.034**	0.120***
Radio	-0.029**	-0.02	-0.028**	-0.015	-0.028**	-0.033	-0.030**	-0.025	-0.028**	-0.018
Year FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Country FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Constant	-0.661***	0.631	-0.568***	1.342	-0.549***	-0.149	-0.606***	0.311	-0.594***	0.991
Wald-Chi2	18117.915	5274.03	18272.888	7479.042	18176.233	4402.71	18496.086	4966.53	17927.406	6696.581
Wald Test of Exogeneity (P-Value)		0.065		0.094		0.114		0.114		0.103
Sample	174702	49966	174788	50005	174761	49992	174706	49975	174568	49920

* p<0.10, ** p<0.05, ***p<0.01

Table A 5.1 (Continued)

	Probit Model	IV Probit Model	Probit Model	IV Probit Model	Probit Model	IV Probit Model	Probit Model	IV Probit Model
DV: HIV Positive								
HIV Transmitted During Pregnancy	0.053***	-1.518*						
HIV Transmitted During Delivery			0.051***	-1.339				
HIV Transmitted During Breastfeeding					0.070***	-1.241		
HIV Awareness Score							0.040***	-0.138
Female	0.176***	0.184***	0.175***	0.241***	0.172***	0.250***	0.166***	0.149***
Age	0.014***	0.014***	0.014***	0.016***	0.014***	0.014***	0.014***	0.015***
Urban	0.241***	0.186***	0.241***	0.198***	0.233***	0.193***	0.244***	0.226***
Has Religion	-0.162***	-0.251**	-0.166***	-0.238**	-0.170***	-0.266***	-0.169***	-0.245**
Family Wealth: Ref. Poorest								
Poorer	0.001	0.006	0.001	0.001	-0.001	-0.006	-0.019	0.008
Middle	0.038**	0.056*	0.038**	0.055	0.032*	0.042	0.028	0.076
Richer	-0.045**	0.004	-0.046**	0.015	-0.060***	0.000	-0.073***	0.029
Richest	-0.048*	-0.073	-0.049*	-0.018	-0.073***	-0.063	-0.077***	-0.014
Occupation: Ref. Not in Work Force								
Professional/Technical/Managerial	-0.057**	0.031	-0.060**	0.063	-0.066**	0.04	-0.071***	-0.001
Clerical	-0.090*	-0.049	-0.089*	0.006	-0.053	0.036	-0.022	0.026
Sales	0.072***	0.057**	0.073***	0.04	0.068***	0.052*	0.066***	0.056
Agricultural	-0.146***	-0.115	-0.146***	-0.156***	-0.147***	-0.167**	-0.144***	-0.185***
Services/Household and Domestic	0.096***	0.076**	0.097***	0.055	0.108***	0.062	0.101***	0.079**
Manual - Skilled and Unskilled	0.102***	0.069**	0.102***	0.064*	0.105***	0.066*	0.103***	0.075*
Mother Alive	-0.079***	-0.064	-0.079***	0.005	-0.086***	-0.008	-0.095***	0.042
Father Alive	-0.336***	-0.205	-0.336***	-0.275**	-0.332***	-0.263**	-0.349***	-0.315***
Illiterate	-0.005	-0.067	-0.004	-0.089	-0.013	-0.073	0.011	-0.083
Smoker	-0.04	0.025	-0.038	0.017	-0.033	0.04	-0.021	0.055
First Sex before 18	0.092***	0.076**	0.092***	0.084***	0.084***	0.083***	0.087***	0.083***
Sex with Only Spouse/Partner	-0.256***	-0.164*	-0.256***	-0.183**	-0.259***	-0.157*	-0.257***	-0.225***
Television	-0.086***	-0.033	-0.086***	-0.028	-0.072***	-0.029	-0.086***	-0.033
Mobile Phone	0.037**	0.056	0.036**	0.103***	0.032**	0.104***	0.025	0.101***
Radio	-0.027**	-0.016	-0.027**	-0.021	-0.018	-0.02	-0.02	-0.029
Year FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Country FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Constant	-0.596***	0.613	-0.598***	0.514	-0.579***	0.446	-0.735***	0.316
Wald-Chi2	15768.811	7807.964	.	6230.615	.	5520.806	17141.85	3796.898
Wald Test of Exogeneity (P-Value)		0.230		0.226		0.324		0.174
Sample	176892	50636	176887	50634	163639	46750	161070	45961

* p<0.10, ** p<0.05, ***p<0.01

Table A 5.2 Effect of Mother's Education on HIV Knowledge: First-Stage Estimates

	Model 2	Model 2	Model 2	Model 2	Model 2	Model 2	Model 2	Model 2	Model 2
	HK 1	HK 2	HK 3	HK4	HK5	HK6	HK7	HK8	HK9
Mother's Education	0.018***	0.009***	0.032***	0.020***	0.014***	0.008***	0.018***	0.017***	0.136***
Female	-0.071***	-0.041***	0.033***	-0.003	-0.028***	0.043***	0.079***	0.104***	0.165***
Age	-0.000**	0.000*	0	0	0.001**	0.001***	0.002***	0.001***	0.003***
Urban	0.002	-0.010**	0.041***	0.018***	0.011**	0.016***	0.013**	0.012**	0.102***
Has Religion	-0.002	0.026	0.070**	0.015	0.040*	-0.038	-0.011	-0.036	0.018
Family Wealth: Ref. Poorest									
Poorer	0.035***	0.032***	0.047***	0.046***	0.021***	0.006	0.002	-0.001	0.200***
Middle	0.032***	0.041***	0.070***	0.082***	0.059***	0.018***	0.018***	0.012*	0.336***
Richer	0.050***	0.046***	0.094***	0.095***	0.062***	0.009	0.020***	0.015**	0.396***
Richest	0.067***	0.061***	0.146***	0.105***	0.076***	-0.011	0.028***	0.017*	0.495***
Occupation: Ref. Not in Work Force									
Professional/Technical/Managerial	0.045***	0.018***	0.051***	0.046***	0.062***	0.045***	0.071***	0.067***	0.405***
Clerical	0.014	0.020*	0.061***	0.041***	0.059***	-0.002	0.041***	0.032*	0.269***
Sales	0.031***	0.015***	-0.025***	0.019***	0.042***	0.015**	0.003	0.011*	0.104***
Agricultural	0.039***	0.015***	-0.016**	-0.006	0.021***	0.022***	0.005	0.008	0.093***
Services/Household and Domestic	0.027***	0.004	-0.038***	0.018***	0.042***	0.009	-0.012*	-0.015**	0.039
Manual - Skilled and Unskilled	0.029***	0.016***	-0.017**	0.019***	0.042***	0.012*	0.006	0.009	0.126***
Mother Alive	-0.032	-0.007	-0.015	0.053	-0.042	-0.039	0.002	-0.016	-0.105
Father Alive	-0.005	-0.01	0.025	-0.007	-0.022*	0.041***	0.02	0.01	0.053
Illiterate	-0.083***	-0.044***	-0.117***	-0.109***	-0.088***	-0.045***	-0.062***	-0.050***	-0.597***
Smoker	0.008	-0.005	0	-0.001	0.005	0.012	0.009	0.015*	0.036
First Sex before 18	0.008**	0.003	-0.009**	-0.006	0.013***	0.006	0.011***	0.015***	0.042**
Sex with Only Spouse/Partner	-0.021***	0.006	-0.007	-0.030***	-0.024***	0.005	0.005	0.016**	-0.054**
Television	0.020***	0.012***	-0.004	0.027***	0.024***	-0.006	0	-0.006	0.075***
Mobile Phone	0.020***	0.015***	0.012**	0.020***	0.035***	0.002	0.030***	0.028***	0.164***
Radio	0.004	0.005	0	0.005	0.006	0.010**	0.008**	0.012***	0.048***
Year FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Country FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Constant	0.941***	0.870***	0.441***	0.658***	0.907***	0.655***	0.690***	0.710***	5.896***
R-squared	0.104	0.069	0.126	0.105	0.109	0.054	0.073	0.079	0.216
F-Statistics	123.411	64.399	204.844	142.509	160.489	74.827	116.465	115.4963	329.212
N	54204	54250	54230	54219	54160	54264	54262	50045	49829

Table A 5.3 Effect of HIV Knowledge on HIV Prevalence: OLS and 2SLS Regressions Results (Sample restricted by the observations for mother education)

	Model 1	Model 3	Model 1	Model 3	Model 1	Model 3	Model 1	Model 3	Model 1	Model 3
DV: HIV Positive										
Reduce Risk: Use Condom	0.013***	-0.153**								
Reduce Risk: One Sex Partner			0.001	-0.529***						
Can't Get HIV: Mosquito Bites					0.001	-0.185***				
Can't Get HIV: Sharing Foods							0.004**	-0.084		
Can Get HIV: Healthy Looking Person									0.002	-0.188
Female	0.013***	0.002	0.012***	-0.009	0.012***	0.019***	0.012***	0.012***	0.012***	0.007*
Age	0.001***	0.001***	0.001***	0.001***	0.001***	0.001***	0.001***	0.001***	0.001***	0.001***
Urban	0.019***	0.021***	0.020***	0.016***	0.020***	0.027***	0.019***	0.021***	0.019***	0.023***
Has Religion	-0.051**	-0.050**	-0.051**	-0.034	-0.051**	-0.038*	-0.051**	-0.049**	-0.051**	-0.044**
Family Wealth: Ref. Poorest										
Poorer	0	0.006	0	0.017**	0	0.010**	0	0.004	0	0.005
Middle	0.003	0.008**	0.004	0.026***	0.004	0.018***	0.003	0.01	0.004	0.014*
Richer	0	0.007	0	0.022**	0	0.018***	0	0.008	0	0.011
Richest	-0.007	0.004	-0.006	0.026**	-0.006	0.023**	-0.007	0.003	-0.006	0.007
Occupation: Ref. Not in Work Force										
Professional/Technical/Managerial	-0.006	0.004	-0.005	0.007	-0.005	0.005	-0.007	-0.002	-0.006	0.008
Clerical	-0.01	-0.005	-0.009	0.005	-0.009	0.004	-0.01	-0.005	-0.009	0.005
Sales	0.003	0.008*	0.003	0.011**	0.003	-0.003	0.003	0.004	0.003	0.011*
Agricultural	-0.012***	-0.005	-0.011***	-0.003	-0.011***	-0.015***	-0.012***	-0.012***	-0.012***	-0.008*
Services/household & Domestic	0.009**	0.013***	0.009**	0.011**	0.009**	0.001	0.009**	0.010**	0.009**	0.017**
Manual - Skilled and Unskilled	0.009**	0.015***	0.010**	0.018***	0.010**	0.007	0.009**	0.011**	0.010**	0.017**
Mother Alive	-0.01	-0.012	-0.011	-0.006	-0.011	-0.002	-0.011	-0.005	-0.012	-0.015
Father Alive	-0.063***	-0.064***	-0.063***	-0.067***	-0.063***	-0.058***	-0.063***	-0.064***	-0.063***	-0.067***
Illiterate	0.003	-0.012*	0.002	-0.025**	0.002	-0.023***	0.003	-0.007	0.002	-0.016
Smoker	0.001	0.003	0.002	-0.003	0.002	0.002	0.002	0.001	0.002	0.003
First Sex before 18	0.007***	0.009***	0.007***	0.009***	0.007***	0.004*	0.008***	0.007***	0.007***	0.010***
Sex with Only Spouse/Partner	-0.034***	-0.037***	-0.034***	-0.029***	-0.034***	-0.036***	-0.034***	-0.036***	-0.034***	-0.039***
Television	-0.008**	-0.005	-0.008**	-0.002	-0.008**	-0.007*	-0.007**	-0.005	-0.008**	-0.004
Mobile Phone	0.005**	0.008***	0.005**	0.014***	0.005**	0.007***	0.005**	0.007**	0.005**	0.012**
Radio	-0.005**	-0.003	-0.004**	-0.001	-0.004**	-0.004*	-0.005**	-0.004*	-0.005**	-0.003
Year FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Country FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Constant	0.367***	0.523***	0.378***	0.836***	0.379***	0.457***	0.377***	0.437***	0.379***	0.551***
R-squared	0.121	0.036	0.12	.	0.12	.	0.12	0.097	0.12	0.011
N	49966	49966	50005	50005	49992	49992	49975	49975	49920	49920

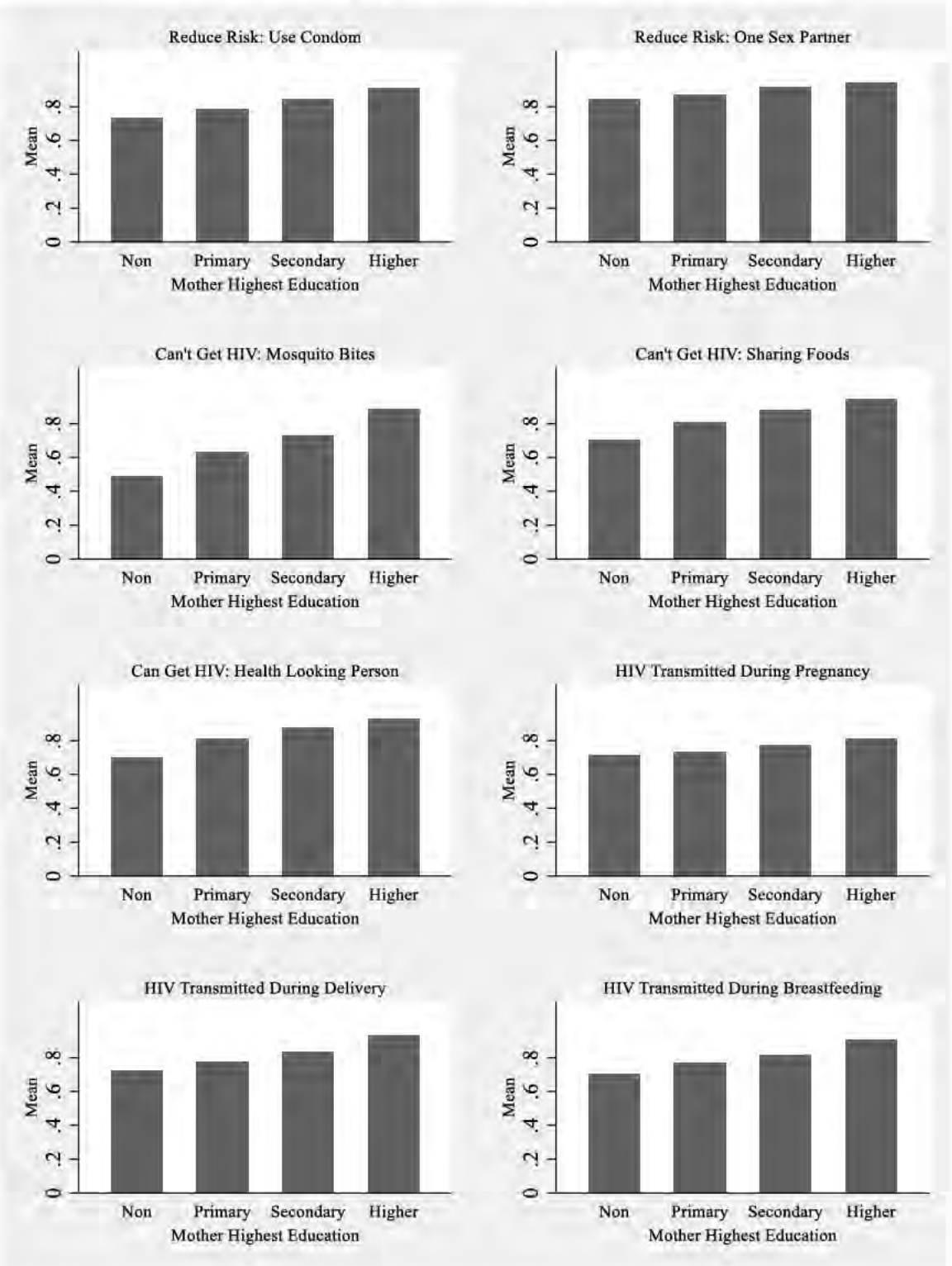
Note: * p<0.10, ** p<0.05, ***p<0.01. Table A5.3 presents our baseline OLS estimates (Model 1) and 2SLS IV regression estimates (Model 3). We included year and country fixed effects (FE) and used heteroskedasticity-robust standard errors throughout. In Model 3, we used maternal education for instrumenting HIV knowledge. Maternal education has four categories; therefore, we put this variable into the model as four dummy variables. Since we run a separate regression for each knowledge measure, there are nine regressions for each equation (Eq [1] and [3]). In addition to the dummy control variables, we have two categorical measures for family wealth and individual occupation.

Table A5.3: (Continued)

	Model 1	Model 3	Model 1	Model 3	Model 1	Model 3	Model 1	Model 3
DV: HIV Positive								
HIV Transmitted During Pregnancy	0.003	-0.656**						
HIV Transmitted During Delivery			0.005**	-0.247***				
HIV Transmitted During Breastfeeding					0.005**	-0.249**		
HIV Awareness Score							0.002***	-0.031***
Female	0.012***	0.039***	0.012***	0.032***	0.011***	0.037***	0.010***	0.016***
Age	0.001***	0.002***	0.001***	0.002***	0.001***	0.002***	0.001***	0.001***
Urban	0.019***	0.033***	0.019***	0.024***	0.019***	0.024***	0.019***	0.024***
Has Religion	-0.051**	-0.071***	-0.051**	-0.053**	-0.051**	-0.059***	-0.051**	-0.049**
Family Wealth: Ref. Poorest								
Poorer	0.000	0.005	0.000	0.002	0.000	0.000	0.000	0.006*
Middle	0.004	0.020**	0.004	0.010**	0.004	0.008**	0.003	0.015***
Richer	0.000	0.009	0.000	0.006	-0.001	0.004	-0.002	0.011*
Richest	-0.006	-0.013	-0.006	0.002	-0.009*	-0.004	-0.010**	0.007
Occupation: Ref. Not in Work Force								
Professional/Technical/Managerial	-0.005	0.023	-0.006	0.014	-0.008	0.011	-0.010*	0.005
Clerical	-0.009	-0.007	-0.009	0.004	-0.003	0.009	-0.004	0.008
Sales	0.003	0.014*	0.003	0.004	0.003	0.006	0.003	0.006
Agricultural	-0.012***	0.002	-0.011***	-0.010***	-0.013***	-0.012***	-0.014***	-0.011***
Services/household and Domestic	0.009**	0.016**	0.009**	0.007	0.009**	0.006	0.009**	0.010**
Manual - Skilled and Unskilled	0.010**	0.019**	0.010**	0.012**	0.010**	0.013**	0.009*	0.014***
Mother Alive	-0.011	-0.038	-0.011	-0.01	-0.002	-0.01	-0.004	-0.003
Father Alive	-0.063***	-0.032	-0.063***	-0.058***	-0.057***	-0.054***	-0.057***	-0.055***
Can Not Read	0.002	-0.031**	0.002	-0.016**	0.002	-0.013**	0.003	-0.019**
Smoke	0.002	0.01	0.002	0.003	0.003	0.007	0.002	0.004
First Sex before 18	0.007***	0.011***	0.007***	0.009***	0.007***	0.010***	0.007***	0.008***
Sex With Only Spouse/Partner	-0.034***	-0.029***	-0.034***	-0.032***	-0.032***	-0.027***	-0.031***	-0.033***
Television	-0.008**	-0.010*	-0.008**	-0.007*	-0.007**	-0.008*	-0.007**	-0.004
Mobile Phone	0.005**	0.005	0.005**	0.012***	0.005**	0.013***	0.005**	0.011***
Radio	-0.005**	0.002	-0.005**	-0.002	-0.005**	-0.002	-0.005**	-0.003
Year FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Country FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Constant	0.377***	0.814***	0.376***	0.555***	0.364***	0.552***	0.358***	0.555***
R-squared	0.12	.	0.12	.	0.119	.	0.119	0.062
N	50019	50019	50017	50017	46157	46157	45961	45961

Note: * p<0.10, ** p<0.05, ***p<0.01

Figure A 5.1 Association between HIV Knowledge/Awareness and Maternal Education



Chapter 6

Air Pollution and Health Outcomes: Evidence from the Black Saturday Bushfires in Australia

6.1 Introduction

Ambient air pollution is a persistent feature of developing and industrialising economies (Mage et al., 1996; Mannucci and Franchini, 2017; Mayer, 1999). While this development is fundamental in reducing global poverty and inequality, it also has the potential to adversely affect public health. For example, the concentrations of toxic substances such as fine particulate matter (PM2.5) and greenhouse gases have been increasing in the global atmosphere for decades since industrialisation (Butt et al., 2017; Smith et al., 2011) and are now at unprecedented levels (Shaddick et al., 2020). This industrial pollution is thought to contribute to several health-related problems, including mortality (Fan et al., 2020; Romero-Lankao et al., 2013), psychological stress (Tao et al., 2021), respiratory-related health problems (Pénard-Morand et al., 2005), cancers (Turner et al., 2020), and even cognitive functions (Ailshire et al., 2017).

Our study adds to the literature that examines the health effects of air pollution. Existing research has examined the effect of smoke on respiratory health, cardiovascular disease, chronic obstructive pulmonary disease, asthma and lungs cancer (Dennekamp et al., 2015; Franzi et al., 2011; Hamon et al., 2018; Kim et al., 2018; Navarro et al., 2019; Pavagadhi et al., 2013), and the effect of psychological stress on birth outcomes (Holstius et al., 2012). Considering the Black Saturday Bushfire incidents (BSB; occurring in Victoria, Australia, in 2009) using longitudinal data before and after the wildfires, Bryant et al. (2020) examined the long-term effects on mental health outcomes and using a cross-sectional survey. And Gallagher et al. (2016) found mental health impacts due to separation from family during the bushfires, while Johnston et al. (2021) identified a reduction in life satisfaction for people living in bushfire-affected areas.

In this chapter, we look to add to the empirical evidence on the health implications of carbon pollution using a relatively unstudied Australian panel data set. We employ a natural experimental approach by taking data from a high-quality survey and exploiting the BSB to obtain identifying variation. The fact that these fires present an exogenous shock to air particle matter allows us to obtain relatively clean estimates of causal effects, which circumvent many of the correlational/causal issues frequently encountered in micro econometric analyses of health (Angrist and Pischke, 2009). We show that individual

health status metrics are significantly lower in the pollution-affected groups, a result that holds across a range of health outcomes, including physical health, general health, and self-assisted health (SAH).

Moreover, extending our analysis using triple difference models (DDD), we show that individuals in major cities are more vulnerable to bushfire air pollution, alongside younger persons and those with lower educational attainments. Accordingly, less educated individuals, smokers, and young and early middle-aged individuals are in higher-risk categories. We attribute these results in part to heterogeneities in exposure to ambient air pollution between urban and regional areas. Due to the higher population density and economic activities (such as industrial works and road transport), which produce higher emission concentrations, air pollution is higher in the city than in the countryside (Strosnider et al., 2017; Yang, 2020). This can be partially attributed to transportation. In megacities, road traffic is a key source of air pollution (Churchill et al., 2021; Mage et al., 1996), producing a significant proportion of total emissions. In seven major world cities, Su et al. (2015) show that most urban populations are exposed to transport-related air pollution. Since cities already have relatively high levels of carbon pollution, additional shocks from bushfires may create a compounding effect.

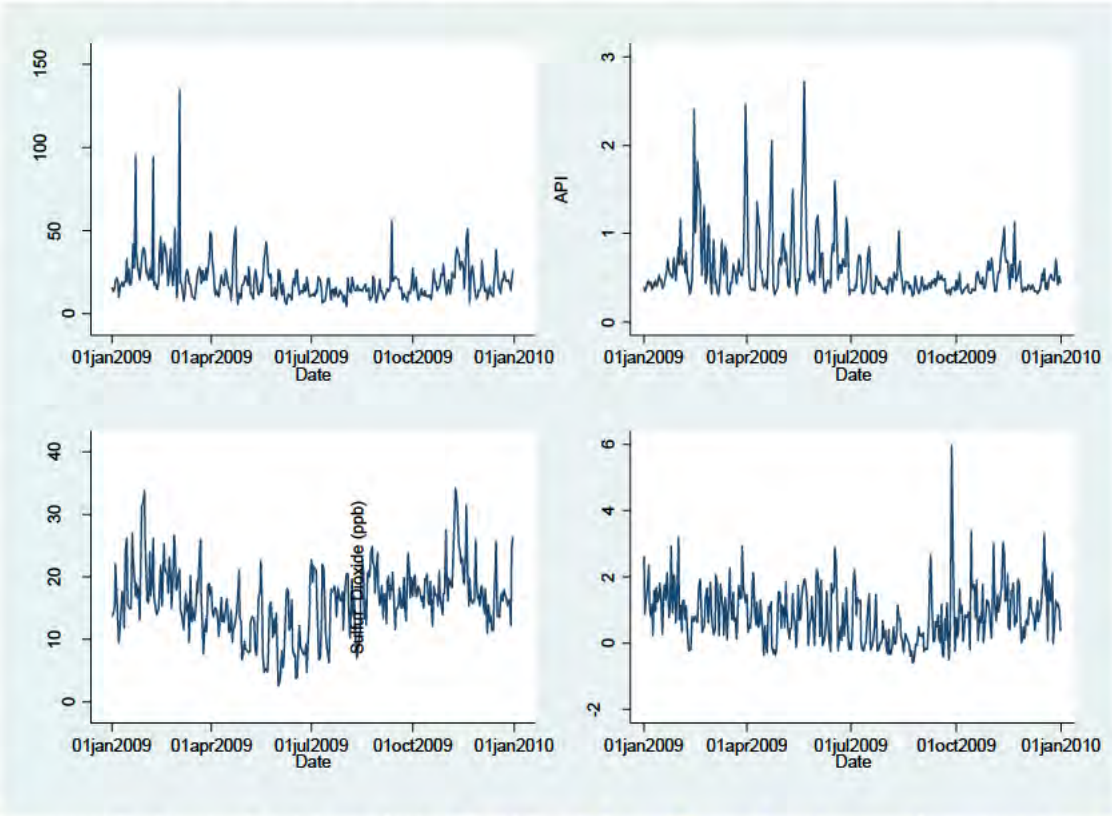
The chapter is structured as follows. The following section outlines the background of the 2009 BSB incident and the levels of air pollution during bushfires. Section 6.3 explains data and methods. Section 6.4 then presents descriptive statistics and regression results. Section 6.5 describes a robustness check procedure and results. The final section discusses the findings and concludes.

6.2 Background to the Black Saturday Bushfires

Bushfires have been one of the most severe natural hazards globally, damaging properties and causing premature death and injuries (Abram et al., 2021; Cameron et al., 2009; Doerr and Santín, 2016; Randerson et al., 2012). The BSB event of 2009 was one of Australia's most severe natural disasters. From 7th February to 14th March, dozens of bushfires burned through regional Victoria in the country's south, killing 173 people and scorching approximately 450,000 hectares of land. Moreover, the estimated direct cost of BSB is AUD 2939 million, which includes the health cost of AUD 1058 million (Stephenson et al., 2012). Using a "life satisfaction approach", Ambrey et al. (2017) estimated a Willingness to Pay of AUD 2991(with regards to household income) for reducing self-reported levels of life satisfaction. There were also subtler, long-term consequences as a

result of air pollutants. For example, Figure 6.1 shows that the concentration of PM_{10} and ozone emission rate during the BSB increased to the highest level of the year, and in particular, the level of PM_{10} increased to more than ten times higher than the WHO recommended concentration level of 20 µg/m3. Furthermore, emissions estimates (Paton-Walsh et al., 2012) show that the fires were dumping a significant amount of carbon dioxide (CO_2) and carbon monoxide (CO) into the atmosphere during the five-week period.

Figure 6.1 Daily Air Pollution Rates in Greater Melbourne and Geelong, Victoria, in 2009

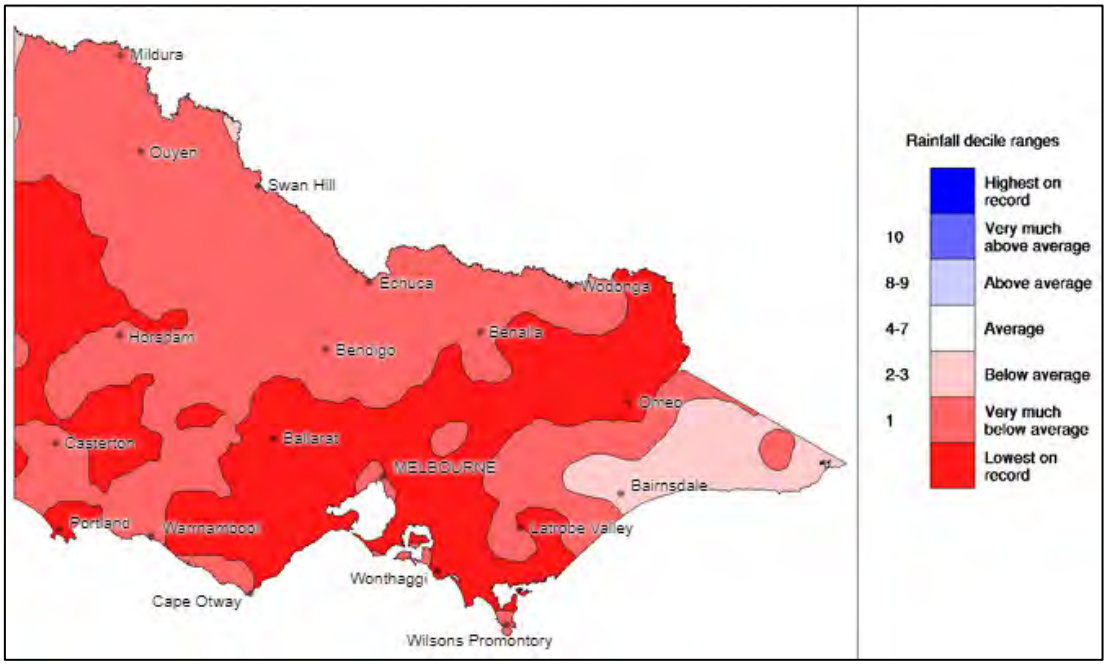


Data source: Environment Protection Authority (EPA) Victoria (https://discover.data.vic.gov.au/dataset/epa-air-watch-all-sites-air-quality-hourly-averages yearly/historical). Note: Figure 6.1 illustrates the four parameters of air pollution during 2009 in Greater Melbourne and Geelong in Victoria. Data were collected from 13 reading points hourly. These time-series graphs are based on daily average values for each location. Air pollution parameters are PM_{10} (particulate matter of diameter ≤ 10), API (airborne particle index), ozone and sulphur dioxide. The standard unit of measures is ppb (parts per billion) for ozone and sulphur dioxide and ug/m3 (micrograms per cubic meter) for PM10.

Australia's vulnerability to bushfires appears to be driven by increasing heat and dry weather conditions (Di Virgilio et al., 2019; Engel et al., 2013; Harris and Lucas, 2019; Nolan et al., 2020; Vardoulakis et al., 2020). Climate statistics show that Southeast Australia had experienced more than ten years of drought at the time of BSB. As Figure 6.2 shows, preceding the fires, the lowest rainfall in many years was recorded in many

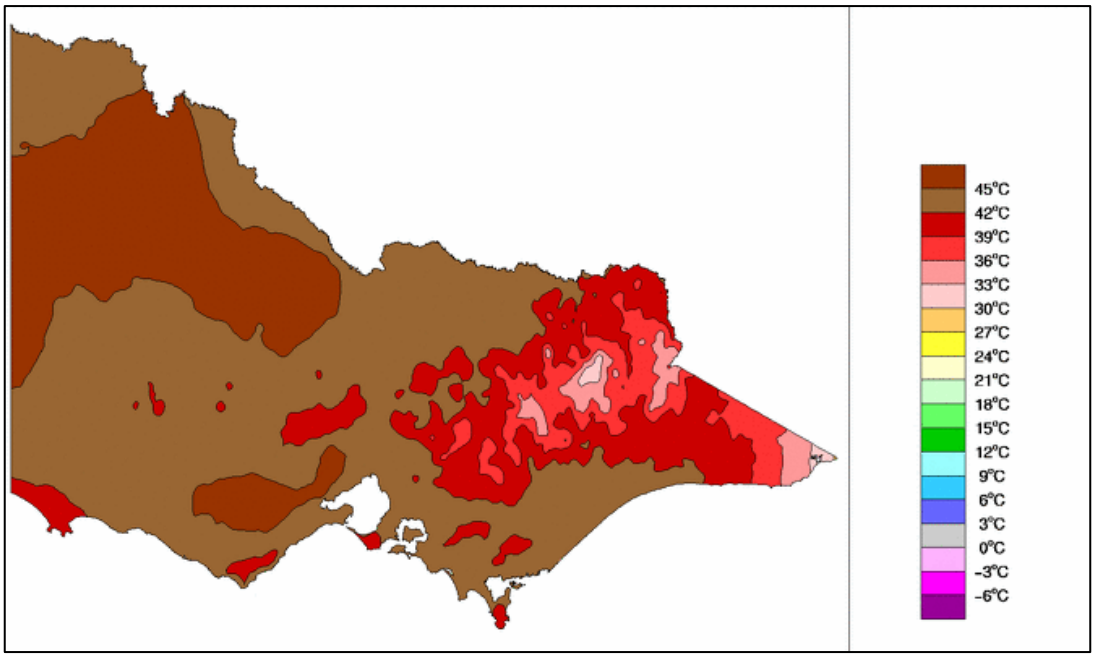
parts of Victoria. During the week before the BSB started, daily temperatures reached 45^0 C in Victoria in an exceptional heatwave (see Figure 6.3). On the date that the BSB began, the temperature in Victoria reached its highest level (46.4^0C), and more than four hundred fires started (Teague et al., 2009).

Figure 6.2 Victoria Rainfall Deciles, March 2005 to February 2009



Source: Commonwealth of Australia, Bureau of Meteorology (2021).

Figure 6.3 Temperature (°C)- First Week of February 2009 in Victoria, Australia



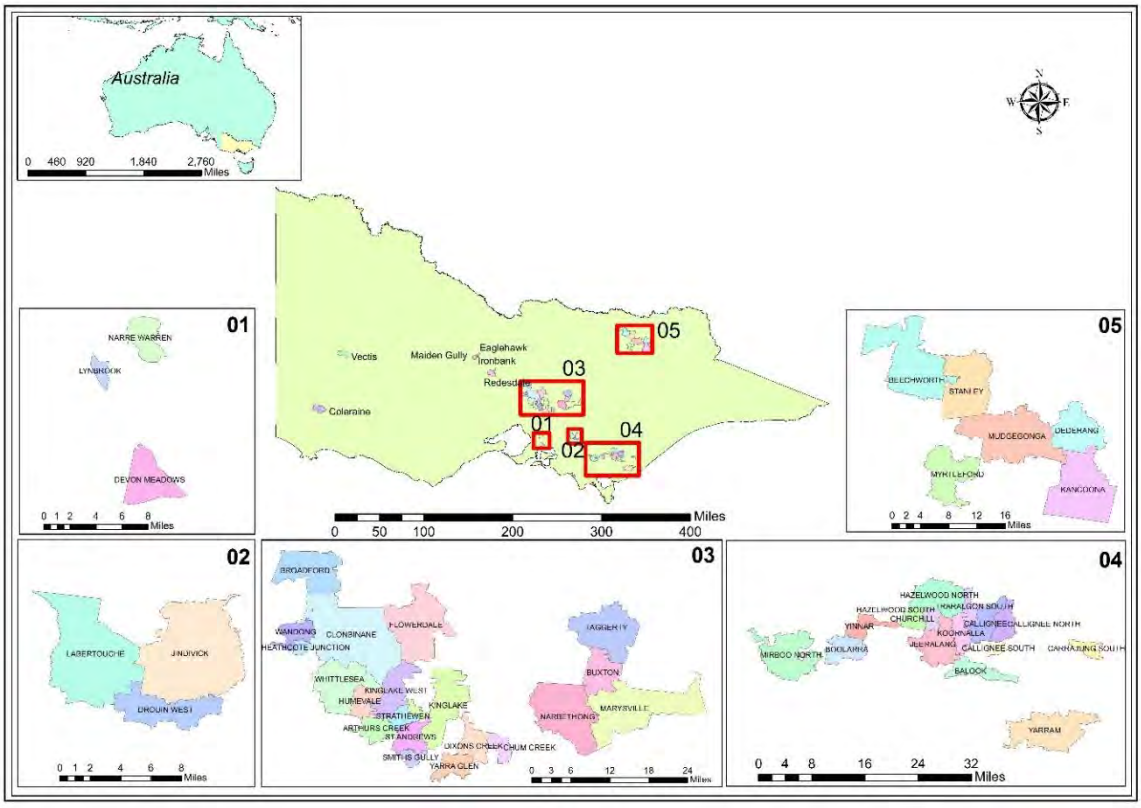
Source: Commonwealth of Australia, Bureau of Meteorology (2021).

6.3 Methodology

6.3.1 Data and Sample

We draw data from nine waves of the HILDA longitudinal survey from 2004 to 2012. We limit our data to Victoria to minimise cross-sectional heterogeneity and take data before and after the 2009 event. HILDA collects a range of information about Australians' socioeconomic, demographics, health and well-being, labour market and family life using a self-completion questionnaire from 2001. The data set follows more than 17000 Australians annually from a nationally representative sample drawn through a stratified three-stage clustered sampling design. Stratification was based on the states and the metropolitan and nonmetropolitan areas of the five most populated states (e.g., New South Wales, Victoria, Queensland, South Australia, and Western Australia).

Figure 6.4 The Map of Affected Areas by 2009 Black Saturday Bushfires



Note: The author developed this map using the geodata of BSB-affected areas. The areas are identified from the reports of the 2009 Victorian Bushfires Royal Commission (2010). The selection is based on the respondent's residential postal code. The shapefile for the suburbs of Victoria is taken from the Department of Industry Science Energy and Resources (2020). The locations where bushfire incidents were found are Boolarra, Churchill, Mirboo North, Yinnar, Arthurs Creek, Broadford, Chum Creek, Clonbinane, Dixons Creek, Flowerdale, Hazeldene, Heathcote Junction, Humevale, Kinglake, Kinglake West, Smiths Gully, St. Andrews, Strathewen, Wandong, Whittlesea ,Yarra Glen, Buxton, Marysville, Narbethong, Taggerty, Balook, Callignee North, Callignee South, Callignee, Carrajung South, Devon, Hazelwood North, Hazelwood South, Jeeralang North, Koornalla, Traralgon South, Yarram, Eaglehawk, Ironbark, Long Gully, Maiden Gully and West Bendigo.

6.3.2 Health Measures

We use five different markers of health as outcome variables (see Table 6.1 for details). Three are indicators for overall individual health: physical health conditions, general health conditions, and self-assessed health (SAH). Physical and general health markers are measured using SF-36, a widely employed self-completion questionnaire and validated using HILDA survey data (Butterworth and Crosier, 2004). The physical and general health measures scale ranges from '0' (lowest health) to '100' (highest health). SAH is split into five ordinal categories referring to the self-assessment of respondent health that ranges from '1' (excellent health) to '5' (poor health). Although SAH has been identified as a measurement with individual reporting heterogeneity (Bago D'Uva et al., 2008), it is widely employed because it has been recognised as a better indicator of disease burden and an even better predictor of mortality (Lorem et al., 2020; Thong et al., 2008). We employ another health measure as a proxy for respiratory health, a binary outcome indicating whether a respondent has shortness of breath or difficulty breathing. Lastly, we use an ordered variable that indicates how much respondent agrees with the statement "gets sick a little easier than other people". The responses are split into five categories that range from '1' (definitely true) to '5' (definitely false).

6.3.3 Observable Characteristics

The triple difference (DDD) method searches for variations in effect sizes across population subgroups. We focus our analysis on socioeconomic, behavioural and demographic characteristics that may drive unequal exposure to air pollution. We differentiate individuals according to region, age, education, income, and their status as a smoker (smokers, in particular, may have differentiated effects from the population as a whole). These are classified using dummies denoting living in a major city, individual over 45 (denoting later middle age and late adulthood – see Medley (1980)), belonging to the first income quantile, having an education above year 12, and being a smoker.

6.3.4 Control Variables

We control individual demographic characteristics and socioeconomic variables that may influence their health status. For example, age and gender may capture the biological and behavioural determinants of health (Bird and Rieker, 2008; Liang et al., 1999). Respondents' age is measured as a scale variable, and we have a dummy for women. Also, to capture the effect of individual socioeconomic status on their health, we have controlled educational attainment and log income. We control for other background

characteristics variables using dummies: whether the origin is Indigenous, Australia is the country of birth, English is the first language learned, and immigration and refugee status.

Table 6.1 Summary of the HILDA Data Extract

Variable	Observation	Mean	Min	Max
General Health	109434	68.55	0	100
Physical Health	109715	83.37	0	100
Breath Problem	33956	0.095	0	1
SAH	109709		1	5
Excellent [1]		0.114		
Very Good [2]		0.358		
Good [3]		0.359		
Fair [4]		0.138		
Poor [5]		0.031		
Get Sick Easier	109455		1	5
Definitely True [1]		0.022		
Mostly True [2]		0.069		
Do not Know [3]		0.122		
Mostly False [4]		0.299		
Definitely False [5]		0.487		
Education Level	125145		1	4
Bachelor and Above [1]		0.214		
Year 12 and Adv Dip [2]		0.439		
Year 11 and Below [3]		0.347		
Undetermined [4]		0.001		
Indigenous	168118	0.010	0	1
Australia	136619	0.798	0	1
English	136640	0.902	0	1
Income (Family)	133442	53381	36	1266283
Smokes	168118	0.134	0	1

Note: Table 1 shows summary statistics for our sample, which includes the waves from 2004 to 2012. For the ordinal and binary variables, the mean value represents the sample proportion of each category. For binary variables, '1' indicates the outcome and otherwise '0'.

6.3.5 Difference-in-Difference (DiD) Procedure

We use a quasi-experimental approach (e.g., (Daley et al., 2021)) to analyse the causal effect of bushfire pollution on health outcomes. We defined the period after 2009 as the "post-bushfire period", while the period before 2009 is called the "pre-bushfire period." We define a dummy variable (*PB*) in which the observations in the post-bushfire period are coded '1' and '0' otherwise. The treatment group (*TG=1*) in the affected suburbs

during both periods, while the observations in the control group *(TG=0)* belong to individuals who lived in unaffected suburbs throughout the period considered. Since, in general, individuals suffer from health issues either pre or post bushfire period, the coefficient of the interaction term *(PB×TG)* indicates the *DiD* estimator. Thus, we first estimate the following standard regression model using ordinary least squares (OLS).

$$H_{it} = \alpha_0 + \alpha_1 PB._{it} + \alpha_2 TG._{it} + \gamma_1 (PB \times TG)_{it} + X'_{it}\delta + \varepsilon_{it} \qquad (6.1)$$

Here, H_{it} denotes health outcomes, X'_{it} represents the set of control variables and ε_{it} is the error term. *i* indexes for the individual while *t* indexes for time. Parameter γ_1 is *DiD* parameter to be estimated (the parameter of interest). α_1, α_2 and δ are the parameters to be estimated for the post-bushfire period, treatment group and control variables, respectively whereas α_0 represents the intercept of the model to be estimated.

6.3.6 Triple Differences Procedure (DDD)

We extend the *DiD* model to a *DDD* model to analyse whether individuals in major cities differ in response to less health status after bushfires. Air pollution in major cities is relatively higher than in the regional area due to higher industrial and urban pollution. Since the cumulative air pollution rate may be higher in the city area during bushfires (see Figure 6.1), we assume that any health effect varies between regional and major city areas. In addition, we examine whether other socioeconomic inequalities modify our parameter estimates.

$$H_{it} = \beta_0 + \beta_1 PB._{it} + \beta_2 TG._{it} + \beta_3 Z_{it} + \beta_4 (PB \times TG)_{it} + \beta_5 (PB \times Z)_{it} + \beta_6 (TG \times LA)_{it} + \beta_7 (PB \times TG \times Z)_{it} + X'_{it} + e_{it} \qquad (6.2)$$

Here, *Z* is a dummy for respondents' observable characteristics, $(PB \times TG \times Z)$ is the *DiD* estimator given the interaction of the post bushfire dummy, bushfire affected dummy and observable characteristic dummy. The coefficient associated with the triple interaction term (β_7) captures the additional effects on health outcomes belonging to a defined subgroup of *Z*. The coefficient of *PB* and *TG* interaction term is then the *DiD* estimator for the reference category of *Z*.

6.4 Results

6.4.1 Descriptive Statistics

Table 6.2 presents the mean values of dependent variables and covariates for the treated and control sample. The mean value of general and physical health variables decreased

in the post bushfire period in the treated group compared to the pre-bushfire period. Likewise, the proportion of individuals with breathing problems increased by 2.3% after the bushfires. Comparing the means of other variables shows that the treated sample and the control sample are relatively homogeneous. For example, both samples have approximately 50% of female respondents. However, the mean ages of the respondents for treated and control samples are 33 and 37 years, respectively, and family income is distinct across the two samples.

Table 6.2 Descriptive Statistics for the Samples

	Treated (n= 4501)			Control (n= 36549)		
Variables	Pre	Post	Total	Pre	Post	Total
General Health	69.871	68.023	68.939	69.113	69.245	69.177
Physical Health	85.384	83.885	84.628	84.183	84.609	84.389
SAH	2.562	2.584	2.574	2.561	2.507	2.535
Breathing Problems	0.077	0.100	0.089	0.125	0.124	0.125
Get Sick Easier	4.227	4.177	4.202	4.158	4.145	4.152
Regional			0.592			0.330
Female			0.503			0.517
Age			32.575			36.745
Refugee			0.013			0.016
Indigenous			0.012			0.005
Australia			0.841			0.788
English			0.930			0.876
Income (Family)			45890			55642
Bachelor and Above			0.142			0.256
Year 12 and Adv Dip			0.498			0.426
Year 11 and Below			0.359			0.318
Undetermined			0.001			0.000

Note: In this table, the first two columns present the means for the treated sample and the last three columns present the means for the control sample. For the health outcomes, means are presented for both pre- and post-bushfire periods.

6.4.2 Regression Results

Table 6.3 presents the results of the regression of the Eq. (6.1) and (6.2) for our markers of health. The *DiD* estimators of Model 2 across all the health outcomes show a negative effect of bushfire smoke on individuals' health. For example, there is a significant (at 10% level) decrease of approximately1.8 points in general health and 1.6 points in physical health measures following exposure. Likewise, people are more likely to assess their health as poor after the bushfire if they were exposed to bushfire smoke. This suggests that people's general health and physical health measures in the affected area decrease after the bushfires than the same health status of the people in an unaffected

area. Nonetheless, indicators for “breathing problems” and “the gets sick a little easier than others” have an insignificant effect.

Table 6.4 presents the interaction variables’ (in Eq. [6.2]) coefficients of interest. Our results reveal some underlying heterogeneity in the health impact of bushfires among different socioeconomic groups. Interestingly, compared to the lower-income groups, relatively higher-income groups are more likely to have adverse health effects. This association holds in both urban and total samples and is significant across most of our health outcomes, while major cities report the highest impact (e.g., general health of the individuals in lower incomes group is 3.2 and 5.9 points higher than those are in relatively higher income group in the total sample and major city respectively). However, people in the upper-income quantiles have a lower chance of getting sick after bushfires than those in the lower-income quantiles (see coefficient of lower-income people in the model for binary health outcome get sick easier is -0.116). Considering the level of education, we show that individuals with relatively low education are more likely to report ill-health due to bushfires. Moreover, smokers and individuals aged < 45 years are particularly vulnerable.

Table 6.3 OLS Regression Results for Diff-in-Diff and Triple Differences Designs

	General Health			Physical Health			SAH		
	Model (1)	Model (2)	Model (3)	Model (1)	Model (2)	Model (3)	Model (1)	Model (2)	Model (3)
Time	-0.473*	-0.693	-0.488	0.748	-0.628	-0.621	-0.065***	-0.007	-0.007
Treatment	1.211**	0.83	0.789	1.205**	0.524	0.585	0.001	-0.027	-0.03
DiD Estimator (γ_1)	-1.584**	-1.839**		-1.928**	-1.417		0.076**	0.074*	
DiD Estimator (β_4)			-2.966***			-3.005***			0.153***
DiD # regional (β_7)			2.050*			2.870***			-0.142***
Regional		1.302***	1.146***		1.046***	0.828***		-0.022*	-0.011
Female		-0.578**	-0.579**		-2.851***	-2.853***		0.051***	0.051***
Age		0.038	0.035		-1.730***	-1.735***		-0.010***	-0.010***
Age Square		-2.816***	-2.777***		16.633***	16.689***		0.315***	0.312***
Refugee		-2.423**	-2.449**		-1.049	-1.083		0.074	0.075
Indigenous		-0.498	-0.565		-1.925	-2.021		0.074	0.079
Australia		-1.589***	-1.579***		-1.371***	-1.357***		0.041**	0.041*
English		4.124***	4.128***		4.577***	4.584***		-0.182***	-0.182***
Log Income (Family)		3.833***	3.833***		4.268***	4.269***		-0.192***	-0.192***
Education (c)									
Year 12 and Adv. Dip		-2.529***	-2.519***		-3.275***	-3.261***		0.228***	0.227***
Year 11 And Below		-4.486***	-4.470***		-6.168***	-6.146***		0.300***	0.299***
Undetermined		-11.273	-11.409		10.750***	10.561***		0.984***	0.994***
Constant	68.665***	44.978***	44.894***	83.833***	8.967**	8.852**	2.569***	2.913***	2.919***
R-squared	0.000	0.066	0.066	0.001	0.246	0.247	0.002	0.136	0.136
N	109434	21282	21282	26879	21287	21287	26875	21262	21262

Note: * p<0.10, ** p<0.05, ***p<0.01. Reference categories are urban, male, non-refugee, non-indigenous, born in/out of Australia, the first language is not English, and bachelor and above (c). This table presents the DiD regression coefficients of covariates. Model 2 represents OLS regression results based on Eq (6.1). Model 3 represents OLS regression results based on Eq (6.2). In Model 2, the DDD term is the interaction of regional area dummy, time and treatment. Here we have considered five different health outcomes. Columns 2, 3, and 4 belong to general health. Columns 5–7 belong to physical health. The last three columns are for SAH. We estimated those models using OLS with time-fixed effects and used heteroskedasticity-robust standard errors throughout.

Table 6.3 (Continued)

	Breath Problem			Get Sick Easier		
	Model (1)	Model (2)	Model (3)	Model (1)	Model (2)	Model (3)
Time	0.019	0.009	0.01	-0.044*	-0.034	-0.034
Treatment	-0.048***	-0.047**	-0.045**	0.069**	0.081**	0.082**
DiD Estimator (γ_1)	0.024	0.031		-0.037	-0.061	
DiD Estimator (β_4)			-0.034			-0.085
DiD # regional (β_7)			0.108***			0.043
Regional		0.005	-0.003		0.092***	0.089***
Female		0.021***	0.021**		-0.155***	-0.155***
Age		0.004**	0.004**		-0.008**	-0.008**
Age Square		-0.026	-0.026		0.193***	0.194***
Refugee		0.028	0.026		-0.114**	-0.114**
Indigenous		0.031	0.03		0.092	0.091
Australia		0.041***	0.042***		0.012	0.012
English		-0.050***	-0.050***		0.279***	0.279***
Log Income (Family)		-0.018***	-0.018***		0.161***	0.161***
Education (c)						
Year 12 and Adv. Dip		0.024**	0.025**		-0.053***	-0.053***
Year 11 And Below		0.050***	0.050***		-0.147***	-0.147***
Undetermined		-0.124***	-0.127***		0.056	0.053
Constant	0.110***	0.238**	0.239**	4.186***	1.386***	1.384***
R-squared	0.002	0.036	0.037	0.001	0.046	0.046
N	7681	6101	6101	26873	21300	21300

Note: * p<0.10, ** p<0.05, ***p<0.01. Reference categories are urban, male, non-refugee, non-indigenous, born in out of Australia, first language not English, and bachelor and above (c). Here, Columns 2, 3 and 4 belong to shortness of breath or difficulty breathing (long-term), and the last three columns are for the health outcome indicating whether the respondent "gets sick a little easier than others". We estimated those models using OLS with time-fixed effects and used heteroskedasticity–robust standard errors throughout.

6.4.2 Placebo Test for Robustness Check

To examine identification within the estimated model, we conducted placebo tests using data from the pre- bushfire period from 2001 to 2008. We re-estimate the models in the core analysis (Eq. [6.1] and Eq. [6.2]) with artificial exogenous bushfire shock that is placed in a year between 2001 and 2008. In this case, we placed artificial shock in 2004 and defined 2001–2003 as the pre-shock period and 2004 –2008 as the post-shock period. We keep the treatment group the same as the core research design. Table A6.6 in the appendix presents the results of the placebo regression models. We show that artificial shock does not decrease the health outcomes of the treatment group compared to the control group. However, the shock improves some health outcomes (e.g., general health, physical health, and getting sick easily) of the individuals in the treatment group.

Table 6.4 Regression Results for Triple Differences Designs with Different Observable Characteristics

	General Health		Physical Health		SAH		Breath Problem		Get Sick Easier	
	Total Sample	Major City	Total Sample	Major City	Total Sample	Major City	Total Sample	Major City	Total Sample	Major City
Eq. (2) DiD Estimator (Post Bushfire × Bushfire Affected) Given:										
1. Income: Lowest (First Quantile) (β_7)	3.187**	5.879***	1.193	-2.956	-0.208***	-0.259**	-0.008	-0.116***	0.088	-0.116***
Income: Above First Quantile (β_4)	-2.345***	-2.792**	-1.557*	-2.070*	0.106***	0.104*	0.026	0.002	-0.043	-0.067
2. Education: Grade 12 or Above (β_7)	0.881	1.924	-3.490***	-1.437	0.056	0.051	-0.049	0.087***	0.199***	0.271***
Grade 11 or Bellow (β_4)	-2.395*	-3.505*	0.847	-1.983	0.031	0.032	0.056*	-0.072*	-0.198***	-0.273***
3. Smoke (β_7)	-5.246***	-0.455	-4.707***	-4.541*	0.206***	0.076	0.107**	-0.018	-0.151**	0.114
Non-Smoke (β_4)	-0.78	-2.069	-0.626	-2.19	0.028	0.056	0.003	-0.019	-0.032	-0.1
4. Age: Above 45 (β_7)	3.055***	1.588	1.476	1.69	-0.167***	-0.102	-0.132***	-0.157***	0.08	-0.038
Age: 15-44 (β_4)	-3.192***	-2.642*	-2.316**	-3.593**	0.148***	0.106	0.110***	0.083*	-0.097*	-0.065

Note: * p<0.10, ** p<0.05,*** p<0.01. This table presents DiD (β_4) and DDD (β_7) estimates (Eq 6.2) for our health outcomes separately, considering different observable characteristics. We obtained these estimates for the total and the major city samples. The total sample includes respondents from regional and urban areas. Respondents from urban areas included in the major city sample. We estimated those models using OLS with time-fixed effects and used heteroskedasticity-robust standard errors throughout all models. The first column presents the different scenarios according to the observable characteristics.

6.5 Discussion

Our empirical results show that people exposed to bushfire smoke are more likely to have health problems. Although some existing empirical findings on the health impact of bushfire smoke (Dennekamp et al., 2015; Franzi et al., 2011; Hamon et al., 2018; Kim et al., 2018; Navarro et al., 2019; Pavagadhi et al., 2013) are similar, we show the effect of air pollution caused by bushfires on individuals' health is robust by using five different health outcomes and applying the quasi-experimental setting with a large panel data set.

Moreover, we examine heterogeneity in effect sizes across various sociodemographic subgroups of the Australian population. For this analysis, we consider respondents' living area, age, level of education, income and whether they smoke. We show that people who live in major cities are relatively more vulnerable and attribute this to pre-existing variations in exposure to air pollution (see the models of urban sample in Table 4). We also show that younger and middle-aged socioeconomically disadvantaged people may become more vulnerable during a bushfire episode (see the estimates of the total and urban samples in Table 4). Some empirical research (Barnes et al., 2019; Mitchell and Dorling, 2003) shows that young and early middle ages in disadvantaged households are more likely to be exposed to polluted air because they are more likely to settle in highly polluted areas. Iversen et al. (2005) find that living in an urban area is associated with a higher prevalence of respiratory health issues for this precise reason. Therefore, we suggest that urban disadvantaged individuals are more likely to experience pollution-related health issues than advantaged individuals. This is in line with some previous studies (e.g., Jephcote et al., 2016; Namdeo and Stringer, 2008).

In contrast, we show that bushfire pollution's health effect is lower for lower-income individuals than for individuals above our higher income threshold. The conflicting results for income and education may have several sources. While statistical noise or econometric misspecification may account for these results, there may be omitted health behaviours (e.g., related to diet or exercise) that are highly correlated with education but only weakly associated with income, which may introduce this heterogeneity (Cutler and Lleras-Muney, 2010; Laaksonen et al., 2003). In Australia, there are only small income differentials associated with social class but large cultural differences that are plausibly correlated with health behaviour.

In addition, we suggest that the health impact of air pollution significantly increases in bushfire-affected areas because (i) mixing the composition of bushfires and human-caused emissions increases the toxicity of air pollution and (ii) increasing the level of greenhouse gas increases atmosphere temperature.

It is plausible that bushfire smoke is equally or more dangerous to human health compared to industrial emissions. Polluted urban air contains toxic chemicals and particles such as PM10, PM2.5, CO, CO2 nitrogen dioxide (NO2), sulfer dioxide (SO2), ozone (O3) and volatile organic compounds emission (U.S. Department of Transportation, 2016). Bushfires dump a large amount of polluted air, including greenhouse gases, photochemically reactive compounds, nonmethane volatile organic carbon and fine and coarse particulate matter into the atmosphere in a short period (see Di Virgilio et al., 2021; Paton-Walsh et al., 2012; Teague et al., 2009). Also, compared to the urban ambient PM, bushfire PM contains a higher concentration of Organic Carbon (OC), Elemental Carbon (EC), and Polycyclic Aromatic Hydrocarbons (PAH) (Liu and Peng, 2019). Therefore, the toxicity of wildfire PM can be higher than the ambient PM (Franzi et al., 2011; Santoso et al., 2019).

Another interactive effect may occur from exposure to excessive heat. Higher pollution concentrations in cities are associated with increasing atmospheric temperature (Tibbetts, 2015). The combined effect of higher air temperature and air pollution could potentially affect human health more than the individual effect (Patel et al., 2019; Walter et al., 2020). Since there are meaningful socioeconomic differences between urban and rural areas, we also expect to see correlations between these socioeconomic factors and sensitivity to bushfire pollution.

6.6 Conclusion

Applying a difference-in-difference research design to the 2009 Black Saturday wildfire incident in Australia, this chapter evaluated the effect of air pollution on human health. We showed that health status (general health, physical health and SAH) is predicted to be less for the people affected by bushfire pollution than those in unaffected areas. Further, we found that the negative health effects are less for the people in regional areas and that smokers, less educated and young individuals are relatively vulnerable.

Our result implies that socioeconomic and demographic disparities play a role in the distribution of sensitivity to air pollution exposure. Therefore, we recommend that the socioeconomic inequalities in resource allocation and individuals' behaviours be considered when addressing the health issues related to air pollution. Moreover, this study opens up further lines of research to determine which group of people are more likely to have adverse health impacts from air pollution

Appendix

Table A 6.1 OLS Regression Results for Eq (6.2) – Observable characteristics: Lowest Income (First Quantile)

	General Health		Physical Health		SAH		Breath Problem		Get Sick Easier	
	Full Sample	Major City	Full Sample	Major City	Full Sample	Major City	Full Sample	Major City	Full Sample	Major City
Time	1.168**	1.241*	1.615***	1.968***	-0.099***	-0.107***	0.002	0.007	-0.005	0.009
Treatment	0.436	-0.629	0.638	0.121	0.002	0.092**	-0.041***	-0.041	0.042	0.032
DiD Estimator (β_4)	-2.345***	-2.792**	-1.557*	-2.070*	0.106***	0.104*	0.026	0.002	-0.043	-0.067
DiD # Lowest Income(β_7)	3.187**	5.879***	1.193	-2.956	-0.208***	-0.259**	-0.008	-0.116***	0.088	0.167
Lowest Income	-6.100***	-5.753***	-7.885***	-8.697***	0.277***	0.262***	0.039***	0.031**	-0.290***	-0.288***
Regional	0.979***		0.915***		-0.006		0.001		0.091***	
Female	-0.560**	-0.644**	-2.739***	-2.790***	0.052***	0.057***	0.014*	0.003	-0.151***	-0.169***
Age	-0.169***	-0.177***	-0.451***	-0.457***	0.014***	0.014***	0.002***	0.003***	0.007***	0.008***
Refugee	-2.764***	-4.660***	-2.343**	-3.289**	0.096**	0.170***	0.023	0.043	-0.119**	-0.152**
Indigenous	0.082	1.131	-4.018*	-3.585	0.039	0.069	0.052	0.04	0.098	0.216**
Australia	-1.161***	-0.197	-1.365***	-0.279	0.028	-0.032	0.030***	0.030**	0.01	0.058**
English	4.271***	3.562***	4.625***	3.640***	-0.191***	-0.136***	-0.037**	-0.013	0.299***	0.282***
Education (c)										
Year 12 & Adv. Dip	-3.001***	-3.096***	-4.540***	-4.442***	0.238***	0.215***	0.022**	0.022**	-0.091***	-0.090***
Year 11 & Below	-4.693***	-4.393***	-8.930***	-8.722***	0.289***	0.260***	0.051***	0.057***	-0.199***	-0.185***
Undetermined	-10.565	-6.438	9.813***	11.506***	0.863***	0.728***	-0.118***	-0.122***	0.078	0.223
Constant	76.508***	77.050***	107.140***	107.495***	1.881***	1.858***	-0.043**	-0.080***	3.748***	3.730***
R-squared	0.061	0.065	0.232	0.244	0.127	0.135	0.036	0.044	0.046	0.048
N	26824	17525	26839	17547	26835	17531	7675	4792	26833	17522

Note: * p<0.10, ** p<0.05, ***p<0.01. Reference categories are urban, male, non-refugee, non-indigenous, born in/out of Australia, first language is not English, and bachelor and above (c). This table presents the DDD regression coefficients of covariates for the total sample and major city separately. The DDD term is the interaction of the lowest income dummy, time and treatment. Here we have considered five different health outcomes. We estimated those models using OLS with time fixed effect and used heteroskedasticity-robust standard errors throughout.

Table A 6.2 OLS Regression Results for Eq (6.2) – Observable Characteristics: Highest Education (Grade 12 or above)

	General Health		Physical Health		SAH		Breath Problem		Get Sick Easier	
	Full Sample	Major City	Full Sample	Major City	Full Sample	Major City	Full Sample	Major City	Full Sample	Major City
Time	-0.469	0.161	-0.566	-0.173	-0.021	-0.044	0.009	0.005	-0.027	-0.002
Treatment	0.517	-0.497	0.806	0.734	0.002	0.099*	-0.047**	-0.054*	0.082**	0.07
DiD Estimator (β_4)	-2.395*	-3.505*	0.847	-1.983	0.031	0.032	0.056*	-0.072*	-0.198***	-0.273***
DiD # Grade 12 or Above (β_7)	0.881	1.924	-3.490***	-1.437	0.056	0.051	-0.049	0.087***	0.199***	0.271***
Grade 12 or Above	3.103***	2.813***	6.837***	6.882***	-0.152***	-0.148***	-0.033***	-0.040***	0.148***	0.139***
Regional	0.762**	0	0.505*	0	0.011	0	0.008	0	0.073***	0
Female	-0.563**	-0.616*	-2.626***	-2.625***	0.047***	0.053***	0.021**	0.011	-0.154***	-0.170***
Age	-0.195***	-0.203***	-0.472***	-0.481***	0.015***	0.015***	0.002***	0.003***	0.006***	0.006***
Refugee	-2.444**	-4.313***	-1.424	-2.642*	0.081	0.160***	0.026	0.044	-0.114**	-0.147**
Indigenous	-0.866	0.328	-3.777*	-5.224	0.087	0.12	0.036	0.046	0.06	0.183
Australia	-1.738***	-0.646	-1.695***	-0.509	0.049**	-0.013	0.043***	0.042***	0.004	0.044
English	4.778***	4.056***	5.174***	4.283***	-0.216***	-0.167***	-0.056***	-0.03	0.304***	0.299***
Family Income	0.000***	0.000***	0.000***	0.000***	-0.000***	-0.000***	-0.000***	-0.000***	0.000***	0.000***
Constant	71.139***	71.862***	96.210***	96.758***	2.220***	2.158***	0.021	-0.007	3.477***	3.475***
R-squared	0.055	0.059	0.218	0.224	0.116	0.126	0.035	0.044	0.039	0.041
N	21282	13962	21287	13978	21262	13947	6101	3802	21300	13963

Note: * p<0.10, ** p<0.05, ***p<0.01. Reference categories are urban, male, non-refugee, non-indigenous, born in/out of Australia and first language is not English. This table presents the DDD regression coefficients of covariates for the total sample and major city separately. The DDD term is the interaction of the highest education dummy, time and treatment. Here we have considered five different health outcomes. We estimated those models using OLS with time fixed effect and used heteroskedasticity-robust standard errors throughout.

Table A 6.3 OLS Regression Results for EQ (6.2) – Observable Characteristics: Smoke (At Least Occasionally)

	General Health		Physical Health		SAH		Breath Problem		Get Sick Easier	
	Full Sample	Major City	Full Sample	Major City	Full Sample	Major City	Full Sample	Major City	Full Sample	Major City
Time	-0.672	-0.002	-0.674	-0.263	-0.011	-0.036	0.01	0.006	-0.031	-0.005
Treatment	0.901	-0.053	1.227*	1.316	-0.021	0.072	-0.049**	-0.055*	0.092***	0.08
DiD Estimator (β_4)	-0.78	-2.069	-0.626	-2.19	0.028	0.056	0.003	-0.019	-0.032	-0.1
DiD # Smoke (β_7)	-5.246***	-0.455	-4.707***	-4.541*	0.206***	0.076	0.107**	-0.018	-0.151**	0.114
Smoke	-7.212***	-6.446***	-2.832***	-2.279***	0.349***	0.306***	0.014	-0.008	-0.137***	-0.103***
Regional	1.071***		0.853***		-0.01		0.006		0.081***	
Female	-0.943***	-0.939***	-2.988***	-2.960***	0.070***	0.072***	0.024***	0.013	-0.164***	-0.177***
Age	-0.218***	-0.221***	-0.485***	-0.489***	0.016***	0.016***	0.002***	0.003***	0.006***	0.006***
Refugee	-2.555**	-4.448***	-1.222	-2.491*	0.081	0.163***	0.027	0.044	-0.115**	-0.149**
Indigenous	0.951	2.495	-2.771	-4.243	-0.012	0.014	0.038	0.047	0.097	0.221*
Australia	-1.763***	-0.615	-1.655***	-0.426	0.048**	-0.015	0.043***	0.042***	0.005	0.049*
English	4.958***	4.102***	5.227***	4.259***	-0.223***	-0.170***	-0.057***	-0.028	0.307***	0.296***
Family Income	0.000***	0.000***	0.000***	0.000***	-0.000***	-0.000***	-0.000***	-0.000***	0.000***	0.000***
Education (c)										
Year 12 & Adv. Dip	-2.095***	-2.245***	-4.357***	-4.513***	0.199***	0.181***	0.026**	0.032***	-0.070***	-0.068***
Year 11 & Below	-4.193***	-3.999***	-9.297***	-9.476***	0.261***	0.242***	0.053***	0.058***	-0.201***	-0.190***
Undetermined	-9.355	-5.697	9.961***	11.971***	0.875***	0.768***	-0.129***	-0.132***	0.077	0.234
Constant	78.203***	78.294***	107.153***	107.510***	1.807***	1.790***	-0.040*	-0.068**	3.727***	3.698***
R-squared	0.079	0.076	0.228	0.234	0.147	0.15	0.038	0.045	0.043	0.043
N	21282	13962	21287	13978	21262	13947	6101	3802	21300	13963

Note: * p<0.10, ** p<0.05, ***p<0.01. Reference categories are urban, male, non-refugee, non-indigenous, born in/out of Australia, first language is not English, and bachelor and above (c). This table presents the DDD regression coefficients of covariates for the total sample and major city separately. The DDD term is the interaction of dummy variable for the smoke respondent, time and treatment. Here we have considered five different health outcomes. We estimated those models using OLS with time fixed effect and used heteroskedasticity-robust standard errors throughout.

Table A 6.4 OLS Regression Results for EQ (6.2) – Observable Characteristics: Age (Above 45 years old)

	General Health		Physical Health		SAH		Breath Problem		Get Sick Easier	
	Full Sample	Major City	Full Sample	Major City	Full Sample	Major City	Full Sample	Major City	Full Sample	Major City
Time	-0.465	0.202	-0.605	-0.12	-0.018	-0.045	0.01	0.005	-0.029	-0.005
Treatment	0.71	-0.249	1.145	1.417	-0.015	0.073	-0.052***	-0.067**	0.090***	0.078
DiD Estimator (β_4)	-3.192***	-2.642*	-2.316**	-3.593**	0.148***	0.106	0.110***	0.083*	-0.097*	-0.065
DiD # Age Above 45 (β_7)	3.055***	1.588	1.476	1.69	-0.167***	-0.102	-0.132***	-0.157***	0.08	-0.038
Age_above45	-6.882***	-7.020***	-14.073***	-13.889***	0.469***	0.485***	0.083***	0.096***	0.187***	0.187***
Regional	0.938***	0.000	0.778**	0	-0.002	0	0.005	0	0.078***	0
Female	-0.804***	-0.897***	-3.052***	-3.134***	0.065***	0.073***	0.020**	0.013	-0.157***	-0.172***
Refugee	-2.432**	-4.334***	-1.814	-3.208**	0.082	0.167***	0.022	0.039	-0.101*	-0.136**
Indigenous	-0.006	1.018	-0.788	-3.03	0.004	0.057	0.017	0.04	0.032	0.166
Australia	-1.337***	-0.368	-0.391	0.563	0.01	-0.041	0.033***	0.033**	-0.006	0.039
English	4.710***	4.049***	4.962***	4.218***	-0.209***	-0.167***	-0.057***	-0.032*	0.304***	0.294***
Family Income	0.000***	0.000***	0.000***	0.000***	-0.000***	-0.000***	-0.000***	-0.000***	0.000***	0.000***
Education (c)										
Year 12 & Adv. Dip	-2.748***	-2.826***	-4.187***	-4.342***	0.225***	0.203***	0.030***	0.032***	-0.093***	-0.086***
Year 11 & Below	-4.848***	-4.481***	-9.443***	-9.584***	0.294***	0.266***	0.063***	0.066***	-0.222***	-0.205***
Undetermined	-10.694	-6.198	7.308***	8.858***	0.965***	0.838***	-0.115***	-0.118***	0.078	0.274
Constant	70.348***	70.522***	90.335***	90.636***	2.376***	2.362***	0.048**	0.024	3.902***	3.885***
R-squared	0.054	0.058	0.17	0.175	0.103	0.112	0.029	0.036	0.036	0.038
N	21282	13962	21287	13978	21262	13947	6101	3802	21300	13963

Note: * p<0.10, ** p<0.05, ***p<0.01. Reference categories are urban, male, non-refugee, non-indigenous, born in/out of Australia, first language is not English, and bachelor and above (c). This table presents the DDD regression coefficients of covariates for total sample and major city separately. The DDD term is the interaction of dummy variable for respondent 45 years old or above, time and treatment. Here we have considered five different health outcomes. We estimated those models using OLS with time fixed effect and used heteroskedasticity-robust standard errors throughout.

Table A 6.5 Lags and Leads Effects

	General Health	Physical Health	SAH	Breath Problem	Get Sick Easier
Time	-0.76	-0.285	0.022	-0.043	-0.005
Treatment	2.365**	2.167*	-0.023	0.098*	-0.007
Lead 1	-0.005	1.138	0.005	-0.029	0.05
Lead 2	-1.638	-1.251	0.019	0.038	-0.021
DiD	-1.325	-2.231	0.029	-0.097	-0.044
Lag 1	-1.026	1.761	0.074	0.047	-0.007
Lag 2	0.000	0.000	0.000	0.000	0.000
Constant	69.566***	84.850***	2.538***	4.192***	0.093***
N	11214	11221	11186	11229	3113

Note: * p<0.10, ** p<0.05, ***p<0.01. This table presents the *DiD* regression coefficients of covariates, including two leads and lags. We estimated those models using ordinary least squares and used heteroskedasticity-robust standard errors throughout.

Table A 6.6 OLS Regression: Placebo Test for Robustness

	General Health		Physical Health		SAH		Breath Problem		Get Sick Easier	
	Model (1)	Model (2)	Model (1)	Model (2)	Model (1)	Model (2)	Model (1)	Model (2)	Model (1)	Model (2)
Time	-0.908***	-0.910***	0.404	0.404	0.041***	0.041***	0.020	0.020	-0.085***	-0.085***
Treatment	-1.487**	-1.451*	-1.233	-1.223	0.043	0.040	-0.012	-0.012	-0.094**	-0.094**
DiD Estimator (γ_1)	2.195**		2.074**		-0.049		-0.028		0.145***	
DiD Estimator (β_4)		1.213		1.807		0.029		-0.034		0.140**
DiD # regional (β_7)		1.566		0.426		-0.124**		0.010		0.007
Regional	0.483	0.378	0.828***	0.800***	-0.002	0.006	-0.003	-0.004	0.076***	0.075***
Female	-0.177	-0.179	-2.400***	-2.401***	0.025**	0.025**	0.009	0.009	-0.148***	-0.148***
Age	-0.183***	-0.183***	-0.483***	-0.483***	0.014***	0.014***	0.003***	0.003***	0.007***	0.007***
Refugee	-3.376***	-3.379***	-2.640**	-2.640**	0.088*	0.088*	0.058*	0.058*	-0.176***	-0.176***
Indigenous	0.545	0.488	-5.274	-5.287	-0.003	0.001	0.068	0.068	0.013	0.013
Australia	-0.568	-0.564	-0.402	-0.400	-0.004	-0.004	0.016	0.016	-0.005	-0.005
English	4.796***	4.802***	4.633***	4.634***	-0.203***	-0.204***	-0.026	-0.026	0.353***	0.353***
Income (Family)	0.000***	0.000***	0.000***	0.000***	-0.000***	-0.000***	-0.000***	-0.000***	0.000***	0.000***
Education (c)	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000
Year 12 & Adv. Dip	-2.471***	-2.465***	-4.188***	-4.187***	0.213***	0.212***	0.012	0.012	-0.054***	-0.054***
Year 11 & Below	-4.766***	-4.759***	-8.867***	-8.865***	0.316***	0.316***	0.044***	0.044***	-0.193***	-0.193***
Undetermined	-2.801	-2.832	1.654	1.646	0.508***	0.511***			0.261	0.261
Constant	75.138***	75.164***	104.969***	104.976***	1.947***	1.944***	-0.044*	-0.044*	3.702***	3.703***
R-squared	0.055	0.055	0.220	0.220	0.114	0.115	0.039	0.039	0.044	0.044
N	22925	22925	22881	22881	22975	22975.000	4662	4662	22936	22936

Note: * p<0.10, ** p<0.05, ***p<0.01. This table presents the placebo regression estimates. Model 1 represents DiD regressions' coefficients, while Model 2 represents DDD regressions coefficients. Here, we use artificial bushfire shock from 2004. Thus, our time dummy is defined for the time frame 2004–2008. Reference categories are urban, male, non-refugee, non-indigenous, born in/out of Australia, the first language is not English, and bachelor and above (c). We estimated those models using OLS with a time-fixed effect and used heteroskedasticity-robust standard errors throughout.

Chapter 7

Conclusion

7.1 Conclusion of Empirical Findings

This thesis focused on a broader theme of health inequality. This area of research has attracted much attention in recent years. This thesis contains four empirical chapters that investigate the different aspects of health inequality related to factors beyond, or of limited, individual control.

The first two empirical chapters (Three and Four) have examined the impact of inter-generational socioeconomic inequalities on Australian health based on the normative framework of IOP. Chapter 5 has investigated the role of maternal education in the association between HIV prevalence and awareness and knowledge about HIV/AIDS in Sub-Saharan Africa. The final empirical chapter has provided novel evidence for the health effects of air pollution.

To conduct these empirical investigations, we have drawn data from the Household, Income and Labour Dynamics in Australia (HILDA) panel and high-quality Demographics and Health Surveys (DHSs) data from 21 Sub-Saharan nations. The HILDA survey collects data from a nationally representative random sample of more than 17,000 individuals from more than 7,000 Australian households. It has, since 2001, collected data on individuals' health status, demographic characteristics, socioeconomic background and family history using face-to-face interviews and self-completed questionaries. DHSs are cross-sectional household surveys designed to choose a representative sample at the national, residential and regional levels (e.g., states) using a stratified two-stage cluster sampling setting and have been conducted separately in 98 low and middle-income counties. All these empirical investigations were carried out using standard econometrics methods. Model specifications for each study depended on the available data, objectives of the studies and theoretical frameworks. The main findings of four empirical studies covered by Chapters 3-6 are briefly summarised below.

Chapter Three examined the effects of various circumstance variables on bodyweight by using regression models applied to Australian microdata. The chapter interpreted the explained component from these models as a measure of inequality of opportunity (IOP); that is, the fraction of variation accounted for by factors beyond an individual's control,

such as their race, gender and inherited social class. Also, the chapter concluded that IOP concerning bodyweight is relatively small. Nevertheless, the previous investigations with different health outcomes (e.g., self-assessed health) reported a significantly higher proportion of IOP. This result explains that body weight is much more responsive to health behaviours than general health indicators, so the proportion explained by background variables is smaller. In addition, although our model showed relatively small IOP estimates, This study found evidence that some parental characteristics have intergenerational effects. Specifically, the SES of an individual's father appears to be a key determinant, with persons born to more advantaged fathers having a small health advantage in later life when considering weight-related health. However, since (i) markers of paternal SES tend to be positively correlated, and (ii) body composition is predictive of a spectrum of negative health outcomes, the aggregate effects of these socially determined inequalities can still be substantial. This study also demonstrated empirical links between circumstances and outcomes that are partially reflective of correlations between background and health behaviours. Moreover, the decompositions estimates based on the approach of Jusot et al. (2013) revealed that desirable background characteristics might produce health benefits that cannot be fully accounted for by improved lifestyle choices.

Using Australian data, chapter 4 presented the empirical evidence for multigenerational inequality of opportunity in health. Modelling the spectrum of mental and physical health outcomes, it showed that markers of grandparental socioeconomic status are predictive of their grandchildren's health outcomes, even after controlling for parental equivalent socioeconomic characteristics. The analysis included some econometric decompositions attributing explained inequalities to various clusters of variables related to intergenerational inequalities. Surprisingly, the results revealed that the contribution to the variation of the children's health is approximately equally weighted towards both parents and grandparents. This result suggested that more complicated causal flows are present beyond those implicit in standard intergenerational inequality models. Based on our empirical results, we offer two possible explanations for this result. Firstly, the grandparents may play an important role in caregiving, and better-educated grandparents may do a better job raising healthier children. The second is that higher socioeconomic status (SES) grandparents may generate different cultural attitudes in ways reminiscent

of social class. These attitudes may then affect various behaviours such as tobacco or alcohol consumption.

Chapter 5 presented evidence for the effects of knowledge of HIV/AIDS on HIV prevalence, instrumenting individuals' knowledge of HIV using the level of maternal education. This chapter used a relatively large data set from DHSs in 21 Sub-Saharan African counties and HIV test results of surveyed individuals who voluntarily attended testing clinics. The regression estimates indicated that the mother's level of education is a valid instrument for modelling HIV prevalence on HIV education or awareness, and there is a consistent causal relationship between HIV prevalence on HIV education or awareness across all the markers of HIV knowledge. The empirical finding of this chapter suggested that higher socioeconomic inequality in comprehensive knowledge about HIV transmission may play a significant role in this casual impact since knowledge can be derived through individuals' inherited background characteristics such as parental characteristics and gender and their present socioeconomic backgrounds, such as education and income.

Moreover, since educational outcomes transmit across generations, our results suggest that maternal education can become a safeguard that prevents HIV/AIDS. Individuals of educated mothers are more likely to have better education, which links with better employment, healthy lifestyles and better health throughout life than children of less-educated mothers. Thus, for HIV prevention, maternal education may have several impacts. Firstly, a higher level of offspring education correlates with higher HIV education. Secondly, a family with an educated mother may be an environment for sharing knowledge, children's social experiences and problems. Thirdly, the mother's comprehensive HIV awareness reduces mother-to-child transmission of the disease.

Chapter 6 applied a difference-in-difference (DiD) research design to the 2009 Black Saturday bushfires (BSB) in Australia and evaluated the effect of air pollution on human health. It showed that health status (such as general health, physical health and self-assessed health (SAH)) is relatively poor among people in bushfire pollution areas than those in unaffected areas. Since bushfires dump a large amount of polluted air, including greenhouse gases, photochemically reactive compounds, nonmethane volatile organic carbon and fine and coarse PM into the atmosphere in a short period, our results have

suggested that bushfire smoke is equally or more dangerous to human health than industrial emissions.

Moreover, this chapter examined whether heterogeneity of exposure to anthropogenic urban air pollution drives the effect of bushfire air pollution on human health. It found that the negative health effects are less for people in regional areas than those in urbanised areas. For this result, the variation of air pollution concentration between urban and regional areas may matter. For example, Iversen et al. (2005) revealed that living in an urban area is associated with a higher prevalence of respiratory health issues than rural residency because of the persistence of higher urban air pollution. In addition, our results showed that smokers, less educated, and younger individuals are relatively vulnerable.

7.2 Policy Implication and Recommendation

The study-specific policy implication and recommendations are outlined as follows.

1. Suppose the correlation between health behaviours and hereditary factors beyond an individual's control affects intergenerational health inequality. In that case, the policy for prioritising healthcare or compensation options based on a Roemerian approach (Roemer, 1998) would reduce unfair disparities in health. For instance, even after allowing for behaviours, individuals who suffer from unhealthy body weight disproportionately originate from disordered families with single-parent status and poor childhood economic backgrounds. These individuals did not control their parents' decisions nor their parents' education or occupation, so it is hard to hold them responsible for health problems caused by their unhealthy body weight. Hence, we propose prioritising them for the provision of health treatment because it may be desirable to promote those from disadvantaged backgrounds when treating lifestyle-related diseases in order to offset other predetermined inequalities.

2. Individuals who inherit favourable circumstances tend to consume healthier diets and engage in more physical activity, which would benefit their health. How we treat these path effects depends upon some subjective value judgements. On the one hand, some fraction of health behaviour is attributable to circumstances and, therefore, seemingly beyond the control of the individual. On the other hand, even if the discipline to lead a healthy lifestyle is predetermined, the effort to do so (and

subsequent disutility) is still experienced. Determining whether to further prioritise individuals who have exogenously driven lifestyle-related diseases remains an issue of practical importance for the healthcare sector.

3. Since ingrained socioeconomic (dis)advantages that persist over multiple generations may be indicative of "social class," we suggest that subtle attitudinal and behavioural characteristics associated with this variable may be a key factor in driving health disparities. Therefore, we recommend that policymakers should consider (1) attitudes and behaviours related to health that might be passed down when grandparents have close contact with grandchildren and (2) economic, social and cultural factors associated with social class, as proxied by the length of time a family has held a given level of social status. To address the first concern, providing better childcare facilities might be beneficial, particularly for disadvantaged children. Providing opportunities to increase lower-class children's educational attainment is highly recommended for the second concern.

4. The results in Chapter 4 have some general implications for measuring inequality of opportunity. It is common for IOP models (which are typically lower-bound estimates as socioeconomic constraints are only partially observable) to produce estimates that seem "too low." The decompositions results indicate that neglecting multigenerational factors may explain some of this missing inequality.

5. Evidence in Chapter 5 reveals a considerable decline in HIV prevalence among advantaged people during HIV/AIDS education and awareness programs (Santelli et al., 2021). (S. Gillespie et al., 2007). However, lower-educated individuals may take considerable time to understand the provided information and may sometimes misinterpret it (Kiviniemi et al., 2018) and have less confidence in discussing sexually transmitted diseases and accessing the health services provided. Consequently, the risk of the disease has shifted from higher to lower social classes. Based on this existing evidence and the results of this study, we argue that socioeconomic disparities in HIV education might be largely responsible for this inequality in HIV prevalence. Therefore, in the short term, expanding the opportunities for disadvantaged people to access HIV education

and the provision of health care concerning HIV/AIDS has implications for reducing the disease's prevalence.

6. The study in Chapter 6 demonstrates that rural communities have less impact of bushfires on health because they have better air quality than their counterparts in cities. This result implies that socioeconomic and demographic disparities play a role in the distribution of air pollution exposure. Therefore, we recommend that the socioeconomic inequalities in resource allocation and individuals' behaviours be considered when addressing the health issues related to air pollution.

7.3 limitation and Future Research Avenues

This thesis's empirical investigations have been restricted to the data from the HILDA panel and DHSs. Nonetheless, these limitations open avenues for further research. This section describes these research opportunities raised from the limitation of each empirical study.

In Chapter 3, we proxied circumstances using some of the parental characteristics and family backgrounds but could not consider possible observable circumstances such as genetic traits. Moreover, this analysis relied only on three waves of the HILDA panel since HILDA does not collect respondents' lifestyle data in every wave. Therefore, we strongly recommend extending this research using more observable circumstances proxies with a panel data set based on these limitations. In addition, this research sheds further light on the deltoid relationship between circumstances, effort and health.

To conduct our analysis in Chapter 4, we required a multigenerational dataset assembled from HILDA by matching observations across three consecutive generations. However, HIDLA does not directly collect grandparental data. Therefore, we must deal with a significant number of missing cases. The result can be more robust if future research can employ a rich multigenerational data set and apply the machine learning approach to estimate IOP rather than rely on parametric approaches. In addition, the empirical work in both Chapters 3 and 4 can be replicated for other developed and developing countries and for professionally measured health outcomes rather than self-reported measures to examine whether the results will be similar.

Our analysis in Chapter 5 is limited to cross-sectional data from 21 Sub-Saharan African countries, and knowledge of HIV has a causal impact on the prevalence of HIV. For

further research, we recommend investigating whether increasing knowledge decreases the HIV risk behaviours using the panel data structure. Considering the model specification of this chapter, we have introduced maternal education for instrumenting individuals' knowledge of HIV. However, including more parental characteristics such as maternal and paternal occupation and education as instruments is highly recommended.

To conduct our analysis in Chapter 6, we grouped the study sample into two subsamples: the treated sample and the control. The treated sample contains the affected respondents, while the rest belong to the control sample. However, since our data set does not recognise whether respondents were affected by the BSB, respondents in the treated sample were identified based on the respondents' residential postcode. As with all causal modelling, our results are dependent upon the exogeneity of treatment. This will hold if the pollution distributed by fires is independent of health. Clearly, there is some potential for individuals living in high-risk areas to have different health profiles to those living in major cities. While unobserved invariant characteristics will be differenced away in our models, any changes in these unobserved traits could still potentially confound our analysis. Further, we intend for our results to be broadly useful for understanding the health implications of carbon pollution. However, the external consistency of our results (i.e., the extent that they can be generalised to other contexts) is unestablished. Our sample is also limited to respondents aged 15 or over. Although the volume of research has examined the health impact of air pollution, the analysis in Chapter 6 opens up further lines of research to not only examine the impact of air pollution on health but also to determine which group of people are more likely to have adverse health impacts due to air pollution. Also, consider the possible identification problem when air pollution can not be measured as an independent shock. For example, when studying the ambient air pollution in cities, it is important to consider that the health effects are related to the destruction of infrastructure or the stress of living in a hazardous area.

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