Socioeconomic status, economic insecurity and the obesity transition in South Africa: generational and life course aspects

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DECLARATION

By submitting this thesis electronically, I declare that all work contained in this document is original, and I am the sole author. Where others have contributed to this work, I have explicitly declared the nature of their contribution and the percentage of their efforts to the work. Furthermore, I have not previously submitted this work, or any parts of this work, to obtain a qualification elsewhere.

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Nature of my contribution	Extent
Conceptualisation, cleaned and analysed data, literature review and write-up of paper.	90 %

The following co-authors have contributed to paper 1.

Name		Nature of the contribution	Extent
Prof. Dieter von Fintel		Assisted with framing and choice of methodology, reviewed and commented on drafts.	10%

Paper 2

Nature of my contribution	Extent
Conceptualisation, cleaned and analysed data, literature review and write-up of paper.	90 %

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Nature of my contribution	Extent
Conceptualisation, cleaned and analysed data, literature review and write-up of paper.	95 %

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1. The declaration above accurately reflects the nature and extent of the contributions of the candidate and the co-author to the papers included in this dissertation as chapter 2, chapter 3, and chapter 4.

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Nine years ago I sat in my econometrics lecturer's office, expressing my struggles and doubts over whether I should even be there doing my Masters at all – whether I had what it takes. He said three words to me that I have carried with me in the face of similar doubts ever since: "you've got it". It is no coincidence that I now submit my PhD nine years later under his supervision. To Prof. Dieter von Fintel, thank you for believing that I could do it, and helping me to believe it too. Thank you for your patient support, careful comments and advice, interesting discussions, and for pushing me to challenge myself and to push the boundaries of what I thought I was capable of.

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ABSTRACT

Worldwide countries are undergoing the 'nutrition transition' – a shift towards diets high in saturated fat, sugar and cheap processed energy-dense foods, with a corresponding increase in rates of obesity. The rich tend to move through the transition ahead of the poor, and with this the burden of obesity tends to shift from the rich to the poor as countries develop, in a process that has been called the 'obesity transition'. This dissertation explores several aspects of the social gradient in body weight in South Africa using the nationally representative National Income Dynamics Study (NIDS) data, proposing that changes across and between generations and over the life course may be one of the drivers of the shift from one stage of the obesity transition to the next.

Chapter 2 explores the possibility that childhood socioeconomic status (SES) and intergenerational mobility may contribute to the reversal of the social gradient in body weight. I find that upward social mobility is associated with increased obesity risk in adulthood compared to individuals who maintained a stable high SES in childhood and adulthood. Furthermore, the social gradient in body mass index (BMI) is flatter among individuals from a high SES childhood background, and already appears to have reversed among women with a high childhood SES who also have a high SES in adulthood. These findings shed light on the future of the obesity transition in South Africa; they suggest that it may take more than one generation of sustained high SES – or perhaps of adequate childhood nutrition – before we see higher adult SES have a protective effect against obesity, and with it a plateau in obesity rates.

Chapter 3 investigates generational aspects of the obesity transition, proposing that younger generations may be the first to see a reversal in the social gradient in body weight as they experience the benefits of upward intergenerational mobility and with it new circumstances and attitudes. I use a machine learning algorithm to find structural breaks in the social gradient in BMI by birth year. I find that the social gradient in BMI is flatter among younger cohorts of South African men, and find some indications that it is flatter among younger cohorts of women too, suggesting that the obesity transition may be driven in part by changes across generations.

Chapter 4 asks whether economic insecurity is more strongly associated with body weight for those with higher levels of income, which yield increased access to excess energy. I find evidence that economic insecurity is more likely to be associated with higher BMI among higher-income women, while economic insecurity is not associated with higher BMI for men.

These results suggest that, given continuing high rates of undernutrition in early life and rising living standards, obesity rates in South Africa are likely to continue to rise, particularly for those from low-SES childhood backgrounds. This calls for policies to attempt to reduce consumption of unhealthy foods, and to improve nutrition in childhood – particularly in the earliest years of life.

Key words:

Nutrition transition; obesity transition; childhood SES; social mobility; age-period-cohort; economic insecurity.

OPSOMMING

Lande wêreldwyd ondergaan tans die 'voedingsoorgang' – 'n verskuiwing na diëte hoog in versadigde vet, suiker en goedkoop geprosesseerde energie-digte kosse, met 'n verwante toename in die voorkoms van vetsug. Die rykes neig om voor die armes deur die oorgang te gaan, en dus is die vetsuglading besig om te skuif van die rykes na die armes tesame met ekonomiese ontwikkeling, in 'n proses wat die 'vetsugoorgang' genoem word. Hierdie proefskrif ondersoek verskeie aspekte van die sosiale gradiënt in liggaamsgewig in Suid-Afrika deur die gebruik van die nasionaal-verteenwoordigende NIDS data. Dit stel voor dat veranderinge oor en tussen geslagte, asook veranderinge in die lewenssiklus een van die oorsake kan wees van die verskuiwing van een fase van die vetsugoorgang tot die volgende.

Hoofstuk 2 ondersoek die moontlikheid dat kinders se sosio-ekonomiese status (SES) en intergeneratiewe mobiliteit tot die omkeer van die sosiale gradiënt in liggaamsgewig bydra. Ek het gevind dat opwaartse sosiale mobiliteit met hoër risiko van vetsug geassosieer is, in vergelyking met individue wie 'n stabiele hoë SES in beide die kinderjare en volwassenheid behou. Verder is die sosiale gradiënt in die liggaamsmassa-indeks (LMI) platter tussen individue van 'n hoë-SES kinderjarige agtergrond, en blyk asof dit reeds omgekeer het tussen vroue met 'n hoë kinderjarige SES wie ook 'n hoë SES in volwassenheid het. Hierdie bevindinge dui daarop dat dit meer as een geslag van volhoue hoë SES mag neem, óf moontlik van voldoende voeding in die kinderjare, voor hoër volwasse SES met laer risiko van vetsug geassosieer word, en Suid-Afrika daarmee 'n plato in die voorkoms van vetsug bereik.

Hoofstuk 3 ondersoek generasie-aspekte van die vetsugoorgang. Dit stel voor dat jonger geslagte die eerste mag wees om 'n omkeer van die sosiale gradiënt in liggaamsgewig te ondervind, as hulle die voordele van opwaarts intergeneratiewe mobiliteit en nuwe omstandighede ervaar, en nuwe houdings ontwikkel. 'n Masjienleer algoritme word gebruik om strukturele breke in die sosiale gradiënt in LMI per geboortejaar te vind. Ek bevind dat die sosiale gradiënt in LMI platter onder jonger kohorte van Suid-Afrikaanse mans is, en vind sommige aanduidings dat dit ook platter onder jonger kohorte van vroue is. Dit dui daarop dat die vetsugoorgang gedeeltelik deur veranderinge oor generasies gedryf mag wees.

Hoofstuk 4 vra of ekonomiese onsekerheid 'n sterker assosiasie het met liggaamsgewig vir dié met hoër inkomstevlakke, wat hoër toegang tot oortollige energie gee. Ek vind bewyse dat ekonomiese onsekerheid neig om 'n hoër assosiasie met LMI te hê onder hoërinkomste vroue, terwyl ekonomiese onsekerheid nie met hoër LMI onder mans geassosieer is.

Hierdie resultate dui daarop dat, gegee die aanhoudende ondervoeding in die vroeë jare en stygende lewenstandaarde, die voorkoms van vetsug in Suid-Afrika waarskynlik sal aanhou styg, veral vir dié van lae-SES kinderjare agtergrond, en dat dit onwaarskynlik is dat hierdie neigings in die nabye toekoms self sal opklaar. Dit vereis beleide om die verbruik van ongesonde voedsel te probeer verminder, en om voeding in die kinderjare te verbeter – veral in die vroegste jare van die lewe.

Sleutelwoorde: Voedingsoorgang; vetsugoorgang; kinderjare SES; sosiale mobiliteit; ouderdom-periode-kohort; ekonomiese onsekerheid.

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LIST OF ABBREVIATIONS

BMI	Body mass index
CSG	Child Support Grant
ECD	Early Childhood Development
FOP	Front-of-pack
GNP	Gross National Product
HPA	Hypothalamic-pituitary-adrenal
IEWB	Index of Economic Well-Being
ISHP	Integrated School Health Programme
LMIC	Low- and middle-income countries
NSNP	National School Nutrition Programme
NCD	Non-communicable disease
NIDS	National Income Dynamics Study
NIECD	National Integrated Early Childhood Development
SDGs	Sustainable Development Goals
SES	Socioeconomic status
SNAP	Supplemental Nutrition Assistance Program
US	United States
VIF	Variance inflation factor

CHAPTER 1

INTRODUCTION

1.1 Overview

Globally the burden of disease is shifting from communicable to non-communicable diseases (NCDs), a phenomenon known as the epidemiological transition. Hand in hand with this, countries are undergoing the 'nutrition transition' – a shift towards diets high in saturated fat, sugar and cheap processed energy-dense foods, known as the 'Western diet', and towards lower levels of physical activity (Popkin & Gordon-Larsen, 2004). With the nutrition transition has come a dramatic increase in worldwide rates of overweight and obesity in the last four decades. The change has not been limited to high-income countries, but has been seen in all regions of the world (NCD Risk Factor Collaboration, 2016). Many developing countries now experience the 'double burden of malnutrition', facing both undernutrition and overnutrition in the same populations. Obesity increases the risk of NCDs, including type 2 diabetes, high blood pressure, coronary heart disease, heart failure, and certain types of cancer (Franks & Atabaki-Pasdar, 2017; Goodarzi, 2018). NCDs account for 71 percent of global deaths, and 77 percent of NCD deaths occur in low- and middle-income countries (World Health Organization, 2021). The widespread and rapid increase in obesity rates thus poses a threat both to public health and to public health expenditure, and the importance of reducing obesity and NCDs has been recognised by both international bodies and the South African government. Target 3.4 of the Sustainable Development Goals (SDGs) is to reduce premature mortality from NCDs by one third by 2030 (United Nations Department of Economic and Social Affairs, n.d.). The South African government has recognised the importance of reducing obesity and NCDs through the adoption of a national strategic plan to curb NCDs (Department of Health, n.d.), and through the implementation of a health promotion levy on sugary beverages in 2018.

The burden of obesity tends to shift from the rich to the poor as countries develop, in a process that has been called the 'obesity transition' (Jaacks et al., 2019)[1]. As the rich tend to move through the nutrition transition ahead of the poor, at some point in countries' development the association between socioeconomic status (SES) and obesity, also known as the social or socioeconomic gradient in body mass index (BMI), shifts from positive to negative, usually occurring first among women

[1] The concept of the 'obesity transition' proposed by Jaacks et al. (2019) also includes the shifting distribution of obesity for men, women and children.

(Monteiro et al., 2004b). This reversal of the SES gradient in obesity tends to occur at a much lower gross national product (GNP) per capita than South Africa's (Monteiro et al., 2004b), and already appears to have begun – at least among women – in some other upper-middle income countries, such as Brazil, Argentina, Mexico and Egypt (Aitsi-Selmi et al., 2012; Basto-Abreu et al., 2018; Jiwani et al., 2019; Monteiro et al., 2004b). But it has not yet reversed in South Africa, except among white women (Wittenberg, 2013). The question remains as to why this reversal has not yet occurred in South Africa.

Aside from costs to individual health, obesity has considerable implications for our public healthcare system. Understanding the socioeconomic gradient in body weight is important for the targeting of policies to address obesity and promote public health, as well as for understanding the likely socioeconomic impact and incidence of such policies. As the obesity transition follows a predictable pattern (Jaacks et al., 2019), understanding the socioeconomic gradient in body weight also provides a glimpse of likely future obesity trajectories and future shifts in the socioeconomic distribution of obesity and associated NCDs, and provides insight into the nutrition transition as it occurs.

Most of the literature views the nutrition and obesity transitions as processes that happen across time and across stages of a country's development, examining the social gradient in body weight for the whole population at a single point in time, or assessing how it has changed over time. This dissertation explores life course, intergenerational and cross-generational (cohort) dimensions of the social gradient in body weight in South Africa, and how this relates to the nutrition and obesity transitions. As one of the most unequal countries in the world, South Africa provides a singular opportunity to study the nuances of the nutrition and obesity transitions. The South African population straddles several stages of the nutrition transition, a wider range than usually seen in developed countries. Furthermore, many people have experienced a rise in their living standards over the course of their lives as a result of South Africa's political transition from apartheid to a democratic government, offering an interesting context in which to explore life course and intergenerational aspects of the relationship between SES and body weight. Given that South Africa has not yet experienced a reversal of the social gradient in obesity, I explore whether younger generations are already undergoing changes that will eventually draw the whole population into this demographic shift. This study acknowledges that some parts of the population may match the trend of a country with the average level of development of South Africa, but others may not.

These heterogeneous trends are important to consider in a country with high levels of inequality. It also investigates the association between body weight and economic insecurity, a dimension of economic wellbeing that has been proposed as a driver of obesity in developed countries, and how its effects differ by SES. Studying these issues in a country that has not yet moved to the final stages of the obesity transition may help us to understand what drives the shift to later stages of the obesity transition, in which the distribution of obesity shifts to the poor and obesity rates level off and eventually may even decline.

Chapter 2 investigates life course and intergenerational aspects of the social gradient in BMI, exploring the association between childhood SES, social mobility and BMI in South Africa. This chapter finds that upward social mobility is associated with increased obesity risk in adulthood compared to individuals who maintained a stable high SES in childhood and adulthood. It shows that the social gradient in BMI is positive for individuals whose mothers had a low SES, but is flatter − and for women even shows signs of already having reversed − among individuals whose mothers had a high SES. This suggests that we need to see two generations of sustained high SES before we see a reversal of the socioeconomic gradient in body weight. This is the first study to explore intergenerational aspects of the obesity transition. These findings highlight the importance of studying transitions for heterogeneous parts of the population in a highly unequal society, and show that sustained upward intergenerational mobility (high SES sustained across more than one generation) is an important mediator of the shift to later stages of the obesity transition, where obesity rates plateau and may eventually start to decline. This provides insights into the slow-moving nature of the transition, and the need to find alternative interventions in unequal societies, where upward intergenerational mobility is slow.

Chapter 3 investigates generational aspects of the obesity transition, proposing that younger generations may be the first to see a reversal in the social gradient in BMI. The obesity transition may happen not only over time and levels of economic development, but also across successive generations. The obesity transition may not be one episode that affects an entire society, but new attitudes and circumstances as younger generations experience the benefits of upward intergenerational mobility may lead younger generations through the transition first. In a methodological first for this literature, I adopt a machine learning algorithm applied elsewhere to find structural breaks in the social gradient in BMI by birth year. This chapter shows that the social gradient in BMI is flatter among younger generations of South African men, but does not differ significantly across generations of women.

Chapter 4 explores the association between BMI and economic insecurity, another dimension of economic wellbeing. The stress caused by economic insecurity has been posited as a possible driver of the obesity epidemic in environments where energy-dense foods are readily available, and research in developed countries has provided support for this hypothesis. However, it is likely that economic insecurity only contributes to obesity when individuals have access to excess calories. The relationship between economic insecurity and obesity has not been explored in developing countries, where much of the population is likely to struggle to meet their nutritional requirements. This chapter asks whether economic insecurity is more strongly associated with body weight for those with higher levels of income, which yield increased access to excess energy. I find evidence that economic insecurity is associated with higher BMI only for higher income women. Economic insecurity is not associated with higher BMI for men.

The remainder of this introductory chapter reviews the literature on obesity and its relationship to SES, and gives an overview of the research questions addressed in this dissertation. It first provides the context for this dissertation, outlining the nutrition and obesity transitions and general theories on the causes of obesity in Section 1.2.1. It moves next to economic models and theories regarding obesity in Section 1.2.2. Section 1.2.3 details theories of how SES relates to body weight, and provides an overview of the empirical evidence on the relationship between several dimensions of SES and body weight. Section 1.2.4 reviews the literature on obesity and its relationship to SES in South Africa. Finally, Section 1.3 outlines the research questions addressed in the remaining chapters.

1.2 Literature Review

1.2.1 Background

1.2.1.1 The nutrition and obesity transitions

The shift from starchy, low variety traditional diets towards diets high in saturated fat, sugar and energy-dense processed foods has been dubbed the 'nutrition transition'. Popkin and Gordon-Larsen (2004) and Popkin (2006) describe five stages of this transition (see Figure 1.1. In the first stage, food is collected by hunting and gathering, and diets are varied. The population is lean and suffers from few nutritional deficiencies. The agricultural revolution of the second stage brings less varied cereal-based diets and the emergence of famine, nutritional deficiencies and declining stature. In the third stage famine recedes, but diets remain starchy and low variety, and nutritional deficiencies such as stunting continue to have a relatively high prevalence. In the fourth stage diets shift towards increased fats, sugar and processed foods, and the associated problems of obesity and degenerative disease emerge. The fifth stage sees a shift towards healthier diets and increased recreational physical activity, leading to a reduction in obesity and NCDs.

As more countries move through the nutrition transition, the burden of obesity is increasing among developing countries, and within countries is shifting increasingly to the poor. While entire countries may move through the stages of the nutrition transition, different stages of the nutrition transition can also coexist for different groups within countries. Historically, only rich societies and rich individuals within societies have been able to consume a high proportion of fat in their diets. However, the increasing availability of cheap vegetable oils has made higher fat consumption available even at low income levels (Drewnowski & Popkin, 1997). The relative prices of energy-dense processed foods in general have also fallen in recent decades (Cawley, 2015; Monsivais et al., 2010), making them increasingly available even to people with low incomes. As countries move through the nutrition transition, the socioeconomic and demographic patterning of obesity prevalence shifts, in what has been called the 'obesity transition' (Jaacks et al., 2019). The model outlined by Jaacks et al. (2019) is illustrated in Figure 1.2. As with the nutrition transition, the obesity transition has distinct phases, some of which overlap with the nutrition transition. In the first stage of the obesity transition, high-SES individuals, women, and adults have a higher prevalence of obesity. In stage 2, obesity prevalence increases, and the male-female gap in obesity prevalence narrows, as do the socioeconomic differentials in obesity prevalence among women. In stage 3 the socioeconomic gradient in obesity shifts from positive to negative, while obesity plateaus among children and high-SES women. The hypothesised fourth stage involves a decline in obesity prevalence, but there is little evidence of this yet having taken place in any country as a whole.

Although the authors do not make this connection, the nutrition and obesity transitions can be thought of as linked: those with higher SES move through the nutrition transition ahead of those with lower SES, and this is behind the reversal of the social gradient in BMI. At earlier stages of

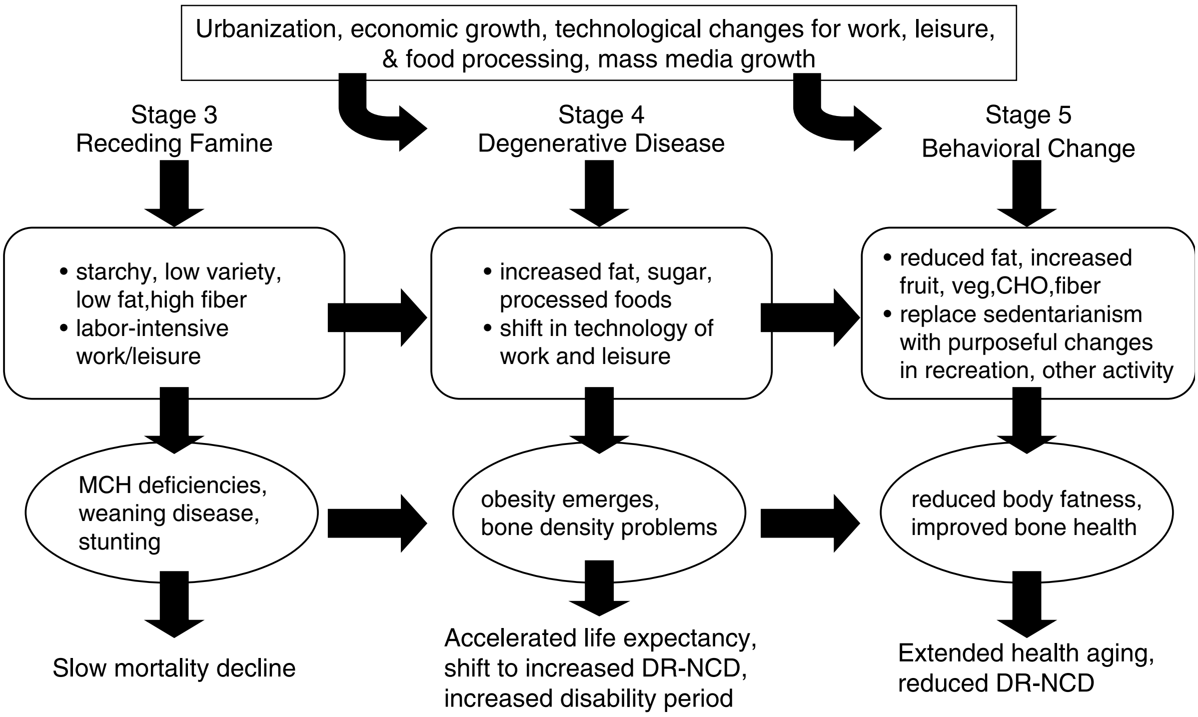


Figure 1.1: Outline of Stages 3 to 5 of the nutrition transition.

Source: Popkin and Gordon-Larsen (2004). Reprinted by permission from Springer Nature: Springer Nature International Journal of Obesity, 'The nutrition transition: worldwide obesity dynamics and their determinants', Popkin et al., Copyright 2004.

Note: DR-NCD = Diet-related non-communicable disease. MCH = maternal and child health.

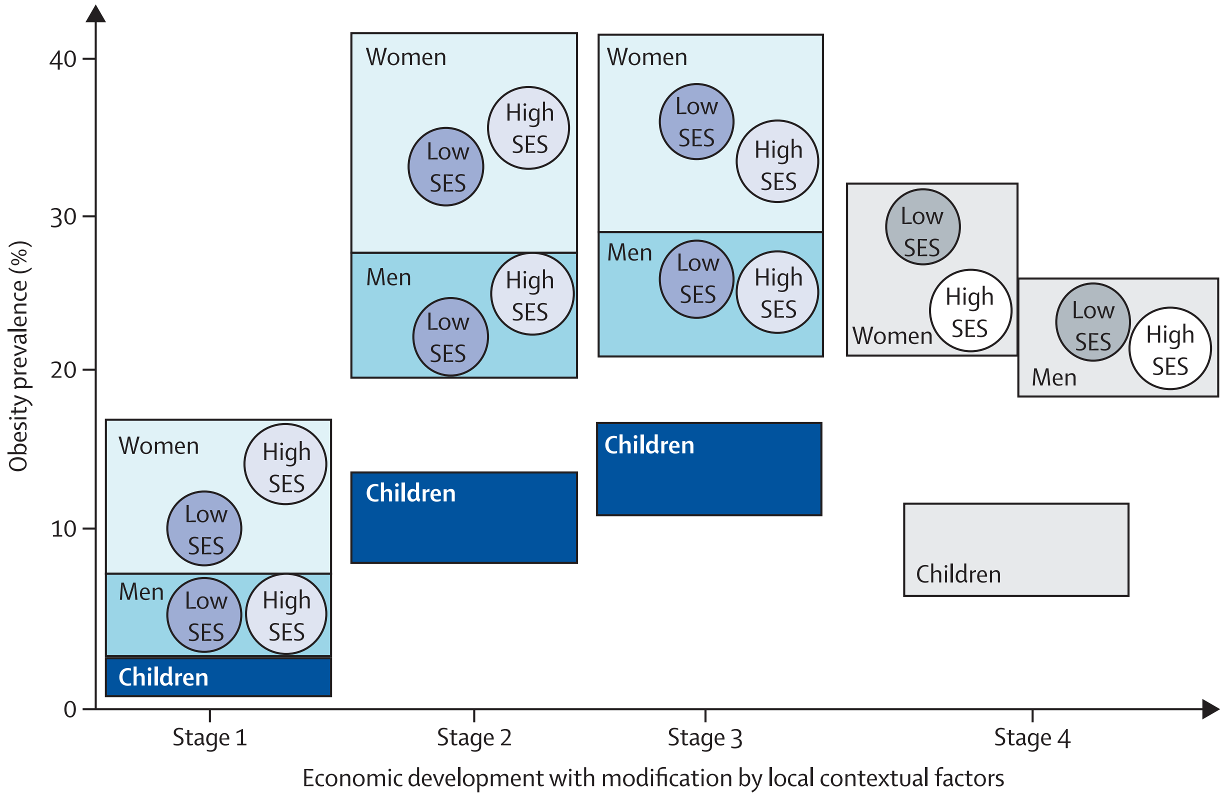


Figure 1.2: Illustration of the stages of the obesity transition according to Jaacks et al. (2019)
Source: Jaacks et al. (2019)

the obesity transition (stages 1 and 2), richer individuals are in stage 4 of the nutrition transition while poorer individuals remain in stage 3. This results in a positive association between SES and obesity. From stage 3 of the obesity transition, some richer individuals start to move to stage 5 of the nutrition transition, while poorer individuals move to stage 4. At later stages of the nutrition transition, higher incomes may enable people to afford healthier diets and increased leisure-time physical activity. This results in a negative association between SES and obesity.

The nutrition transition is closely linked to urbanisation. The globalisation of the food industry has contributed to the nutrition transition, and urban dwellers may be more exposed to the energy-dense processed foods offered by 'Big Food' companies, through greater penetration of fast food outlets, supermarkets and convenience stores in urban areas (Hawkes, 2006). Urbanisation is associated with higher consumption of fats, sugars, processed foods and foods prepared outside of the home, and a shift toward more sedentary jobs and physical activity patterns (Popkin, 1999). There is evidence from South Africa that proximity to supermarkets and fast food outlets leads to higher BMI and risk of obesity (Otterbach et al., 2021).

1.2.1.2 Obesity, BMI and its limitations

Body mass index (BMI) is commonly used as a measure of weight status. BMI is a person's weight in kilograms divided by the square of their height in metres. A BMI of 18.5 to 24.9 is considered normal weight, while a BMI of less than 18.5 is regarded as underweight, a BMI of 25 to 29.9 as overweight, and 30 or above as obese. The obese category is sometimes further broken down into obese class I, a BMI of 30 to 34.9, obese class II, a BMI of 35 to 39.9, and obese class III or severe or extreme obesity, a BMI of 40 or above.

The use of BMI and even the focus on body weight per se is not without criticism. BMI does not measure body fat directly, and does not take into account factors such as fat distribution, muscle mass or bone structure (Rothman, 2008). While higher BMIs are associated with increased disease risk, the BMI category cut-offs themselves are fairly arbitrary. Other measures like waist circumference and waist-to-hip ratio are also used as alternatives, as central obesity (i.e. the accumulation of fat around the abdomen) is more strongly associated with cardiovascular risk factors than is BMI (Lee et al., 2008). However, BMI and waist circumference are highly correlated, and for women BMI is more strongly correlated with body fat percentage than is waist circumference (Flegal et al., 2009). BMI data are also much more commonly available than alternative measures or data on conditions associated with high BMI. In spite of the limitations associated with BMI as a health measure, data on BMI and obesity can thus be a valuable source of information on health risk.

There is ongoing debate surrounding what is known as the 'obesity paradox'. Numerous studies have found that being overweight or even obese as opposed to normal weight has a protective effect on mortality among certain groups, such as those with cardiovascular disease and older people (Hamer, 2013). However, this could be due to some form of selection bias or omitted variable bias (Banack, 2014). For example, a pre-existing illness could lead to weight loss, and also to mortality. It is also possible to be obese and yet be metabolically healthy (suffering from few or no components of the metabolic syndrome, such as high blood pressure, blood glucose and cholesterol). Nonetheless, risk of cardiovascular disease and type 2 diabetes still appears to be higher for this group than in metabolically healthy individuals of normal weight (Stefan et al., 2018). Furthermore, metabolically healthy obesity may not remain so in the long term. Several studies have found that a substantial percentage of initially metabolically healthy obese people show symptoms of the metabolic syndrome over long-term follow-up (Phillips, 2017).

The association of obesity with cardiovascular disease, diabetes, and other metabolic conditions is well-established, but there has been some debate around whether obesity is the cause of these conditions, or whether the associations are the result of reverse causality or some confounding factor. However, recent evidence supports a probable causal role for obesity in type 2 diabetes, high blood pressure, coronary heart disease, heart failure, and certain types of cancer (Franks & Atabaki-Pasdar, 2017; Goodarzi, 2018).

1.2.1.3 Theories of obesity

A wide range of theories have been put forward in an attempt to explain the large increases in obesity rates in the last few decades, as well as cross-country variation in obesity prevalence. At the simplest level, obesity is seen as the result of an imbalance between energy consumed and energy expended. 'Obesogenic environments', or the proliferation of cheap energy-dense foods and declining physical activity levels associated with urbanisation and more sedentary jobs, are often identified as potential culprits. A further possibility is that obesity results from factors related to how the body processes and expends energy.

1.2.1.3.1 Genes and environments Several theories have been put forward regarding the genetic and physiological mechanisms influencing individual susceptibility to obesity. Studies have identified a wide range of genetic factors associated with individual susceptibility to obesity (Goodarzi, 2018). Body weight is highly heritable, with some estimates from twins studies of the variance in BMI explained by genes as high as 50 to 90 percent (Maes et al., 1997). However, genetic factors cannot explain the substantial increases in average BMI over the last few decades, and it is clear that environmental factors also play an important role in generating obesity. Genes interact with environmental triggers to produce obesity in susceptible individuals.

Several theories posit that the current high rates of obesity are due to some form of mismatch between our genes and modern environments. For most of our evolutionary history, our ancestors were hunter-gatherers who faced food scarcity and insecurity, and expended large amounts of energy to obtain food. According to the 'thrifty gene' hypothesis proposed by Neel (1962), metabolic features that evolved because they promoted survival under these conditions may predispose people to obesity in the environments of energy abundance and low energy expenditure that are now commonplace in developed countries, and increasingly in many developing countries. An alternative to this hypothesis was proposed by Speakman (2008), who argues instead that our risk of being preyed upon was lowered by the development of weapons, fire and social organisation, and that this removed any natural selection against genes promoting obesity (because obesity would make it harder to escape from predators), allowing genetic 'drift'. In modern energy-abundant environments, individuals with such a genetic predisposition to obesity become obese. What these two hypotheses have in common is the idea that genetic predispositions to obesity interact with modern environments where food is no longer scarce, to promote obesity. Both hypotheses centre on a mismatch between the environments in which we evolved and the environments currently faced by modern humans.

A further hypothesis by Hales and Barker (1992), the 'thrifty phenotype' hypothesis, proposes metabolic programming as a result of foetal and early life nutritional deprivation as a source of susceptibility to diabetes and associated metabolic conditions. If the metabolism is programmed to metabolic 'thrift' by early nutritional deprivation, but the individual's environment changes to one of abundant nutrition, diabetes and other metabolic dysfunctions may result. This hypothesis concerns diabetes, but may also apply to obesity, which is associated with diabetes and is often another symptom of metabolic disease.

1.2.1.3.2 Food insecurity A further theory regarding obesity argues that fat storage serves as insurance against starvation in times of scarcity. Food insecurity indicates that access to food is uncertain, and provides a cue for the body to store more fat, in some cases leading to obesity. A large body of evidence shows an association between perceived food insecurity and BMI among women in developed countries, lending some support to this hypothesis (Nettle et al., 2017). However, this evidence is correlational. Further support for this hypothesis has been drawn from experimental studies of birds showing that food insecurity (an unpredictable food supply, holding constant the total amount of food) is linked to weight gain (Nettle et al., 2017), though extrapolating evidence observed in birds to humans is contentious.

1.2.1.3.3 Stress Stress activates the hypothalamic-pituitary-adrenal (HPA) axis, which releases cortisol, as well as the sympathetic nervous system (governing the 'fight or flight' response). Activation of the HPA axis and sympathetic nervous system for short periods of time enables one to respond effectively to threats, and thus promotes survival. However, evidence from human and animal studies has linked chronic activation of these systems to fat storage, visceral obesity (fat located within the abdominal cavity, which bears a particularly strong association with metabolic disease) and other metabolic problems (Björntorp, 2001; Scott et al., 2012). The evidence also suggests that stress may promote cravings for and consumption of energy-dense 'comfort' foods (Dallman et al., 2005). There is some uncertainty surrounding the direction of causation between stress and metabolic disease, but evidence from longitudinal studies as well as experimental animal studies suggests that chronic stress causes obesity (Scott et al., 2012). Stressful life events, such as losing a family member or a financial shock, have also been associated with weight change, but there is some evidence that effects may differ depending on one's initial weight, with weight loss for those who initially had a healthy weight, and with weight gain for those who were initially overweight (Proper et al., 2013).

1.2.2 Economic theories of obesity

The above literature suggests that while genes are an important influence on individual susceptibility to obesity, environmental factors are also a crucial part of the puzzle. All this suggests that aspects of the socioeconomic environment are likely to play a role in obesity. As poverty and other socioeconomic factors may promote stress, chronic stress is one possible pathway linking the socioeconomic environment to metabolic disease. This section outlines economic models of obesity, as well as the literature on the possible links between the macroeconomic and individual socioeconomic environment and obesity.

1.2.2.1 Economic models of obesity

Traditional economic models view obesity as the outcome of a rational utility-maximising decision-making process. The traditional economic model of the demand for health was developed by Grossman (1972). In this model, individuals inherit a stock of health at birth that depreciates over time. The stock of health determines the amount of healthy time available in which an individual can produce earnings. The stock of health can be increased by investment, which takes the form of time and goods such as medical care, exercise and diet. The efficiency of the production of health can be increased by education. According to this model, the demand for health is likely to be correlated with wages, because the higher a person's wage rate, the higher will be the benefit of reducing time lost to illness. Both education and the wage rate shift the demand curve for health upwards. In this model, an individual's optimal level of BMI would be where the marginal costs of obesity (in terms of the health costs of obesity as well as the monetary costs of food consumption) are equal to the marginal benefits, such as the pleasure derived from consuming food (Cawley & Ruhm, 2011). The theory of rational addiction of Becker and Murphy (1988) also has some relevance to obesity, as overeating may be seen as a form of addiction for some people. According to this model, addiction may be utility-maximising for some individuals.

More recent literature has expanded upon or modified the traditional economic model to incorporate elements that may result in a level of BMI that is not optimal, while still retaining the assumption that an individual's BMI is the outcome of a rational utility-maximising process. First, individuals may be subject to information constraints: they may not fully understand the health risks associated with obesity, or how dietary choices and physical activity affect obesity risk. Second, individuals may have time-inconsistent preferences, displaying hyperbolic discounting and present-biased preferences. This may lead to problems with self-control and failure to follow through on intentions to engage in healthy behaviours (Cawley & Ruhm, 2011). Third, individuals may suffer from bounded rationality or cognitive limitations, in which they fail to maximise utility in the face of complex utility-maximisation problems. Individuals may not fully account for their time-inconsistent preferences, or other aspects of their utility-maximisation problem (Cawley & Ruhm, 2011).

Behavioural economic theory, which relaxes the assumption that individuals are rational utility maximisers, has also been applied to the study of obesity. The idea of an internal conflict between two 'selves' in the form of a farsighted 'planner', who values future utility and behaves in accordance with the rational decision-maker envisaged by traditional economic models, and a myopic 'doer', who is heavily biased towards immediate gratification (Thaler & Shefrin, 1981), can be applied to various risky health behaviours (Cawley & Ruhm, 2011), including those leading to obesity. The 'doer' makes decisions, but the 'planner' can constrain the 'doer' through willpower or precommitment devices. Ruhm (2012) argues that an interaction between the deliberative (corresponding to the traditional rational decision-maker) and affective systems of the brain can help to explain overeating, particularly in environments where foods engineered to stimulate the affective system of the brain are abundant.

Explanations for rising obesity rates in recent decades based on traditional economic theory have focused on falling food prices, or changes in relative food prices and availability, and sedentary technological change as drivers of increasing obesity (Cutler et al., 2003; Lakdawalla & Philipson, 2009; Philipson & Posner, 2003). Nutrient-dense foods tend to be more expensive per unit of energy than less nutritious energy-dense foods, and this disparity has grown (Monsivais et al., 2010). The real prices of energy-dense foods such as fast foods and sugar-sweetened beverages have fallen over the past few decades in the US, while the real prices of nutrient-dense foods such as fruit and vegetables have risen (Cawley, 2015). Some studies have examined the impact of the price or availability of fast food and groceries, but evidence that food prices or availability have an effect on BMI is mixed (Cawley, 2015).

Technological change has also been proposed as a potential explanation for rising obesity rates. According to Cutler et al. (2003), technological changes resulting in the centralised mass production of food led to falling time costs of consuming food, and thus greater consumption of (processed) food. Philipson and Posner (2003) argue that sedentary technological change has driven falling food prices and reduced job-related physical activity, resulting in a lower cost of consuming calories and a higher cost of expending them. Lakdawalla and Philipson (2009) provide another spin on this theory, suggesting that relative food prices have fallen due to increased supply driven by innovation in agricultural technologies. They argue that technological change and economic development have resulted in work becoming more sedentary, effectively raising the time cost of physical activity.

1.2.2.2 The macroeconomic environment

At the macroeconomic level, a number of studies have linked various aspects of the macroeconomic environment to obesity and to health more broadly. It has been hypothesised that one of the reasons for cross-country differences in obesity rates among rich countries may lie in economic regimes. Lawson et al. (2016) found that economic freedom (a measure of market liberalism) had a small positive association with BMI only among men in developing countries. Among rich countries, market liberalism has been found to be associated with higher average BMI and obesity (Egger, 2012; Offer et al., 2010). It has also been hypothesised that inequality could be linked to obesity, but the evidence is mixed. Some studies have shown a positive association between income inequality and obesity or BMI across rich countries (Egger, 2012; Pickett et al., 2005) and across US states (Volland, 2012). Offer et al. (2010), on the other hand, unexpectedly find weak positive effects of equality on obesity, while Zala (2013) finds no relationship between regional inequality and obesity in the UK. Adjaye-Gbewonyo et al. (2018) find no relationship between changes in district-level inequality and changes in BMI or any other cardiovascular disease risk factors in South Africa, though it is possible that inequality matters at the national level but not at the district or regional level. There is also some evidence that BMI fluctuates with the business cycle. Some studies have found that BMI is procyclical (Ruhm, 2000, 2005), though recent evidence has been more mixed (Bellés-Obrero & Castelló, 2018). There is also some evidence that leisure-time physical activity and healthy eating are countercyclical (Ruhm, 2000).

1.2.3 SES and obesity

In addition to the possible influence of macroeconomic factors, features of the socioeconomic environment at the level of individuals and households may also play a role in excess weight. An influential review by Sobal and Stunkard (1989) found a strong negative relationship between SES and obesity among women in developed countries, but no consistent relationship for men or children. In developing countries, on the other hand, a strong positive relationship was observed for men, women and children. This review was updated by McLaren (2007) for the period 1988 to

2004. This later review finds similar patterns to Sobal and Stunkard (1989), but the difference between women in high-income versus low-income countries in the relationship between SES and obesity was less pronounced.

The burden of obesity appears to shift from the rich to the poor as countries develop. A review focused on developing countries finds that obesity in developing countries is no longer only a disease of the rich, but that the burden of obesity tends to shift towards lower SES groups as countries develop (Monteiro et al., 2004b). The burden of obesity appears to shift towards lower SES women at an earlier stage of development than for men. For men, the relationship between SES and obesity is positive in half the studies reviewed, and not significant in the other half. For women, on the other hand, a majority of studies find an inverse relationship. The relationship is estimated to reverse for women at a GNP per capita of US$2500 (Monteiro et al., 2004a). While these studies use data from individual countries, this reversal of the relationship between SES and weight status is also supported by cross-country data (Pampel et al., 2012) and by a study using cross-country panel data attempting to control for the possible endogeneity of SES through dynamic panel methods (Windarti et al., 2019).

There is some evidence that results differ depending on the measure of SES used. Education is often used as a marker of SES in these studies, but its effect does not necessarily work in the same direction as income. To the extent that education raises income, it would be expected to have a similar effect to income. However, education could also increase nutritional knowledge or awareness of health risks associated with an unhealthy diet and lack of physical activity, and may thus have a protective effect on obesity risk. In Grossman's (1972) model of the demand for health, education raises efficiency in the production of health (i.e. enables one to produce more health with the same inputs). Grossman (2006) finds that education is more strongly correlated with health than income or occupation, and that the effect of education on health is not entirely accounted for by the impact of education on income or occupation. Monteiro et al. (2001) find that income is positively related to obesity in Brazil, while education may have a protective effect. Similarly, in Mexico Basto-Abreu et al. (2018) find that education is consistently inversely related to BMI among women, while neither an asset index nor income is significantly related to BMI. There is some evidence that wealth may be more weakly associated with obesity among more educated women. Aitsi-Selmi et al. (2012) find a significant interaction between education and wealth among Egyptian women: greater wealth is associated with higher odds of obesity for women with the lowest education level, but is not associated with obesity for women with the highest education level. Aitsi-Selmi et al. (2014) find that among middle-income countries, wealth is positively related to obesity for women with no or primary education, but negatively or insignificantly related to obesity for women with higher education. In low-income countries, on the other hand, wealth and education are both positively associated with obesity, with no significant interaction between them.

1.2.3.1 Theoretical reasons for a relationship between SES and obesity

Economic theory suggests a number of possible reasons for a relationship between SES and BMI or obesity. This section discusses the theoretical reasons for a relationship between various elements, measures or indicators of SES and obesity, while the next section gives an overview of the empirical evidence regarding several elements of SES (namely income, education and employment) separately.

The theoretical impact of income on weight is ambiguous. A higher income enables higher consumption of food, which would be expected to increase BMI. Alternatively, lower incomes may lead to substitution towards cheaper more energy-dense foods, which may also increase BMI. Food consumption would be expected to rise with income initially, but declining marginal utility of food implies that after a certain point food consumption levels off. Income also affects the affordability of health goods such as healthy foods and gym memberships, and if health, fitness and thinness are desired and can be considered normal goods, investments in these goods should increase as income rises (Cawley, 2015). This implies that the relationship between income and weight may show an inverted U-shape, at least in developed countries (Lakdawalla & Philipson, 2009). Philipson and Posner (2003) also predict an inverted U-shaped relationship between income and BMI, as weight has a non-monotonic relationship with utility, increasing utility at low levels of weight and decreasing it above a certain level.

In the traditional economic model of the demand for health (Grossman, 1972), both education and the wage rate shift the demand curve for health upwards. This presents one possible reason for an association between SES and obesity. More educated (higher SES) individuals are more efficient producers of health, resulting in a higher equilibrium level of health (and by extension lower levels of obesity) for more educated individuals. Higher earners have a greater incentive to invest in health, similarly resulting in a higher level of health for higher earners.

Philipson and Posner (2003) also predict an inverse U-shaped relationship between income and BMI across countries as a result of technological change. In low income countries, technological change raises incomes while at the same time reducing strain on the food supply and reducing the physical activity required to generate income. This implies a positive relationship between income and BMI. Part of this positive relationship between income and BMI may be due to the poor being more likely to have physically strenuous jobs. In technologically advanced countries, on the other hand, income may increase the demand for thinness (and health), leading eventually to weight reductions and a negative relationship between income and weight.

Obesity may also be seen as a signal of health or wealth or alternatively of illness or poverty depending on context (Philipson & Posner, 2003). This may be a reason for the social gradient in obesity, and for the reversal of this gradient as countries develop. Obesity may signal good health and wealth in poor contexts, where many cannot afford sufficient food and diseases causing thinness are widespread. There is evidence that this is the case in some South African contexts, where thinness or weight loss may be associated with HIV or TB (Draper et al., 2015; Matoti-Mvalo & Puoane, 2011). In richer contexts where food is plentiful, obesity may be a negative signal, and thinness a signal of self-control or self-discipline (Philipson & Posner, 2003). However, Philipson and Posner (2003) argue that body weight is a very imperfect signal, and that the signalling explanation of obesity implies falling rather than rising obesity rates over time, making signalling an incomplete explanation.

Neighbourhood or peer effects may also play a role in the relationship between SES and BMI. Independent of an individual's or household's own income, features of high or low income neighbourhoods (e.g. availability of supermarkets, exercise facilities, public transport or lack thereof) may promote or discourage obesity. Peers may also influence an individual's body weight preferences. Evidence for neighbourhood effects on obesity is provided by the Moving to Opportunity study, where individuals living in high-poverty housing projects who received a housing voucher by lottery to enable them to move to a lower poverty neighbourhood experienced a 5 percentage point reduction in obesity four to seven years later (Kling et al., 2007).

Another possible reason for an association between SES and obesity is time preferences. Those with more patient time preferences are likely to invest more in education (and therefore earn a higher income), and also to be more willing to bear the cost of healthier behaviours and forego food consumption in the present in exchange for future benefits in the form of lower risk of obesity. Alternatively, education may directly affect time preferences, resulting in greater investment in health behaviours (Fuchs, 1982).

It is possible that those with lower education[2] are subject to greater information constraints, which may be a barrier to engaging in healthy behaviours. Similarly, if education increases cognitive ability, and cognitive ability is related to health behaviours, this may explain some of the relationship between education and health behaviours, and in turn between education or SES more broadly and BMI. Indeed, Cutler and Lleras-Muney (2010) find that 30 percent of the association between education and health behaviours can be explained by knowledge and cognitive ability.

A further pathway that may link income to weight is the impact of financial concerns related to low income on decision-making processes. There is evidence that poverty may decrease cognitive 'bandwidth', or cognitive capacity and executive function (Mani et al., 2013; Schilbach et al., 2016). These capacities are involved in logical reasoning, decision-making and impulse control. According to these authors, cognitive bandwidth is not an inherent capacity, but is a finite resource that, when taxed with concerns about finances or other scarce resources, leaves less mental capacity for dealing with decisions in other areas of life (Schilbach et al., 2016). If cognitive bandwidth is consumed with financial concerns, this may reduce the ability to make healthy lifestyle decisions, thus increasing the risk of excess weight and metabolic disease. There is some experimental evidence that people who are more mentally taxed are more likely to choose less healthy food options. For example, Shiv and Fedorikhin (1999) find that subjects who are asked to memorise a seven-digit number are more likely to choose cake over fruit salad than subjects who are given a two-digit number to memorise.

The next section provides a short overview of the causal evidence on the impact of income and education on body weight.

1.2.3.2 Empirical evidence on the relationship between various measures of SES and body weight

1.2.3.2.1 Income Evidence from natural experiments suggests that income shocks tend to have a small or no significant impact on weight and on food expenditures in developed countries, but may have larger effects in developing countries (though the literature here is very small) and for

[2]Richer individuals tend to attain higher levels of education, and educational attainment is often used as a measure of SES.

those with very low income. However, there is evidence that calorie consumption fluctuates in the short-term in response to the monthly cycle of income from government transfers among low-income households in the US (for a review of this literature see Cawley, 2015). It is also possible that what matters for body weight is permanent income, particularly given the slow-changing nature of BMI and the cumulative nature of weight gain. Long-run income may thus have an effect on body weight, even if short-run income shocks have little effect.

1.2.3.2.2 Education Many studies show an association between education and BMI. Much of the literature linking SES and obesity relies on education as a measure of SES, particularly in low-income countries (McLaren, 2007; Sobal & Stunkard, 1989). However, the effect of education may not work in the same direction as the effect of other indicators of SES such as income. As well as influencing BMI through its effects on income, education may also have an independent effect on obesity.

There is a sizeable literature estimating the causal impact of schooling on income, wages or other measures of life success, but the literature on the causal effect of education on health and on BMI specifically is more limited. The relatively few studies that have attempted to identify the causal impact of education on BMI or obesity have largely relied either on within-twin fixed effects estimators or on changes in compulsory schooling laws or state-level differences in education policies as a source of exogenous variation in education level. Results have been mixed.

Using compulsory schooling reforms, Brunello et al. (2013) show that additional years of schooling decrease BMI for women in nine European countries, particularly for overweight women, but have no effect on males. Grabner (2009) finds large negative effects of schooling on BMI in the US, particularly for females. Using the raising of the school-leaving age in the UK, Clark and Royer (2013) find no significant effects on BMI, overweight or obesity, while Davies et al. (2018) find that additional schooling reduces BMI. The discrepancy between these two studies could be due to the fact that they were based on different datasets involving different age groups. Arendt (2005) finds insignificant effects using education reforms in Denmark.

Using sibling fixed effects models, Kim (2016) finds that additional schooling is associated with a lower BMI in Wisconsin, USA. In a study of Australian twins, Webbink et al. (2010) find that education reduces the probability of being overweight for men, but find no effect for women. Among female twins in the UK, Amin et al. (2013) find some evidence that education reduces BMI. However, also using twin fixed effects, Lundborg (2013) finds no significant effects on BMI.

Kenkel et al. (2006) use US state-level variation in education policies and education spending as instruments, and find no significant effect of high school completion on obesity. Using state-level variation in the timing of educational expansion in Germany, Jürges et al. (2011) do not find any consistently significant effects of schooling on overweight or obesity.

There is also some evidence from random assignment to early childhood preschool programmes in the US that these programmes affect obesity rates in childhood and adulthood for at-risk children, but the evidence is mixed. Those exposed to the Carolina Abecedarian Project in early childhood had lower rates of cardiovascular and metabolic disease in their mid-30s, particularly for males, though differences in overweight and obesity were not significant (Campbell et al., 2014). Results from the Perry Preschool Project suggest the programme had no significant effect on overweight, obesity, or other symptoms of the metabolic syndrome at age 40 (Muennig et al., 2009). For Head Start, programme eligibility reduced the probability of being overweight in adolescence for boys (Carneiro & Ginja, 2014).

1.2.3.3 SES in early life

Most of the research on the relationship between SES and obesity has focused on contemporaneous SES in adulthood. However, there is evidence that SES and other circumstances in utero and in early life also play an important role in the development of obesity and the metabolic syndrome. As outlined above, Hales and Barker (1992) suggest that nutritional deprivation in utero can programme the metabolism to deal with an environment of energy scarcity, causing the body to process energy in a way that increases susceptibility to the metabolic syndrome when exposed to a surplus of energy later in life. If low SES makes nutritional deprivation more likely, it could thus increase susceptibility to the metabolic syndrome. While the causal pathway may be physiological, it is also possible that there is a behavioural pathway from early deprivation to later eating habits.

There is causal evidence that early nutritional deprivation induced by nutritional shocks affects chances of obesity and the metabolic syndrome later in life. Evidence from the well-known Dutch Hunger Winter study shows that adults exposed to famine during early gestation are more likely to suffer from obesity and coronary heart disease (Roseboom et al., 2006). Almond and Mazumder

(2005) do not examine BMI directly, but find that exposure to the 1918 influenza pandemic in certain months of gestation led to higher incidence of associated metabolic conditions: diabetes, heart problems and hypertension. Beyond the foetal period, Hoynes et al. (2016) find that having access to the US Food Stamp Program, which effectively increases household resources, in early childhood significantly reduces the incidence of obesity and other symptoms of the metabolic syndrome in adulthood. Low birthweight and stunting in early childhood, an indicator of undernutrition, and subsequent catch-up growth and weight gain are also associated with higher BMI and with symptoms of the metabolic syndrome in adulthood (Adair et al., 2013; Victora et al., 2008). Early nutritional deprivation and low early life SES appear to have a stronger influence on women (Case & Menendez, 2009; Giskes et al., 2008).

Nutritional deprivation very early in life followed by a rise in living standards that makes surplus consumption of energy possible later in life may thus put people at increased risk of obesity and metabolic problems. This may be particularly problematic in developing countries experiencing urbanisation and a rapid rise in living standards, where children may have been more likely to experience nutritional deprivation in utero due to poverty early in life, but are subsequently exposed to increased access to energy-dense foods.

1.2.4 Obesity in South Africa

1.2.4.1 Obesity trends

The prevalence of obesity among South African women is among the highest in the world (NCD Risk Factor Collaboration, 2016). Furthermore, it appears that obesity rates continue to rise in SA. Reddy et al. (2012) found rapid increases among adolescents between 2002 and 2008, while Cois and Day (2015) found that BMI increased among adults between 2008 and 2012, particularly among women. SA is in stage 2 of the obesity transition (Jaacks et al., 2019). The question remains, is South Africa on the verge of shifting to stage 3? Are there any signs that the socioeconomic gradient in obesity is beginning to reverse?

1.2.4.2 The social gradient in body weight in South Africa

Among some other upper-middle income developing countries such as Brazil, Argentina, Mexico and Egypt, there are signs that among women the burden of obesity has already started to shift towards the poor (Aitsi-Selmi et al., 2012; Basto-Abreu et al., 2018; Jiwani et al., 2019; Monteiro et al., 2004b). One might expect that this pattern would already be present in South Africa too. However, a number of studies have shown a positive association between SES and BMI or obesity in SA among both women and men (Alaba & Chola, 2014; Ardington & Case, 2009; Ardington & Gasealahwe, 2012; Sartorius et al., 2015; Wittenberg, 2013). Indeed, Wittenberg (2013) argues that BMI is positively related to SES to the point that BMI can be considered a measure of economic wellbeing in SA. In most cases the relationship is stronger for men, though Case and Menendez (2009) find a relationship for women and no relationship for men. Alaba and Chola (2014), on the other hand, find a positive socioeconomic gradient in obesity for men, but find that the relationship among women is fairly flat (though still positive).

The evidence on the relationship between education and obesity in SA is mixed. There is some evidence suggesting an inverse U-shaped relationship between education and obesity among South African women. Sartorius et al. (2015) find that while women with primary or secondary education are significantly more likely to be obese than those with no schooling, tertiary-educated women are not significantly more likely to be obese (but it is worth noting that sample sizes of tertiary-educated individuals are low). This echoed the finding of Puoane et al. (2002) that women with post-school education are less likely to be obese than those with less schooling, and not significantly more likely than those with no schooling. Ardington and Case (2009), however, find no significant relationship between education and obesity amongst women, while Ardington and Gasealahwe (2012) find a positive relationship between years of education and female obesity.

None of the existing studies have attempted to identify the causal impact of any measures of SES on obesity in SA. The relationship between measures of SES and obesity is potentially bidirectional and may be confounded by reverse causation. There is international evidence (Kim & Von dem Knesebeck, 2018) and some suggestive South African evidence (Henry & Kollamparambil, 2017; Some et al., 2016) that obesity may lower wages and the probability of employment, possibly due to discrimination or due to lower productivity because of associated chronic diseases.

1.3 Outline of chapters

This dissertation explores several aspects of the relationship between SES and body weight in South Africa. The first two chapters explore whether South Africa is starting to experience shifts in the relationship between SES and body weight. Chapter 2 considers whether there is an intergenerational and a life course aspect to the social gradient in BMI, while Chapter 3 considers whether there are generational differences in the social gradient in BMI. Chapter 4 explores whether economic insecurity is associated with higher BMI, and whether this association is only present for those with sufficient income to enable consumption of excess calories. Chapter 5 concludes and discusses the policy implications of the findings.

1.3.1 Life course social mobility, childhood SES and adult obesity risk

Nutritional deprivation in childhood may programme the metabolism to deal with nutritional scarcity, and in combination with access to surplus energy enabled by rising incomes in adulthood may cause increased risk of obesity (Gluckman & Hanson, 2008). The international literature, mainly from developed countries, has found that low childhood SES may increase the risk of obesity in adulthood (González et al., 2009; Parsons et al., 1999; Senese et al., 2009). Additionally, upward social mobility from a low childhood SES background to a high adult SES is associated with increased obesity risk compared to a stable high SES in childhood and adulthood, particularly for women (Vieira et al., 2019).

The relationship between childhood SES, social mobility and obesity risk has not been adequately investigated in the developing country context, and particularly in the South African context. These relationships may differ from those in developed countries in a developing country experiencing the 'double burden of malnutrition'. It is particularly interesting to consider the effects of social mobility in the context of a country in the midst of the nutrition transition, where urbanisation, improving living standards and changing diets may result in individuals experiencing very different socioeconomic and nutritional environments in adulthood from those they experienced in childhood – in other words, a potentially substantial 'mismatch' between childhood and adult environments. Furthermore, almost all studies investigating the association between childhood SES and BMI have been conducted in countries where adult SES is negatively associated with obesity, at least for women, and so the finding of a negative association between childhood SES and adult obesity echoes that for adult SES. It is especially interesting to explore whether higher childhood SES is also associated with lower adult obesity in a context where higher adult SES is associated with higher adult obesity. Furthermore, exploring the relationships between childhood SES, social mobility and body weight in a country that remains in stage 2 of the obesity transition may help us to understand what drives the shift to later stages of the obesity transition, where the distribution of obesity shifts to the poor and obesity rates level off and eventually may even decline. Two South African studies have looked at issues related to childhood SES and social mobility (Case & Menendez, 2009; Ginsburg et al., 2013), but they are based on localised urban samples that do not represent the entire country, and in the second case examines BMI in adolescence rather than adulthood.

Chapter 2 explores the interplay between childhood SES, social mobility and the nutrition transition in South Africa using the nationally representative National Income Dynamics Study (NIDS) data. In this paper I explore the hypothesis that the nutrition transition may pose a particular risk for people from poorer childhood backgrounds, and particularly for those who experience upward social mobility between childhood and adulthood. Using mother's education as a proxy for childhood SES, I explore whether upward social mobility from a low childhood SES to a high adult SES is associated with higher adult BMI compared to those who had a high SES in both childhood and adulthood. I investigate whether the social gradient in body weight is closer to reversing for individuals from a high childhood SES background, hypothesising that the gradient may reverse first for individuals who experience more than one generation of sustained high SES. I use random effects within-between models to explore whether the social gradient in body weight is driven by changes in income over time within individuals, representing short-run changes in socioeconomic circumstances in adulthood, or differences in average incomes between individuals, representing longer-run factors related to SES.

1.3.2 Generational change in the association between SES and BMI

As countries develop and move through stages of the nutrition and obesity transitions, the SES gradient in obesity tends to reverse. The nutrition and obesity transitions have been seen as a process that takes place over time and over stages of a country's development. I propose that the nutrition and obesity transitions may not only occur over time or across levels of economic development, but also across generations. The reversal of the gradient may already be taking place among younger generations, even if it is not observed for the whole population in aggregate. A few international studies have found that SES gradients in BMI are flatter among younger people (Baum & Ruhm, 2009; Clarke et al., 2009; Yang et al., 2021), but the international literature has not considered the possibility that these differences may be driven by cohort rather than life cycle effects. The SES gradient may become steeper over the life course as individuals age (an age effect); alternatively, individuals from younger cohorts may experience a more permanent flattening of the SES gradient in BMI, so that the SES gradient remains flatter than for previous generations even as these individuals age (a cohort effect). Younger cohorts are likely to have been exposed to different nutritional environments in childhood or at other sensitive periods in the life course, which may have lasting effects on the social gradient in BMI. Alternatively, younger generations may be more likely than their elders to adopt a thinner ideal body size, which may result in a flatter SES gradient for younger cohorts. Thus the stages of the nutrition and obesity transitions may coexist within a country: individuals from a younger generation may be in stage 3 or even stage 4 of the obesity transition, even while individuals from older generations, possibly even within the same household, remain in stage 2.

In Chapter 3, I explore whether there is a generational aspect to the obesity transition in South Africa using the five waves of NIDS data. I use a machine learning algorithm to investigate whether the positive relationship between income and BMI is weakening for younger generations of South Africans. Uniquely, I use the model-based recursive partitioning algorithm (Zeileis et al., 2008) to find structural breaks in the SES gradient in BMI by birth cohort. The algorithm allows us to detect differences in the SES-BMI association across generations. I then test whether the SES-BMI relationship differs for younger and older cohorts by interacting SES with the birth year splits identified by the algorithm in linear regressions. Using panel data allows me to observe the same cohort at different ages, providing a starting point to understand differences between life cycle and generational effects.

1.3.3 Economic insecurity and obesity risk

Economic insecurity, or “the anxiety produced by a lack of economic safety” (Osberg, 1998), has been proposed as a possible driver of obesity (Smith et al., 2009; Wisman & Capehart, 2010). As described in Section 1.2.1.3.3, stress may promote consumption of energy-dense ‘comfort’ foods, and chronic stress has been linked to obesity. The stress created by economic insecurity may thus promote weight gain and obesity in environments where excess energy is readily available. A number of studies in developed countries have found support for this. For example, in the well-known Whitehall II study, the threat of job loss increased BMI among British civil servants in a department facing privatisation (Ferrie et al., 1998). Offer et al. (2010) find that across developed countries, economic insecurity is associated with higher obesity rates, and is a stronger predictor of obesity rates than the increased accessibility and fall in relative prices of fast foods and energy-dense processed foods.

Economic insecurity may only increase obesity when excess calories are available and accessible. The relationship between economic insecurity and obesity has only been explored in developed countries, where most people are able to access energy-dense processed foods and easily obtain excess calories – developed countries are at stage 4 or 5 of the nutrition transition outlined in Section 1.2.1.1. In many developing countries the well-off may already be in stage 5, but much of the population remains in stage 3 or early in stage 4, without access to food surplus to their nutritional requirements.

Chapter 4 explores the association between economic insecurity and BMI in South Africa, proposing that we are only likely to see an association between economic insecurity and increased obesity risk for those with sufficient income to obtain excess calories. As the concept of economic insecurity involves subjective anxiety due to potential economic losses (Bossert & D'Ambrosio, 2013; Osberg, 1998), even the relatively well-off may experience economic insecurity, with potential consequences for their health. I explore whether economic insecurity is more positively associated with body weight for those with higher household incomes by interacting our economic insecurity measures with household income.

1.4 Summary

This thesis explores life course, intergenerational and cohort aspects of the nutrition and obesity transitions using South Africa's nationally representative National Income Dynamics Study data. The existing literature has viewed the nutrition and obesity transitions as processes that occur over time and the course of economic development. This thesis adds to the literature by proposing that the nutrition and obesity transitions may have life course, intergenerational and cohort dimensions. Chapter 2 explores intergenerational aspects of the obesity transition, asking whether the social gradient in BMI differs for those from relatively advantaged childhood backgrounds compared to those from disadvantaged backgrounds, whether social mobility between childhood and adulthood poses an increased obesity risk, and whether the SES-BMI association is driven more by long-run SES or short-run changes in income. Given that South Africa has not yet moved into stage 3 of the obesity transition, marked by a reversal of the social gradient in body weight, in Chapter 3 I explore whether younger generations are already experiencing changes in the social gradient in BMI that will eventually be reflected in the whole population. Chapter 4 explores heterogeneities in the association between economic insecurity and BMI in South Africa by SES, acknowledging that the high levels of inequality in South Africa mean that different income groups are at different stages of the nutrition transition. Parts of the population at later stages of the nutrition and obesity transitions may reflect trends seen in developed countries, which are already at later stages of these transitions, while other parts may not.

CHAPTER 2

CHILDHOOD SOCIOECONOMIC STATUS, SOCIAL MOBILITY AND THE OBESITY TRANSITION IN SOUTH AFRICA

2.1 Introduction

The globalisation of the food industry has contributed to the nutrition transition throughout the world, and these forces may have different effects for the rich and the poor, and residents of urban and rural areas (Hawkes, 2006). The rich and urban residents may be more exposed to the energy-dense processed foods offered by 'Big Food' companies, through greater penetration of fast food outlets, supermarkets and convenience stores in urban areas and their greater ability to afford these foods. However, the burden of obesity tends to shift from the rich to the poor as countries develop, in a process that has been described as the 'obesity transition' (Jaacks et al., 2019). At some point in countries' development, the relationship between socioeconomic status (SES) and obesity (known as the social gradient) shifts from positive (stages 1 and 2 of the obesity transition) to negative (stage 3 of the obesity transition), usually at an earlier stage of economic development for women (Jaacks et al., 2019; Monteiro et al., 2004b).

However, little is known about what drives the shift from one stage of the obesity transition to the next. These changes tend to happen with economic development (Jaacks et al., 2019), but little is known about the specific mechanisms that drive the change. The obesity transition is usually viewed through the lens of SES in adulthood, and the literature has not considered how SES across generations and at various points in the life course may interact to drive the reversal of the social gradient in body weight. A fairly large literature suggests that higher SES in childhood may have lasting protective effects against adult obesity (González et al., 2009; Parsons et al., 1999; Senese et al., 2009). Studies have also found that, particularly for women, upward social mobility from a low childhood SES confers a greater risk of obesity compared to maintaining a stable high SES throughout life (Vieira et al., 2019). It has been proposed that a mismatch between early life and adulthood environments may put individuals at greater risk of obesity (Gluckman & Hanson, 2008).

This paper explores the possibility that childhood SES and intergenerational mobility may contribute to the shift from stage 2 to stage 3 of the obesity transition – the reversal of the social gradient in body weight and a corresponding plateau in obesity rates. We use the South African nationally representative National Income Dynamics Study (NIDS) panel data to explore the association between childhood SES, upward social mobility and adult body mass index (BMI), and how these may interact to drive the shift from one stage of the obesity transition to the next. We investigate whether the relationship between adult SES and BMI differs by levels of childhood SES. Uniquely, we also exploit the panel dimension of the NIDS data to explore whether the social gradient in BMI is driven by differences in average income between individuals or by changes in income within individuals' adult lives using random effects within-between models. We also explore whether these relationships differ between urban and rural areas, as the nutrition and obesity transitions are likely to be more advanced in urban areas and residents of urban areas are thus likely to have greater exposure to energy-dense processed foods.

South Africa makes an intriguing case study precisely because it is still in the second stage of the obesity transition: the relationship between adult SES and BMI is generally still positive. The reversal of the association between SES and obesity among women has been estimated to occur at a gross national product (GNP) per capita of around US$2500 (Monteiro et al., 2004b), and there are signs that the SES-BMI relationship has already started to reverse among women in some other upper middle-income countries, such as Brazil and Mexico. South Africa is already well past this point, but the social gradient has not yet reversed, except among white women (Wittenberg, 2013). The question remains why the social gradient in body weight has not yet reversed in South Africa. However, studies on the relationship between SES and body weight in South Africa have focused almost exclusively on SES measured in adulthood. Most studies examining the relationship between childhood SES, social mobility and body weight are from high-income countries in the later stages of the nutrition and obesity transitions, where the social gradient in body weight has already reversed, at least for women. In these contexts higher childhood SES and adult SES are both associated with lower BMI. It is particularly interesting to consider whether childhood SES still has a protective effect[1] against obesity in a context where higher adult SES increases obesity risk. South Africa has also undergone rapid social change and urbanisation in recent decades, so in this context there is the potential for a large mismatch between individuals' early life and adult nutritional environments.

[1] The word 'effect' and other similar words are used loosely here, and are not meant to imply a causal effect. Our data do not allow us to estimate a causal effect of childhood SES on adult obesity risk.

Only a handful of studies have addressed the association between childhood SES, social mobility and adult obesity risk in developing countries at earlier stages of the nutrition and obesity transitions. Doing so allows us to explore whether these factors are possible drivers of the obesity transition.

We find that the social gradient in BMI is flatter among individuals from a high SES childhood background, and in particular already shows signs of having reversed among women with a high childhood SES who also have a high SES in adulthood. These findings shed light on the future of the obesity transition in South Africa; they suggest that it may take more than one generation of sustained high SES – or alternatively perhaps of adequate childhood nutrition – before we see higher adult SES have a protective effect against obesity, and with it a plateau in obesity rates. It also suggests that obesity rates are likely to continue to rise in South Africa, particularly among individuals from low-SES childhood backgrounds.

Section 2.2 outlines the literature on the nutrition transition and the association between SES across the life course and obesity risk. Section 2.3.2 describes the NIDS data and our methodology. Section 2.4 presents our results, Section 2.5 discusses our results and Section 2.6 concludes.

2.2 SES across the life course and obesity risk

2.2.1 The nutrition transition

South Africa is undergoing the nutrition transition: a shift from traditional diets high in starch and fibre to diets higher in fat, sugar and processed foods (Popkin & Gordon-Larsen, 2004). The nutritional transition is accompanied by an epidemiological transition: a shift from a high burden of infectious diseases towards a high prevalence of non-communicable diseases. As a result of these ongoing transitions, South Africa experiences a double burden of malnutrition: a high burden of undernutrition as well as a high burden of overweight and obesity, often within the same household and even within the same individual. This is illustrated by South Africa's persistently high rate of childhood stunting of 27 percent, coupled with high rates of overweight and obesity: 68 percent of women and 31 percent of men are overweight or obese (Department of Health, 2019). Mean BMI among women in South Africa is among the highest in the world (NCD Risk Factor Collaboration, 2016).

The nutrition transition is closely linked to urbanisation. Hawkes (2006) describes how the globalisation of the food industry has contributed to the nutrition transition throughout the world, and how these forces may have different effects for the rich and the poor, and urban and rural dwellers. Urban dwellers may be more exposed to the energy-dense processed foods offered by big food companies, through greater penetration of fast food outlets, supermarkets and convenience stores in urban areas. Urbanisation is associated with higher consumption of fats, sugars, processed foods and foods prepared outside of the home, as well as a shift toward more sedentary jobs and physical activity patterns (Popkin, 1999). Popkin (1999) documents a greater share of sugar consumption in total calories in more urbanised low-income countries than in less urbanised low-income countries. In South Africa, Otterbach et al. (2021) find that proximity to supermarkets and fast food outlets leads to higher BMI and risk of obesity. Steyn (2006) reports that consumption of sugar, fats, animal products, vegetable oils and alcohol is higher among urban South Africans, while consumption of vegetables, legumes and cereals is higher among rural dwellers. Furthermore, rural-urban migrants decrease physical activity, shift towards a less healthy diet, increase alcohol use, and are more likely to start smoking the longer they spend in urban areas (Steyn, 2006). Between 1998 and 2008 in South Africa, obesity grew faster among urban women than among rural women: by 2.5 percentage points among rural women, and nearly 10 percentage points among urban women. Increases in obesity were also greater for urban compared to rural men (Frayne et al., 2014)[2].

2.2.2 Models of life course SES and obesity risk

Several models have been proposed relating SES across the life course to obesity, as well as other risk factors for cardiovascular disease (Ben-Shlomo & Kuh, 2002; Pollitt et al., 2005). First, the 'critical periods' model proposes that childhood or in utero SES may have direct effects on adult health, independent of any effects of childhood SES on later SES or lifestyle behaviours (Pollitt et al., 2005). According to this model, there are critical and sensitive periods during development when the body is more sensitive to certain risk exposures (as well as to positive inputs). Exposures may only

[2]However, it is worth noting that obesity is growing rapidly in rural areas worldwide. The NCD Risk Factor Collaboration (2019) found that over 55 percent of the average rise in BMI globally between 1985 and 2017 was driven by increases in rural areas. The figure was even higher in some developing regions. However, Sub-Saharan African women were an exception to this pattern: here mean BMI increased faster among urban women, and in 2017 women in SSA had the largest urban-rural BMI gap in the world (NCD Risk Factor Collaboration, 2019). In many developed countries mean BMI is slightly higher in rural areas, which may be due in part to lower education and incomes in rural areas in developed countries (NCD Risk Factor Collaboration, 2019). This suggests that the higher obesity rates seen in urban areas of developing countries may be a temporary phenomenon, and that as with SES, the burden of obesity may shift towards rural areas as countries develop. It seems that the nutrition transition begins in urban areas, but eventually penetrates rural areas too as rural market penetration of national food retailers increases and national food distribution systems expand (Popkin, 1999).

have a long-term effect if they are experienced during a critical period for that exposure, or have a stronger effect if they are experienced during a sensitive period. A well-known form of this idea is the 'foetal origins' hypothesis (Hales & Barker, 1992), also known as 'metabolic programming'; this is the idea that nutritional deprivation in early life – particularly in utero – may programme the metabolism to cope with an environment of nutritional scarcity. Empirically, this model predicts an association between early SES and adult obesity that remains when controlling for current SES and health behaviours.

Second, the 'accumulation' model proposes that low SES has cumulative effects: the health risks associated with low SES accumulate with the number of adverse risk exposures, or the length of time exposed to these risks (Ben-Shlomo & Kuh, 2002; Pollitt et al., 2005). This model predicts increasing adverse health effects with a greater number of periods exposed to low SES or adverse conditions. Assuming an inverse relationship between SES and health, individuals exposed to low SES in more than one period would have worse health than those exposed to low SES in one period only. Conversely, in South Africa and other developing countries with a positive social gradient in BMI as well as other cardiovascular disease risk factors, we would expect to see increasing BMI with the number of periods exposed to high SES.

Finally, the 'social mobility' model proposes that social mobility across the life course poses unique health risks. There are various forms of this model; one form proposes that the upwardly mobile will be at reduced risk of ill health compared to those who retain a low SES, while another proposes that the upwardly mobile will be at increased risk of ill health compared to those who retained a high SES throughout their lives. We focus on the latter. The idea that a mismatch between individual SES in early and later life may increase obesity risk is a form of the 'mismatch' hypothesis proposed by Gluckman and Hanson (2008), which argues that a mismatch between the environment experienced in early life and that faced in adulthood may increase an individual's risk of obesity (the mismatch hypothesis extends the 'foetal origins' hypothesis of Hales and Barker (1992)). Poor nutrition or other adverse exposures in early life may signal to the developing child that it will face an adverse environment in the future, leading to changes in the developmental trajectory through epigenetic processes to adapt to the anticipated future environment. If the individual is instead faced with an obesogenic environment of high energy availability later in life, this may increase his or her risk of obesity and other metabolic problems. While a mismatch between nutritional environments in early life and adulthood may result from social mobility, it is also possible that such a mismatch occurs due to rising living standards or changing food environments across society. Social mobility implies a change in the relative position of individuals within a society, but in countries undergoing a rise in average living standards or the nutritional transition an individual may experience an increase in the availability of energy-dense processed foods between childhood and adulthood without any increase in their relative social position. This mismatch has been proposed to explain rapidly increasing obesity rates in low- and middle-income countries undergoing the nutritional transition (Gluckman & Hanson, 2008).

2.2.3 Empirical evidence for life course SES models

These models are not necessarily mutually exclusive; it is possible for all three to be acting simultaneously in some contexts (see e.g. Chung et al., 2020; Heraclides & Brunner, 2010), and they can be difficult to disentangle empirically. The most compelling evidence for the critical periods model comes from famine studies. For example, the Dutch Hunger Winter studies show that people who were exposed to famine during early gestation (Lumey et al., 2021; Roseboom et al., 2006) or during the postnatal period (Van Abeelen et al., 2012) were more likely to be overweight or obese in adulthood. However, there is some conflicting evidence suggesting that these effects may depend on context: in Vietnam, exposure to famine reduced BMI (Guven et al., 2021). Vietnam is at an earlier stage of the nutrition and obesity transitions than the Netherlands, suggesting that the effects of nutritional deprivation in childhood may depend on the phase of these transitions a country is in. There is also ample evidence that less extreme negative childhood exposures, such as those associated with low SES, may increase obesity risk. A large number of studies have documented an independent association between low childhood SES and increased risk of obesity in adulthood for women, but no consistent relationship for men (see reviews by González et al., 2009; Parsons et al., 1999; Senese et al., 2009)[3]. Studies have also found significant associations between childhood SES and related outcomes, including adult body composition (Bann et al., 2014; Bridger Staatz et al., 2021), diabetes (McEniry, 2013; Stringhini et al., 2013), heart disease (McEniry, 2013), and chronic inflammation (Stringhini et al., 2013).

[3] Many of these studies have failed to account for possible confounding by associations between parental and offspring BMI. However, a study in Sweden by Lowry et al. (2020) found that the association between low childhood SES and adult BMI was attenuated in some cohorts when controlling for genetic predisposition to obesity and maternal BMI, but remained significant in others.

Multiple studies have found evidence for cumulative effects of low SES across the life course. Echoing the findings for adult SES, multiple studies, mostly from developed countries, have found that stable low life course SES is associated with higher BMI and obesity risk than stable high life course SES (Newton et al., 2017; Vieira et al., 2019)[4]. In a meta-analysis, Vieira et al. (2019) find evidence for the risk accumulation model for women – accumulation of SES disadvantage through the life course was associated with higher BMI for women – but find no significant association for men. Heraclides and Brunner (2010) and Chung et al. (2020) also find higher BMI among those who spent a higher number of periods with low SES.

Findings on social mobility have been inconsistent, stemming in part from differing specifications used across studies, but there are indications that the upwardly mobile are at increased risk of obesity compared to the stably high SES. Most studies split respondents into two SES groups – 'low' and 'high' childhood SES and adult SES groups – and compare the upwardly mobile (low-high) either with those who maintained a stable low SES in childhood and adulthood (low-low) or those who maintained a stable high SES (high-high). Several studies have found that upward mobility is associated with lower obesity compared to those who remain low SES (Salmela et al., 2020); though Heraclides and Brunner (2010) find no such significant association. A problem with this strategy is that many of these studies do not control for SES in adulthood, and if adult SES is associated with adult BMI, then these studies may conflate social mobility with adult SES: those who moved from a low childhood SES to a high adult SES will by definition have a higher adult SES than those who remained in the low SES group in both childhood and adulthood[5]. The lower BMI observed in the upwardly mobile compared to the stably low SES is consistent with the inverse relationship between adult SES and BMI observed for women in developed countries. Studies using the latter specification have generally found an increased obesity risk for the upwardly mobile compared to those who maintained a high SES in both childhood and adulthood (Chung et al., 2020; Heraclides & Brunner, 2010; Savitsky et al., 2017; Savitsky et al., 2021), with some exceptions (Salmela et al., 2020). The association appears to be more consistent for women; in a meta-analysis, Vieira et al. (2019) find that upward mobility among women was associated with a higher BMI compared to those who maintained a high SES throughout life, but find no significant association for men.

[4]Estimation of the cumulative effects of SES is complicated by the inclusion of current SES, which would be expected to have direct effects on health

[5]Conversely, studies comparing upward mobility to stable high SES may conflate the persistent effects of low childhood SES with those of upward mobility. This literature faces an unacknowledged multicollinearity problem: it is not possible to control for childhood SES, adult SES and social mobility simultaneously.

Few studies have explicitly explored whether a mismatch between early life and adult environments more broadly is associated with obesity, though the same mechanisms may be behind associations between upward social mobility and obesity. Savitsky et al. (2021) investigate whether a mismatch between childhood and adult environments increases the risk of obesity in Israel, but the measures of early and adult environments they use mainly consist of SES measures, so this is very similar to studies investigating social mobility. Schooling et al. (2007) compare Hong Kong Chinese adults who grew up in a then relatively undeveloped region of China to those who grew up in the more economically developed Hong Kong. Schooling et al. (2007) find that, among women living in Hong Kong as adults, those who grew up in the more economically developed Hong Kong had a lower waist-hip ratio than those who grew up in a then relatively undeveloped region of China: in other words, those who experienced no mismatch between their childhood and adulthood environments had a lower waist-hip ratio than those who did experience a mismatch. The opposite was true for men.

2.2.4 Evidence from developing countries

Most studies of the relationship between childhood SES, social mobility and BMI have been conducted in developed countries. These relationships may be different in developing countries in the midst of the nutrition transition. The relationship between childhood SES, social mobility and adult obesity may depend on what stage of the nutrition and obesity transitions countries are in, and particularly the level of access to energy-dense processed foods. For example, it is possible that childhood SES may only be protective against adult obesity in the later stages of the nutrition transition, in contexts where energy-dense processed foods are readily available and accessible. It is also possible that childhood SES has no special relationship with adult obesity, and that the relationship between childhood SES and adult body weight merely mirrors that between adult SES and body weight, reflecting the stage of the obesity transition a country is currently in. If childhood SES is associated with BMI in the same direction as adult SES, it would suggest cumulative effects of SES over the life course. Alternatively, if childhood SES is associated with BMI in the opposite direction to adult SES, it would add to the evidence suggesting that childhood is a sensitive period for the development of adult obesity risk, and that the associations between adult and childhood SES and adult BMI are driven by different mechanisms. We are only aware of a handful of studies from developing countries. In Brazil, higher childhood SES is associated with lower obesity among women, but higher obesity among men (Aitsi-Selmi et al., 2013; Wagner et al., 2018). Similarly, in China childhood SES is associated with higher odds of metabolic syndrome for women but not for men, while higher childhood SES is marginally associated with lower waist circumference for women but a significantly higher waist circumference for men (Schooling et al., 2008). Among urban civil servants in Ghana, both adult and pre-adult wealth are positively associated with obesity for men, but no significant association is found for either adult or pre-adult wealth for women (Addo et al., 2009). In Brazil, Barros et al. (2006) find no association between social mobility and the probability of being overweight at 19. There is clearly a need for more research on the association between childhood SES, social mobility and obesity in developing countries. Studying the relationships between childhood SES, social mobility and adult obesity in countries in earlier stages of the nutrition and obesity transitions may help us to understand whether these factors contribute to the shift to later stages of the obesity transition.

2.2.5 Evidence from South Africa

A handful of South African studies have considered questions related to those addressed in the present study, but all of these have been based on samples of Africans living in specific urban neighbourhoods, were not nationally representative, and in only one case investigated outcomes in adulthood. Given that metabolic health risks peak in adulthood, it is important to study these outcomes among adults. Several studies have examined outcomes in adolescence using the Birth to Twenty cohort of children born in Soweto, Johannesburg. Pradeilles et al. (2015) find that lower caregiver education (secondary compared to higher education)[6] is associated with a lower probability of being overweight for male adolescents, but find no association for females. Ginsburg et al. (2013) find no association between SES at birth and BMI at 15. They find that, compared to those who did not change SES, an increase in SES between birth and 15 years of age was associated with higher BMI among female adolescents, but not among males. In a study of blood pressure (another indicator of metabolic health) also using the Birth to Twenty sample, Kagura et al. (2016) find that those who experienced upward social mobility between infancy and adolescence had lower systolic blood pressure at 18 than those who maintained a stable low SES. However, these studies control for SES at birth but not current SES, which could conflate upward social mobility with the

[6]This is not explicitly used as a measure of childhood SES in their study, but is mentioned here as it is relevant to this study.

effects of having a higher current SES.

In a sample of African adults in Khayelitsha, Cape Town, Case and Menendez (2009) find a higher risk of obesity among women who were nutritionally deprived as children, but find no relationship for men. They find no significant interaction between current income and childhood nutrition. However, this study also finds a positive relationship between current income and BMI for women, but no significant relationship for men, a pattern contrary to that generally observed in the South African literature of a stronger relationship between SES and BMI for men (see e.g. Wittenberg, 2013). This suggests that the findings of this study may not be generalisable to the broader South African population.

Given the findings from high-income countries on the associations between childhood SES, social mobility and increased obesity risk, it is particularly interesting to explore these associations in developing countries in the midst of the nutrition transition, yet very few studies have done so. Furthermore, almost all studies (Addo et al. (2009) being the only exception) that have found an association between higher childhood SES and lower risk of obesity in adulthood have been conducted in countries where higher adult SES tends to be associated with lower obesity risk: countries that are already in stage 3 of the obesity transition. In other words, in these countries the association between childhood SES and BMI mirrors that between adult SES and BMI – childhood SES reflects the stage of the obesity transition these countries are currently in. It is thus particularly interesting to study the association between childhood SES and BMI in South Africa, a country that is still in stage 2 of the obesity transition: where the relationship between adult SES and BMI is still positive. Does the association between childhood SES and adult body weight mirror that of adult SES, or does it follow the pattern observed in the international literature? Does the association between childhood SES and adult body weight depend on what stage of the nutrition and obesity transitions the country is in? And do childhood SES and intergenerational mobility contribute to the shift to the later stages of the obesity transition? No studies of which we are aware have considered these questions. This paper aims to fill these gaps by investigating whether childhood SES and intergenerational social mobility are associated with higher obesity risk in South Africa, and whether these factors may contribute to the shift in the social gradient in body weight.

2.3 Data and methodology

2.3.1 Data

This study uses data from all five waves of the nationally representative National Income Dynamics Study (NIDS), collected in 2008, 2010-11, 2012, 2014-15 and 2017 (Southern Africa Labour and Development Research Unit, 2018).

The primary measure of body weight used in this study is BMI, which is defined as weight in kilograms divided by the square of height in metres. NIDS includes measures of the height, weight and waist circumference of adult respondents. These measures were plagued by inconsistencies, so the height and weight data were cleaned using the panel dimension of NIDS to flag inconsistencies and implausible values (see Appendix A.2 for more detail on the cleaning procedure). Because the cleaning procedure relies on consistency in height, the sample is limited to adults aged 25-64. Increases in height are possible even in the early twenties (Hulanicka & Kotlarz, 2009), and height starts to decline later in life (Cline et al., 1989; MedlinePlus, n.d.). The sample sizes of older adults, particularly those whose mothers had matric, are also relatively small, so limiting the sample to those below 65 helps to avoid issues with small sample sizes at older ages. We used several other measures of body weight as robustness checks: waist circumference, waist-height ratio, and a binary indicator for being obese[7]. Obesity is defined as a BMI of 30 or above. Waist-height ratio is the waist circumference divided by height.

The primary measure of childhood SES used in this study is an indicator for the respondent's mother having completed Grade 12. Studies in the international literature have commonly used parental education or occupation as measures of childhood SES[8]. The NIDS adult questionnaire includes questions on the education of respondents' parents[9]. Respondents were asked about the highest school grade completed by each of their parents, where the parent was deceased or not co-resident

[7]Probit models were used for the obesity indicator; the continuous outcomes all use ordinary least squares.

[8]Mother's education could be interpreted as representing childhood SES, as one's parents' SES effectively determines one's SES in childhood. This interpretation could lend itself to evaluating the accumulation model. However, mother's education could also be interpreted as representing one's mother's SES, seen through an intergenerational lens. When discussing social mobility, this study cannot distinguish between intergenerational social mobility (a change between one's parents' circumstances and one's own adult circumstances) and lifetime social mobility (a change between one's circumstances between childhood and adulthood).

[9]NIDS also includes questions on the occupation of respondents' parents, so this could also have been used as a measure of childhood SES. However, a large proportion of respondents reported that their mothers had never worked, and it is unclear whether this is an indicator of low SES or high SES (not working could have been due to unemployment or due to a choice to be a homemaker). Data on respondents' fathers has been found to be less consistent than that on mothers (Von Fintel & Posel, 2016). We therefore opted instead to use mother's education.

with the respondent. Where the parents were also sample members (co-resident or otherwise), the parental education data as reported by respondents was replaced with that reported by the parents themselves, as it is expected that parents will be able to report their own education more accurately than their children will. The national school-leaving certificate, or matric, is taken at the end of Grade 12. Achieving matric is a meaningful indicator in SA, as it is the gateway to higher education and the associated higher labour market returns – the returns to education increase sharply at around 12 years of schooling (Keswell & Poswell, 2004). We use mother's education because a relatively high number of children in SA grow up without their fathers present in the household. As such, it would be expected that mothers' education would be more accurately reported than that of fathers. Indeed, Von Fintel and Posel (2016) find that there was less inconsistency in mother's education than father's education across waves 1 and 2 of NIDS. However, the main analyses were also run for father's education as a robustness check.

In line with the findings of Von Fintel and Posel (2016) for the first two waves of NIDS, there was considerable inconsistency between waves in responses to the question on mother's education. In order to clean this variable, individuals with inconsistent responses across waves were excluded (i.e. all observations for that individual were dropped). This cleaning procedure only applied to respondents with maternal education data in more than one wave, and only applied to inconsistencies in 'known' values. If the respondent answered 'don't know' in one wave, but had at least one consistent non-missing response in other wave(s), then that unknown response was replaced with the non-missing value. If there was any inconsistency in known responses (excluding 'don't know' responses) across waves, the new variable was set to missing[10]. In order to maximise sample size, individuals were only dropped if there was inconsistency in the matric indicator variable itself, as opposed to any inconsistency in reported maternal education[11]. Furthermore, where mother's education was not available in one wave but was available in other waves, the value for the available wave(s) was used. This approach assumes that parents' schooling does not increase over time. Given that the sample is limited to adults aged 25-64, this seems a reasonable assumption for most cases. While it is possible that a respondent's parent did actually increase their schooling level

[10]As a robustness check, the main regressions were also run using a stricter cleaning procedure, where individuals were dropped if there was any inconsistency in their responses, including where respondents answered 'don't know'. The results were similar to those reported here, but with larger coefficients and smaller p-values.

[11]In other words, individuals were dropped from the sample if they said that their mother had matric in one wave but less than matric in another wave. If, however, they said that (for example) their mother had Grade 6 in one wave but Grade 10 in another wave, they were retained in the sample.

between waves, it is assumed that in the vast majority of cases parents will have completed their schooling by the time their children are aged 25. The cleaning procedure for maternal education resulted in 2.7 percent of the observations in the original estimation sample being dropped. The resulting estimation sample (including mother's education) did not differ markedly from the full sample (with complete data for the control variables) across a range of demographic characteristics (gender, race, province, income quintile, education, urban residence, age, marital status or wave).

2.3.2 Methods

In order to investigate the association between childhood SES and adult BMI, BMI was regressed on the indicator for the respondent's mother having matric[12], measures of current SES, and a series of controls using ordinary least squares. Given the different relationships between SES and obesity for men and women observed in much of the literature (see e.g. Case & Menendez, 2009), and in order to detect possible gender differences in the relationship between childhood SES and BMI, regressions were run separately for males and females.

Household income is used as the primary measure of current SES, which we use to explore social mobility between childhood and adulthood. Deflated household income is divided by the square root of household size, and then transformed using the inverse hyperbolic sine transformation. The inverse hyperbolic sine transformation is similar to a logarithmic transformation, but allows for the inclusion of zero values (Bellemare & Wichman, 2020). Household income is deflated to March 2017 values using the deflator files provided with NIDS. Household income is divided by the square root of household size in order to create a measure of household income per capita that adjusts for economies of scale within the household[13] (OECD, 2013). Regressions are shown for both a linear and quadratic specification of income. The linear specification allows us to assess whether the overall social gradient in BMI is positive or negative, but exploratory analyses (see Figure 3.2 in Chapter 3) revealed non-linearities in the SES-BMI association, suggesting that a quadratic specification is appropriate.

To explore whether the social gradient in BMI differs by childhood SES, we interact the indicator for mother having matric (hereafter 'childhood SES') with current household income. Instead of

[12] Unknown maternal education was initially included as a separate category, but it was not significantly different from mother having less than matric, so in the final results unknown maternal education was included with less than matric.

[13] E.g. a household with four members is likely to need fewer resources than four households with one member each.

dividing income into 'high' and 'low' or other arbitrary cutoffs, we use continuous income and allow it to vary by childhood SES. We explore the interaction using predictive margins, or average adjusted predictions[14] (i.e. the predicted BMI for various values of income at each level of mother education). This also allows us to see at what point in the income distribution childhood SES has the strongest association with BMI, and allows us to explore associations between social mobility and BMI. By contrasting predicted BMI for those with a low childhood SES versus those with a high childhood SES at high levels of adult income, we are able to draw some conclusions about social mobility.

The regressions also control for age, race (reference group African), respondents' own education (categorised into 'less than matric', 'matric' and 'post matric', with 'less than matric' used as the reference category), an indicator for being employed, an indicator for being married or cohabiting, an indicator for residing in an urban area, and province. Indicators for each wave are also included to account for possible time trends in BMI, such as secular increases in BMI or shocks induced by an economic crisis. A quadratic term for age was included to allow for a non-linear relationship between age and BMI.

We would expect urban areas in South Africa to be further along in the nutrition transition, and initial results showed that mean BMI is higher in urban areas. We explored whether the association between childhood SES and BMI differs for urban and rural residents by interacting the indicator for mother having matric with the indicator for urban residence. After finding a significant negative interaction for women (i.e. higher childhood SES has a more negative association with BMI for urban residents), we ran separate regressions for urban and rural residents to allow for different relationships between childhood SES, social mobility and BMI in urban and rural areas.

We also investigate possible transmission mechanisms using a series of regressions sequentially adding controls for potential transmission mechanisms: current SES variables and health behaviours. The health behaviours considered are indicators for smoking and exercising weekly. Alcohol use was excluded because the questions on alcohol were not asked in wave 5. If these variables are significant transmission mechanisms, we would expect the size of the coefficient on mother having matric to be reduced. These regressions were all run on the same sample, i.e. the sample with complete observations for the health behaviour variables as well as the other covariates.

[14]In the calculation of the adjusted predictions the values of the independent variables are set at their observed values, rather than at their means. See Williams (2012) for a discussion of the differences between these options.

We use the pooled sample of observations from all five waves of NIDS. To account for similarities between repeated observations on the same individual, standard errors are clustered by individual. The OLS regressions are weighted using the post-stratified weights provided with NIDS. These weights are calibrated to mid-year population estimates so that the sample is representative of the population (Brophy et al., 2018). The post-stratified weights are meant to compensate for the under- or over-representation of certain subgroups in the sample, but these weights do not correct for missing values on BMI, mother's education, or any of the control variables. Table A.3.1 in Appendix A.3 shows the impact of weighting, with the first column showing the characteristics of the estimation sample without weights, the second column showing the characteristics of the estimation sample with weights, and the final column the weighted full sample (with non-missing values on the sample characteristics in question), which represents estimates of the population characteristics. The unweighted sample proportions are considerably different to the estimates of the population proportions (the full sample weighted proportions). Weighting the data using the post-stratified weights does not fully bring the estimation sample characteristics in line with the estimated population characteristics, but it does bring them much closer. This is particularly notable in the case of urban residence, gender, race, income and mother education.

2.3.2.1 Random effects methodology

We investigate whether the social gradient in BMI and its interaction with childhood SES is driven by changes in income within individuals over time or differences in average income between individuals using random effects within-between models[15]. We also use random effects within-between models to explore whether there is any interaction between childhood SES and changes in urban residence within individuals. Because mother's education does not vary within individuals (by construction), a fixed effects model is not appropriate as it cannot explore the effects of variables that do not change over time. A random effects model allows time-invariant variables to be included.

[15] We distinguish here between three types of mobility:

1. Intergenerational social mobility, captured by comparing mother's SES (childhood SES) with the respondent's own adult SES.
2. Short-run mobility, captured by changes in own SES (income) between waves.
3. Long-run life course mobility. This is mobility over the individual's own life course, independent of their parents or starting point in life. Given the relatively short time period covered by NIDS, we cannot compare the same person at different stages of their own long-run life course trajectory. However, by comparing different people with different levels of SES at different points in the life cycle, the 'between' effects in the random effects within-between models could be seen as proxying for the effects of changing SES over the life course.

The random effects model (also known as a mixed, multilevel or hierarchical model in some disciplines) consists of two (or more) levels, where repeated observations of individuals are the first level and individuals are the second level. Thus variables that change within individuals over time are at level 1, and variables that only vary between individuals are at level 2. The random effects within-between model, also known as a hybrid model (Allison, 2009), is an extension of the random effects model. It allows within-individual and between-individual effects to be estimated separately. Instead of the raw time-varying variables, it includes deviations from the individual-specific mean of those variables, as well as the individual means themselves. The inclusion of the individual-specific means of time-varying variables is similar to introducing fixed effects, and allows the assumption of no correlation between these variables and the random effects to be relaxed (Schunck & Perales, 2017). This model is represented in equation (2.1):

$$BMI_{it} = \beta_0 + \beta_W(I_{it} - \bar{I}_i) + \beta_B\bar{I}_i + \beta_3 mother\_matric_i + \mu_i + \epsilon_{it} \tag{2.1}$$

Here I_{it} represents household income[16] for individual i in wave t, and $\bar{I}_i$ is the average household income for individual i across all waves. β_W and β_B are the coefficients for income within and between individuals respectively[17]. The demeaned within-individual coefficients are identical to the coefficients from a fixed effects model. Individual time-invariant heterogeneity is modelled by the inclusion of an individual-specific 'random effect' for each individual at level 2, represented by μ_i, which is assumed to have a mean of zero and to be normally distributed. ϵ_{it} is the level 1 residual. The variance of ϵ_{it} is the within-individual variance, while the variance in μ_i is the variance between individuals[18].

The random effects within-between models also include random slopes for income, and where appropriate, squared income, because failing to include random slopes when estimating a cross-level

[16]Wherever this paper refers to income or household income, it refers to the inverse hyperbolic sine transformation of deflated household income, adjusted for economies of scale as detailed above.

[17]Other time-varying and time-invariant covariates are included, but for simplicity are not shown here.

[18]This model is very similar to the 'Mundlak' or correlated random effects model (Mundlak, 1978), which includes the raw time-varying variables and their means instead of the demeaned variable. The two models are mathematically equivalent, but differ in their interpretation of the between-individual effects (Schunck, 2013). In both the random effects within-between and Mundlak models, the demeaned within-individual coefficients are identical to the coefficients from a fixed effects model. As with the fixed effects model, these coefficients will be unbiased by any time-invariant individual characteristics, but may still be biased by unobserved time-varying characteristics. Furthermore, any individual-level time invariant variables in the model may still be biased by correlation with unobserved individual characteristics.

interaction can lead to standard errors that are too small (Barr, 2013; Bell et al., 2019; Heisig & Schaeffer, 2019)[19]. Sampling weights were included at each level of the random effects within-between models. Incorporating sampling weights into random effects models, including the within-between model, is more complex than in the single-level case (as described in Section 2.3.2 for the OLS models). The details of the procedure used to calculate appropriate weights for the random effects within-between models is available in Appendix A.1.

2.4 Results

2.4.1 Descriptive statistics

Table 2.1 shows the weighted sample characteristics by childhood SES. Respondents in the youngest age group (25-34), whites and Indians, and urban residents were more likely to have high childhood SES. Those with high childhood SES were also more likely to be in the top household income quintile, to have achieved matric or post matric themselves, and to be employed. Table 2.2 shows the unadjusted mean BMI by demographic characteristics. Women with high childhood SES had a lower mean BMI than those with low childhood SES, with the opposite pattern for men.

2.4.2 Main results

Tables 2.3 and 2.4 show the results of OLS regressions (not within-between; these models do not distinguish between SES differences between individuals and changes within individuals) of BMI on childhood SES, adult SES variables and other demographic characteristics[20]. The first two columns (columns 1 and 2) show regressions without interactions, with a linear and quadratic income specification respectively. While higher adult SES (income) was associated with higher BMI, as observed in the previous South African literature, high childhood SES was associated with a roughly 1.4 kg/m^2 lower BMI for women, but only at the 10 percent level, and a reduction in BMI of around 1.2 kg/m^2 for men. The following two columns show regressions interacting the childhood SES indicator with linear (column 3) and quadratic (column 4) adult income. There was

[19]Random slopes allow the coefficients of time-varying variables to vary between individuals. As with a random intercept, random slopes are assumed to be drawn from a normal distribution. Allowing for random slopes can improve the fit of models estimating direct effects, but is crucial when estimating interactions between variables at different levels of the model, i.e. between time-invariant and time-varying variables (Barr, 2013; Bell et al., 2019; Heisig & Schaeffer, 2019).

[20]Across all regressions the R-squared is low, particularly for women, but this is to be expected given that much of the variation in BMI is explained by genetic factors.

Table 2.1: Sample characteristics by mother education

	Mother matric		Mother less than matric	
	Mean	N	Mean	N
Total	0.07	1359	0.93	35284
BMI	27.41	1359	27.48	35284
Female	0.07	680	0.93	21192
Male	0.08	679	0.92	14092
Age 25-34	0.10	731	0.90	11634
Age 35-44	0.07	315	0.93	9137
Age 45-54	0.04	173	0.96	8250
Age 55-64	0.07	140	0.93	6263
African	0.03	711	0.97	29147
Coloured	0.05	109	0.95	5142
Asian/Indian	0.15	38	0.85	414
White	0.49	501	0.51	581
Income quintile 1	0.02	84	0.98	7412
Income quintile 2	0.02	75	0.98	7939
Income quintile 3	0.03	142	0.97	8340
Income quintile 4	0.05	235	0.95	7215
Income quintile 5	0.23	823	0.77	4367
Less than matric	0.02	227	0.98	25232
Matric	0.10	354	0.90	4639
Post matric	0.20	778	0.80	5413
Not employed	0.04	390	0.96	17223
Employed	0.10	969	0.90	18061
Rural	0.02	297	0.98	17222
Urban	0.10	1062	0.90	18062
Not married/cohabiting	0.07	688	0.93	19559
Married/cohabiting	0.08	671	0.92	15725

Table 2.2: Mean BMI by demographic characteristics

	Women			Men		
	Mean BMI	S.E.	N	Mean BMI	S.E.	N
Female	30.21	0.17	21872	24.41	0.16	14771
Mother less than matric	30.26	0.18	21192	24.31	0.17	14092
Mother matric	29.50	0.50	680	25.51	0.35	679
Age 25-34	28.53	0.20	6813	23.34	0.15	5552
Age 35-44	30.70	0.24	5679	24.45	0.21	3773
Age 45-54	31.55	0.30	5266	25.18	0.28	3157
Age 55-64	31.17	0.26	4114	26.10	0.28	2289
African	30.21	0.19	17995	24.04	0.16	11863
Coloured	30.01	0.36	3020	24.71	0.56	2231
Asian/Indian	28.28	0.88	261	25.07	0.63	191
White	31.04	0.63	596	28.14	0.45	486
Income quintile 1	29.00	0.24	5044	22.53	0.16	2452
Income quintile 2	29.38	0.22	5225	22.61	0.17	2789
Income quintile 3	30.18	0.22	5068	23.36	0.14	3414
Income quintile 4	31.05	0.26	3917	24.59	0.18	3533
Income quintile 5	31.41	0.39	2611	27.09	0.23	2579
Less than matric	30.09	0.18	15710	23.62	0.16	9749
Matric	29.45	0.31	2705	25.06	0.30	2288
Post matric	31.03	0.32	3457	25.83	0.26	2734
Not employed	29.96	0.20	12301	23.25	0.16	5312
Employed	30.48	0.20	9571	24.90	0.18	9459
Rural	29.50	0.23	10958	23.59	0.13	6561
Urban	30.63	0.23	10914	24.79	0.23	8210
Not married/cohabiting	29.51	0.23	12624	23.21	0.18	7623
Married/cohabiting	30.99	0.21	9248	25.77	0.19	7148

a significant negative interaction between high childhood SES and income for both women and men (column 3), suggesting that the social gradient (of adult SES) in BMI is more negative for those with high childhood SES. In models including an interaction between childhood SES and squared income (column 4), only the quadratic term was significant, suggesting that the interaction was driven by those with relatively high levels of income.

Figures 2.1 to 2.4 illustrate the interaction between childhood SES and adult income using the predictive margins, or adjusted predictions, of BMI at various levels of income for respondents with high versus low childhood SES (corresponding to column 3 in Tables 2.3 and 2.4). Figure 2.1 shows that household income in adulthood is positively related to BMI for women with low childhood SES, but insignificantly negatively related to BMI for women with high childhood SES (the slope is -0.48, with a p-value of 0.341). The average marginal effect of income is significantly lower for women with high childhood SES (p-value of difference in slopes = 0.014). Predicted BMI only differs significantly between those with high childhood SES and those with low childhood SES at high levels of household income. Among men (Figure 2.2), income is positively related to predicted BMI for both low and high childhood SES groups, but the average marginal effect of income is not significantly different from zero for those with high childhood SES, and the marginal effect of income is significantly lower for those with high childhood SES (p-value of difference in slopes = 0.005).

Figures 2.3 and 2.4 illustrate the interaction between income and childhood SES allowing for a nonlinear relationship between income and BMI (corresponding to column 4 in Tables 2.3 and 2.4). For women with low childhood SES, predicted BMI increases with additional income across the whole range of household income, but for women with high childhood SES, after a certain level of income predicted BMI decreases with additional income. The income-BMI relationship flattens out at high income levels for men with high childhood SES, but does not fully reverse as it does for women. At high levels of adult household income, those with a high childhood SES had a significantly lower predicted BMI. Those who came from a low SES childhood background but have high income in adulthood are at greater risk of high BMI than those high-income adults who also had high childhood SES, suggesting that social mobility from low childhood SES to high adult SES may increase adult obesity risk compared to adults who remained high SES in both childhood and adulthood. The protective effect of high childhood SES seems to be driven by those with high levels

Table 2.3: OLS regressions for combined urban and rural samples, women

	(1)	(2)	(3)	(4)	(5)
Mother matric	−1.49**	−1.40**	0.31	0.01	1.78
	(0.72)	(0.71)	(0.91)	(0.89)	(1.49)
Household income	0.71***	0.73***	0.79***	0.79***	0.71***
	(0.14)	(0.14)	(0.14)	(0.14)	(0.14)
Income sq.		−0.09*		−0.03	
		(0.05)		(0.05)	
Urban	0.63**	0.65**	0.62**	0.62**	0.76***
	(0.28)	(0.28)	(0.28)	(0.28)	(0.28)
Mother matric x Household income			−1.29***	0.13	
			(0.49)	(0.64)	
Mother matric x income sq.				−0.52**	
				(0.20)	
Mother matric x Urban					−3.85**
					(1.54)
Age	0.56***	0.57***	0.57***	0.57***	0.57***
	(0.09)	(0.09)	(0.09)	(0.09)	(0.09)
Age sq.	−0.01***	−0.01***	−0.01***	−0.01***	−0.01***
	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
Matric	−0.03	−0.01	−0.08	−0.08	−0.08
	(0.37)	(0.37)	(0.37)	(0.37)	(0.37)
Post matric	0.72**	0.80**	0.71*	0.75**	0.70*
	(0.37)	(0.37)	(0.37)	(0.37)	(0.37)
Employed	−0.24	−0.25	−0.23	−0.24	−0.24
	(0.21)	(0.21)	(0.21)	(0.21)	(0.21)
Married/cohabiting	1.12***	1.13***	1.12***	1.14***	1.11***
	(0.26)	(0.26)	(0.26)	(0.26)	(0.26)
Coloured	−1.53***	−1.50***	−1.49***	−1.50***	−1.52***
	(0.55)	(0.55)	(0.55)	(0.55)	(0.55)
Asian/Indian	−4.67***	−4.50***	−4.75***	−4.71***	−4.69***
	(0.93)	(0.92)	(0.92)	(0.92)	(0.93)
White	−1.37	−1.20	−1.02	−1.01	−1.13
	(0.92)	(0.93)	(0.95)	(0.95)	(0.93)
Constant	16.20***	16.03***	15.97***	15.95***	16.01***
	(1.83)	(1.83)	(1.83)	(1.83)	(1.83)
Observations	21889	21889	21889	21889	21889
Adjusted R^2	0.08	0.08	0.08	0.08	0.08

* p <0.1, ** p <0.05, *** p <0.01. Standard errors in parentheses.

Note: Standard errors clustered by individual. Controls for province and wave included but not displayed.

Table 2.4: OLS regressions for combined urban and rural samples, men

	(1)	(2)	(3)	(4)	(5)
Mother matric	−1.14***	−1.23***	0.18	0.33	−0.09
	(0.41)	(0.41)	(0.47)	(0.41)	(0.68)
Household income	1.14***	1.08***	1.21***	1.15***	1.15***
	(0.11)	(0.10)	(0.11)	(0.10)	(0.11)
Income sq.		0.15***		0.20***	
		(0.03)		(0.03)	
Urban	0.39**	0.34*	0.37*	0.31	0.43**
	(0.20)	(0.20)	(0.20)	(0.19)	(0.20)
Mother matric x Household income			−0.98***	−0.30	
			(0.33)	(0.33)	
Mother matric x income sq.				−0.39**	
				(0.16)	
Mother matric x Urban					−1.18
					(0.80)
Age	−0.04	−0.05	−0.03	−0.05	−0.03
	(0.06)	(0.06)	(0.06)	(0.06)	(0.07)
Age sq.	0.00	0.00	0.00	0.00	0.00
	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
Matric	1.20***	1.16***	1.16***	1.08***	
	(0.29)	(0.29)	(0.29)	(0.29)	
Post matric	1.11***	0.96***	1.11***	0.91***	
	(0.25)	(0.26)	(0.25)	(0.25)	
Employed	0.33*	0.36**	0.33*	0.35**	0.33*
	(0.17)	(0.17)	(0.17)	(0.17)	(0.17)
Married/cohabiting	1.87***	1.89***	1.87***	1.88***	1.88***
	(0.22)	(0.22)	(0.21)	(0.21)	(0.22)
Coloured	−0.31	−0.35	−0.32	−0.39	−0.32
	(0.39)	(0.39)	(0.39)	(0.38)	(0.39)
Asian/Indian	−1.56*	−1.70*	−1.46*	−1.60**	−1.51*
	(0.83)	(0.87)	(0.79)	(0.79)	(0.83)
White	1.28**	0.93	1.65***	1.34**	1.33**
	(0.60)	(0.60)	(0.63)	(0.62)	(0.62)
Matric					1.16***
					(0.29)
Post matric					1.07***
					(0.25)
Constant	21.99***	22.16***	21.80***	22.08***	21.89***
	(1.31)	(1.30)	(1.31)	(1.29)	(1.31)
Observations	14771	14771	14771	14771	14669
Adjusted R^2	0.19	0.19	0.19	0.20	0.19

* p <0.1, ** p <0.05, *** p <0.01. Standard errors in parentheses.

Note: Standard errors clustered by individual. Controls for province and wave included but not displayed.

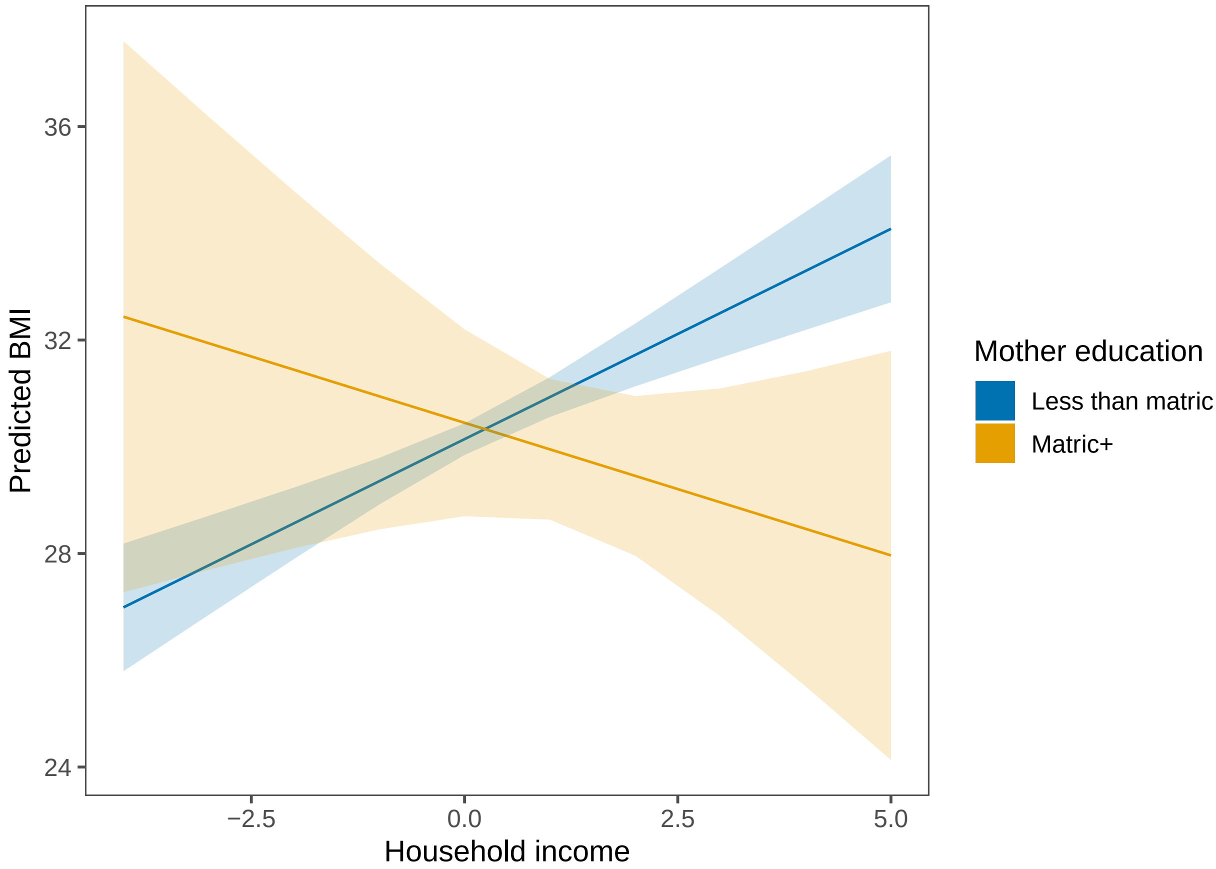


Figure 2.1: Illustration of interaction between mother education and linear household income, women

Source: Unless otherwise stated, all figures are based on the author's own calculations using the NIDS data.

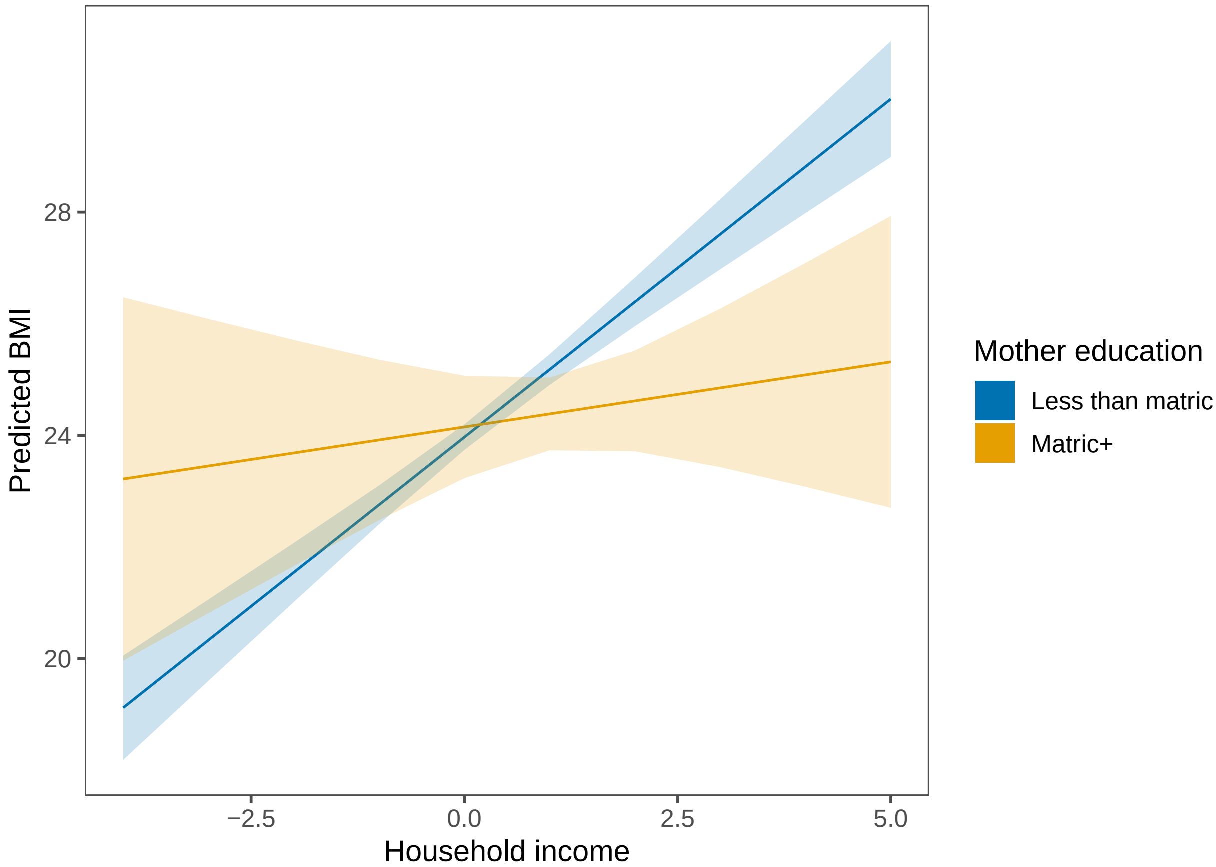


Figure 2.2: Illustration of interaction between mother education and linear household income, men

of adult household income: predicted BMI was only significantly higher for those from a low SES background at relatively high income levels.

The results presented above are for urban and rural areas combined; this may mask differences between urban and rural areas, which may be at different stages of the nutrition transition. The significant negative interaction between childhood SES and urban residence in the last column (5) of Table 2.3 suggests that high childhood SES is associated with lower BMI in urban but not rural areas. Therefore, the remaining tables show results for urban and rural areas separately. Tables 2.5 and 2.6 present results of OLS regressions controlling for current SES variables and other covariates, separately for urban and rural areas. In urban areas, high childhood SES was associated with a 1.8 kg/m^2 lower BMI for women and with a 1.15 kg/m^2 lower BMI for men. Childhood SES was not significantly associated with BMI in rural areas. Results for obesity, waist circumference and waist-to-height ratio (see Tables A.4.3 to A.4.8 in Appendix A.4) show similar patterns: in urban areas, high childhood SES is associated with a lower probability of obesity; a 0.03 points lower waist-to-height ratio for women and a 0.01 points lower waist-to-height ratio for men; and a 3.24 cm lower waist circumference for women, though the latter was only significant at the 10% level. In rural areas the association between childhood SES and these other body weight measures is not significant. This, together with the fact that predicted BMI is only significantly lower among those with high childhood SES at high levels of household income, suggests that childhood SES may be more likely to be protective against obesity where the nutrition transition is more advanced.

Results for the random effects within-between models (Tables 2.7 and 2.8) show that the social gradient in BMI is driven primarily by differences in average income between individuals, rather than relatively short-run changes in income within individuals. Changes in household income within individuals were not significantly associated with BMI for women, though they were positively associated with BMI for urban men. Even for urban men, within-individual income changes were dominated by between-individual differences in average income. The flatter social gradient in BMI among those with high childhood SES also appears to be driven more by between-individual differences in the social gradient than by within-individual income changes, as shown by the generally insignificant interaction between childhood SES and within-individual income (except for a significant positive interaction for rural women), and the significant negative interaction between childhood SES and between-individual income for urban residents (though only significant at the

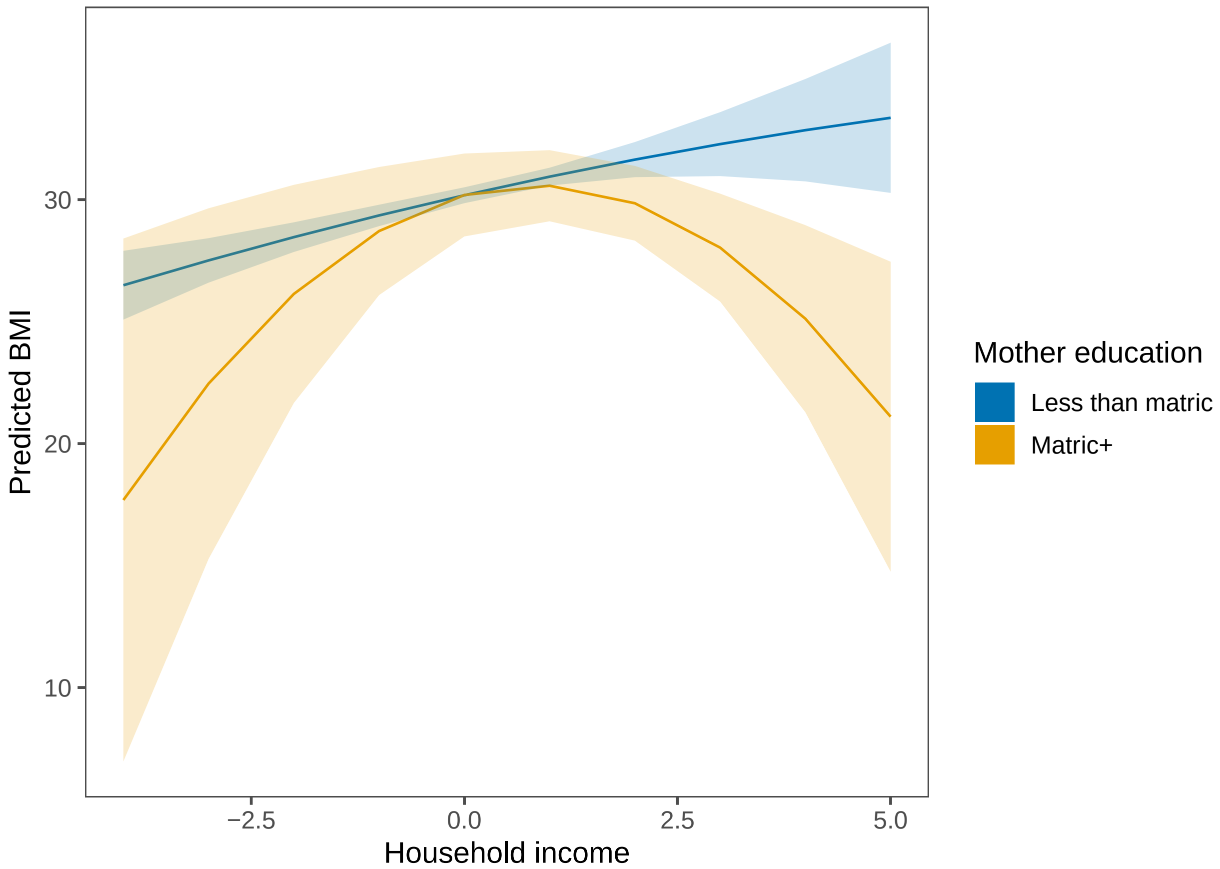


Figure 2.3: Illustration of interaction between mother education and quadratic household income, women

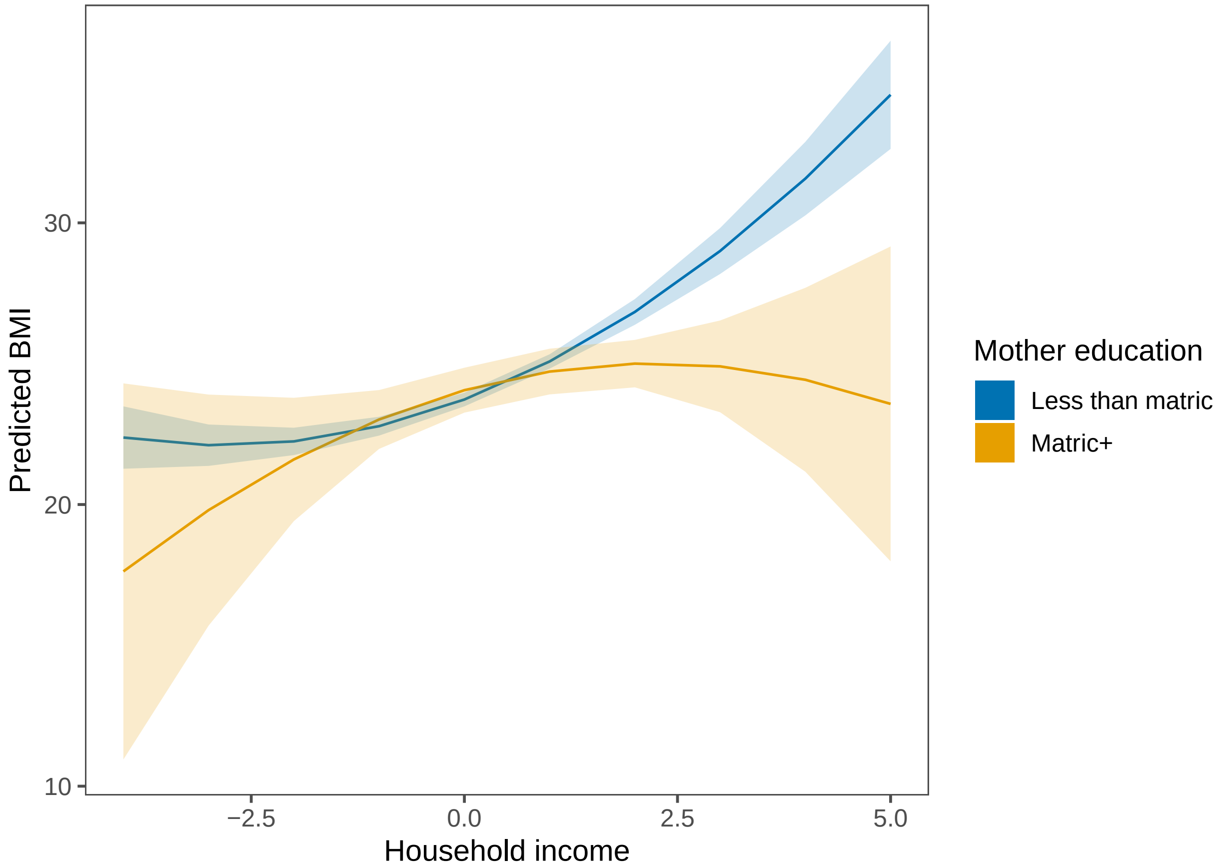


Figure 2.4: Illustration of linear interaction between mother education and quadratic household income, men

Table 2.5: OLS regressions stratified by urban residence, women

	Urban				Rural			
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Mother matric	−1.84**	−1.77**	−0.51	−1.32	1.01	0.99	1.78	2.62
	(0.75)	(0.74)	(0.91)	(0.86)	(1.43)	(1.44)	(1.50)	(1.86)
Household income	0.62***	0.66***	0.69***	0.71***	0.94***	0.97***	0.99***	1.03***
	(0.19)	(0.18)	(0.20)	(0.19)	(0.16)	(0.17)	(0.16)	(0.18)
Income sq.		−0.08		−0.03		0.06		0.09
		(0.05)		(0.05)		(0.07)		(0.07)
Mother matric x Household income			−0.86*	1.34			−1.56**	−0.91
			(0.51)	(0.81)			(0.77)	(0.63)
Mother matric x income sq.				−0.73***				−0.96*
				(0.24)				(0.57)
Age	0.55***	0.56***	0.55***	0.55***	0.57***	0.57***	0.58***	0.57***
	(0.12)	(0.12)	(0.12)	(0.12)	(0.10)	(0.10)	(0.10)	(0.10)
Age sq.	−0.01***	−0.01***	−0.01***	−0.01***	−0.01***	−0.01***	−0.01***	−0.01***
	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
Matric	−0.49	−0.47	−0.53	−0.53	0.85*	0.85*	0.84*	0.82*
	(0.49)	(0.48)	(0.49)	(0.48)	(0.48)	(0.48)	(0.48)	(0.48)
Post matric	0.17	0.24	0.16	0.19	2.33***	2.28***	2.32***	2.26***
	(0.46)	(0.47)	(0.46)	(0.47)	(0.48)	(0.48)	(0.47)	(0.47)
Employed	−0.22	−0.23	−0.20	−0.23	−0.20	−0.19	−0.21	−0.21
	(0.30)	(0.30)	(0.30)	(0.30)	(0.24)	(0.24)	(0.24)	(0.24)
Married/cohabiting	1.21***	1.22***	1.22***	1.24***	1.05***	1.04***	1.05***	1.04***
	(0.37)	(0.37)	(0.37)	(0.37)	(0.28)	(0.28)	(0.28)	(0.28)
Coloured	−1.40**	−1.37**	−1.37**	−1.42**	−2.18**	−2.18**	−2.30**	−2.34**
	(0.60)	(0.60)	(0.60)	(0.60)	(1.06)	(1.06)	(1.05)	(1.05)
Asian/Indian	−5.09***	−4.89***	−5.17***	−5.13***	−2.98***	−3.02***	−3.01***	−3.13***
	(1.12)	(1.13)	(1.12)	(1.13)	(1.15)	(1.15)	(1.15)	(1.15)
White	−0.71	−0.59	−0.55	−0.60	−4.92***	−5.07***	−4.73***	−4.84***
	(0.98)	(0.98)	(1.00)	(1.01)	(1.31)	(1.35)	(1.28)	(1.33)
Constant	17.02***	16.89***	16.87***	16.89***	15.90***	15.95***	16.00***	16.18***
	(2.53)	(2.53)	(2.54)	(2.53)	(2.54)	(2.54)	(2.52)	(2.49)
Observations	10942	10942	10942	10942	10947	10947	10947	10947
Adjusted R^2	0.07	0.07	0.07	0.07	0.10	0.10	0.11	0.11

* p <0.1, ** p <0.05, *** p <0.01. Standard errors in parentheses.

Note: Standard errors clustered by individual. Controls for province and wave included but not displayed.

Table 2.6: OLS regressions stratified by urban residence, men

	Urban				Rural			
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Mother matric	−1.17**	−1.23***	0.48	0.55	−0.44	−0.55	−0.33	−0.49
	(0.46)	(0.46)	(0.55)	(0.46)	(0.62)	(0.62)	(0.64)	(0.76)
Household income	1.29***	1.16***	1.40***	1.23***	0.73***	0.80***	0.73***	0.81***
	(0.15)	(0.12)	(0.15)	(0.13)	(0.12)	(0.12)	(0.12)	(0.13)
Income sq.		0.14***		0.23***		0.12***		0.12***
		(0.05)		(0.05)		(0.03)		(0.03)
Mother matric x Household income			−1.18***	−0.26			−0.11	−0.49
			(0.36)	(0.37)			(0.44)	(0.51)
Mother matric x income sq.				−0.45***				0.20
				(0.17)				(0.31)
Age	0.02	0.01	0.02	−0.00	−0.17**	−0.17**	−0.17**	−0.17**
	(0.09)	(0.09)	(0.09)	(0.09)	(0.08)	(0.08)	(0.08)	(0.08)
Age sq.	0.00	0.00	0.00	0.00	0.00**	0.00***	0.00**	0.00**
	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
Matric	0.99***	0.97***	0.94***	0.87**	1.58***	1.52***	1.58***	1.51***
	(0.34)	(0.34)	(0.35)	(0.34)	(0.42)	(0.43)	(0.42)	(0.43)
Post matric	0.85***	0.73**	0.83***	0.63**	1.55***	1.41***	1.55***	1.42***
	(0.31)	(0.32)	(0.31)	(0.31)	(0.34)	(0.35)	(0.35)	(0.35)
Employed	0.37	0.42*	0.36	0.41*	0.32*	0.31	0.32*	0.31
	(0.25)	(0.25)	(0.25)	(0.24)	(0.19)	(0.19)	(0.19)	(0.19)
Married/cohabiting	2.09***	2.13***	2.09***	2.13***	1.46***	1.43***	1.46***	1.43***
	(0.29)	(0.29)	(0.28)	(0.27)	(0.26)	(0.26)	(0.26)	(0.26)
Coloured	−0.26	−0.29	−0.28	−0.35	−1.18*	−1.16	−1.19*	−1.16
	(0.43)	(0.43)	(0.43)	(0.43)	(0.72)	(0.71)	(0.72)	(0.71)
Asian/Indian	−1.61	−1.81	−1.34	−1.56	−1.15	−1.27	−1.15	−1.28
	(1.18)	(1.25)	(1.01)	(1.01)	(1.10)	(1.11)	(1.10)	(1.11)
White	1.03	0.76	1.42**	1.10*	4.14**	3.94*	4.17**	3.93*
	(0.63)	(0.64)	(0.66)	(0.65)	(2.09)	(2.10)	(2.11)	(2.13)
Constant	21.02***	21.08***	20.88***	21.12***	25.20***	25.27***	25.19***	25.23***
	(1.71)	(1.70)	(1.71)	(1.70)	(1.90)	(1.90)	(1.91)	(1.91)
Observations	8212	8212	8212	8212	6559	6559	6559	6559
Adjusted R^2	0.21	0.21	0.22	0.22	0.11	0.11	0.11	0.11

* p <0.1, ** p <0.05, *** p <0.01. Standard errors in parentheses.

Note: Standard errors clustered by individual. Controls for province and wave included but not displayed.

10 percent level for men). Inspection of the random effects components shows that the variance in BMI between individuals (the variance in the intercept) was much greater than that within individuals (the residual variance). The intraclass correlation coefficient[21], calculated on the null model (i.e. with no explanatory variables, not shown), was a fairly high 0.81 for women and 0.78 for men. This indicates a high similarity between BMI measurements on the same individual, which is to be expected given the slow-changing nature of BMI. Taken together, these results suggest that the social gradient in body weight may be driven more by long-run factors related to SES and by social mobility over the entire life course than by very short-run changes.

2.4.3 Robustness checks

Results for a series of robustness checks are available in Appendices A.6 to A.9.

2.4.3.1 Alternative measures of childhood SES

Mother's education is an imperfect proxy for childhood SES. As educational attainment has steadily increased over many decades in South Africa (see e.g. van der Berg, 2007), for older cohorts completing matric may indicate a higher SES than for younger cohorts. Furthermore, it is possible that low maternal education may to some extent be capturing the effect of having a mother who grew up in a rural area. Mothers who grew up in rural areas may be more likely to have lower levels of education, and we do not observe where mothers grew up. Having a mother who grew up in a rural area could plausibly affect one's BMI other than through one's childhood SES, for example through epigenetic pathways[22]. Finally, mother's education could be interpreted as an indicator of the mother's health literacy rather than childhood SES per se (though either pathway is likely to affect early life nutrition). We therefore explore the robustness of the results to using several alternative measures of childhood SES.

First, using an indicator for father having matric showed similar patterns to those for mother having matric reported above (Appendix A.6). Father having matric was associated with lower BMI for urban women, and for both urban and rural men, though only at the 10 percent level for men. For women, the interaction terms between father having matric and household income

[21] The intraclass correlation coefficient is the ratio of the individual-level error variance to the total error variance.

[22] For example, if one's mother grew up in a rural area but then migrated to an urban area, she may face a higher risk of obesity due to a mismatch between her own childhood and adult nutritional environments, and this obesity risk could then be passed on to her offspring.

Table 2.7: Random effects within-between models stratified by urban residence, women

	Urban				Rural			
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Fixed coefficients								
Mother matric	−1.76***	−1.64**	0.21	−0.43	1.19	1.12	1.64	3.03
	(0.67)	(0.67)	(0.95)	(0.93)	(1.35)	(1.37)	(1.60)	(1.95)
Matric	−0.12	−0.10	−0.14	−0.13	0.33	0.32	0.33	0.29
	(0.35)	(0.35)	(0.35)	(0.35)	(0.25)	(0.25)	(0.25)	(0.25)
Post matric	0.20	0.23	0.19	0.21	0.75***	0.72***	0.74***	0.70***
	(0.33)	(0.33)	(0.33)	(0.33)	(0.25)	(0.25)	(0.25)	(0.25)
Within coefficients:								
Income (W)	−0.03	−0.01	−0.04	−0.03	0.08	0.07	0.06	0.05
	(0.09)	(0.09)	(0.09)	(0.09)	(0.08)	(0.09)	(0.09)	(0.09)
Income sq. (W)		−0.01		−0.00		−0.01		−0.02
		(0.05)		(0.05)		(0.04)		(0.04)
Mother matric x income (W)			0.21	0.93			0.68	0.64
			(0.40)	(0.70)			(0.41)	(0.41)
Mother matric x income sq. (W)				−0.25				0.09
				(0.19)				(0.29)
Employed (W)	0.04	0.03	0.04	0.03	−0.22	−0.23	−0.23	−0.23
	(0.13)	(0.13)	(0.13)	(0.13)	(0.14)	(0.14)	(0.14)	(0.14)
Age (W)	0.63***	0.63***	0.62***	0.63***	0.46***	0.45***	0.46***	0.46***
	(0.11)	(0.11)	(0.11)	(0.11)	(0.11)	(0.11)	(0.11)	(0.11)
Age sq. (W)	−0.01***	−0.01***	−0.01***	−0.01***	−0.01***	−0.01***	−0.01***	−0.01***
	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
Married/cohabiting (W)	0.52***	0.52***	0.52***	0.51***	0.13	0.13	0.13	0.13
	(0.20)	(0.20)	(0.20)	(0.20)	(0.20)	(0.20)	(0.20)	(0.19)
Between coefficients:								
Income (B)	0.82***	0.85***	0.95***	0.96***	1.70***	1.75***	1.74***	1.81***
	(0.22)	(0.24)	(0.23)	(0.23)	(0.24)	(0.26)	(0.24)	(0.27)
Income sq. (B)		−0.10		−0.05		0.10		0.15**
		(0.09)		(0.08)		(0.08)		(0.08)
Mother matric x income (B)			−1.38***	0.95			−0.79	2.20
			(0.50)	(0.97)			(1.07)	(1.43)
Mother matric x income sq. (B)				−0.81***				−2.30**
				(0.30)				(1.01)
Employed (B)	−0.45	−0.43	−0.39	−0.43	−0.31	−0.33	−0.32	−0.32
	(0.46)	(0.46)	(0.46)	(0.46)	(0.42)	(0.43)	(0.43)	(0.43)
Age (B)	0.64***	0.65***	0.63***	0.64***	0.58***	0.57***	0.58***	0.57***
	(0.15)	(0.15)	(0.15)	(0.15)	(0.12)	(0.12)	(0.12)	(0.12)
Age sq. (B)	−0.01***	−0.01***	−0.01***	−0.01***	−0.01***	−0.01***	−0.01***	−0.01***
	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
Married/cohabiting (B)	0.94**	0.97**	0.98**	1.03**	1.11***	1.10***	1.11***	1.13***
	(0.44)	(0.44)	(0.44)	(0.44)	(0.33)	(0.33)	(0.33)	(0.33)
Constant	14.23***	14.08***	14.06***	14.05***	16.04***	16.08***	16.10***	16.29***
	(3.06)	(3.06)	(3.06)	(3.05)	(2.84)	(2.84)	(2.82)	(2.77)
var(Income (W))	0.82	0.73	0.81	0.73	0.62	0.62	0.60	0.60
var(Intercept)	42.32	42.25	42.15	42.02	31.99	31.98	31.98	31.83
var(Residual)	8.12	8.12	8.12	8.12	10.18	10.18	10.19	10.18
var(Income sq. (W))		0.02		0.01		0.00		0.00
Observations	10942	10942	10942	10942	10947	10947	10947	10947

* p <0.1, ** p <0.05, *** p <0.01. Standard errors in parentheses.

Note: Controls for race group, province and wave included but not displayed.

Table 2.8: Random effects within-between models stratified by urban residence, men

	Urban				Rural			
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Fixed coefficients								
Mother matric	−0.48	−0.66	0.39	0.38	0.35	0.15	0.37	−0.05
	(0.42)	(0.41)	(0.59)	(0.49)	(0.55)	(0.55)	(0.58)	(0.74)
Matric	0.39*	0.33	0.37*	0.27	0.95***	0.83***	0.95***	0.82***
	(0.22)	(0.21)	(0.22)	(0.21)	(0.27)	(0.26)	(0.27)	(0.26)
Post matric	0.17	0.09	0.16	0.03	1.05***	0.91***	1.05***	0.90***
	(0.19)	(0.19)	(0.19)	(0.19)	(0.24)	(0.23)	(0.24)	(0.24)
Within coefficients:								
Income (W)	0.30***	0.30***	0.33***	0.32***	0.17	0.16	0.17	0.15
	(0.07)	(0.07)	(0.08)	(0.08)	(0.10)	(0.11)	(0.10)	(0.11)
Income sq. (W)		0.01		0.03		−0.02		−0.02
		(0.04)		(0.04)		(0.04)		(0.04)
Mother matric x income (W)			−0.37	−0.27			0.12	−0.24
			(0.24)	(0.25)			(0.65)	(0.41)
Mother matric x income sq. (W)				−0.07				0.30
				(0.09)				(0.35)
Employed (W)	−0.17	−0.17	−0.17	−0.16	0.02	0.02	0.01	0.02
	(0.15)	(0.15)	(0.15)	(0.15)	(0.14)	(0.14)	(0.14)	(0.14)
Age (W)	0.44***	0.43***	0.44***	0.43***	0.26***	0.26***	0.26***	0.26***
	(0.09)	(0.09)	(0.09)	(0.09)	(0.10)	(0.10)	(0.10)	(0.10)
Age sq. (W)	−0.00***	−0.00***	−0.00***	−0.00***	−0.00**	−0.00**	−0.00**	−0.00**
	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
Married/cohabiting (W)	0.21	0.21	0.20	0.21	0.12	0.12	0.12	0.11
	(0.20)	(0.20)	(0.20)	(0.20)	(0.23)	(0.23)	(0.23)	(0.23)
Between coefficients:								
Income (B)	1.51***	1.43***	1.58***	1.55***	1.08***	1.20***	1.09***	1.22***
	(0.23)	(0.16)	(0.24)	(0.16)	(0.22)	(0.19)	(0.22)	(0.20)
Income sq. (B)		0.18***		0.24***		0.28***		0.28***
		(0.04)		(0.04)		(0.05)		(0.05)
Mother matric x income (B)			−0.64	0.28			−0.02	−0.68
			(0.46)	(0.68)			(0.55)	(0.67)
Mother matric x income sq. (B)				−0.49*				0.40
				(0.27)				(0.47)
Employed (B)	0.71*	0.74*	0.68*	0.64*	0.36	0.30	0.36	0.30
	(0.39)	(0.39)	(0.39)	(0.39)	(0.30)	(0.29)	(0.30)	(0.29)
Age (B)	0.01	0.00	0.02	0.00	−0.24**	−0.26***	−0.24**	−0.26***
	(0.10)	(0.10)	(0.10)	(0.10)	(0.10)	(0.10)	(0.10)	(0.10)
Age sq. (B)	0.00	0.00	0.00	0.00	0.00***	0.00***	0.00***	0.00***
	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
Married/cohabiting (B)	2.21***	2.22***	2.22***	2.23***	2.01***	1.98***	2.01***	1.98***
	(0.33)	(0.32)	(0.33)	(0.32)	(0.30)	(0.30)	(0.30)	(0.30)
Constant	21.32***	21.39***	21.16***	21.23***	27.13***	27.36***	27.12***	27.34***
	(2.00)	(1.98)	(2.02)	(2.00)	(2.25)	(2.25)	(2.25)	(2.26)
var(Income (W))	0.10	0.11	0.09	0.10	0.47	0.42	0.47	0.42
var(Intercept)	17.85	17.68	17.80	17.48	12.00	11.75	12.00	11.75
var(Residual)	4.95	4.95	4.96	4.95	6.34	6.36	6.34	6.35
var(Income sq. (W))		0.00		0.00		0.00		0.00
Observations	8212	8212	8212	8212	6559	6559	6559	6559

* p <0.1, ** p <0.05, *** p <0.01. Standard errors in parentheses.

Note: Controls for race group, province and wave included but not displayed.

were only significant at the 10 percent level in the combined sample, and in the separate urban and rural regressions only the quadratic income interaction term was significant for urban women, and then only at the 10 percent level. The interaction remained significant for urban men, and as with mother having matric was not significant for rural men. However, the samples with father education available were slightly smaller than those for mother education and father's education is measured with greater error in South Africa (Von Fintel & Posel, 2016). These factors may explain the decreased significance of the interaction terms (particularly because larger sample sizes are required to detect interactions than to detect main effects).

Second, we use respondents' subjective assessment of their household's ranking on a six-step ladder, from poorest to richest, at age 15. This subjective assessment of childhood SES has been shown to be subject to substantial recall bias, being sensitive to both current income and subjective appraisals of one's current ranking (Von Fintel & Posel, 2016). It is nonetheless useful as a robustness check. For women, it is positively and significantly related to BMI (speculatively, this may be due to the anchoring effects documented by Von Fintel and Posel (2016), or because early childhood rather than adolescence may be a critical period in the development of obesity risk). However, the interaction between this measure and income is negative and significant for women, and likewise for squared income for men, echoing the pattern of the main results.

Third, we use adult height as a proxy for childhood SES. In the face of limited data on early childhood, adult height has commonly been used as a proxy for early childhood health and nutrition (see e.g. Currie & Vogl, 2013). Greater adult height is associated with lower BMI for women, and also for men at the 10 percent level. As in the main results, the interaction between income and height is also negative and significant for women, though it is not significant for men. This approach comes with the limitation that height may be related to BMI by construction: weight is divided by the square of height in the BMI formula, so it is difficult to tell whether the association between height and BMI is due to the formula penalising shorter people, or due to height picking up the effects of the nutritional environment in early childhood. Nonetheless, despite the limitations of all three additional measures of childhood SES, it is reassuring that they generally reflect the patterns seen using mother's education.

2.4.3.2 Alternative measures of adult SES

Using per capita household income as our primary measure of adult SES but mother's education as our measure of childhood SES means that there is a mismatch between our measures of SES in childhood and adulthood. Furthermore, African respondents in particular may be more likely to support extended family members who do not live under the same roof and therefore are not counted as part of the household. This implies that the standard of living implied by a given per capita household income may differ across race groups. As a robustness check, we therefore use three alternative measures of current SES.

First, we use respondents' own education. For men, the interactions with mother's education are not significant. For women, there is a significant negative interaction between one's mother having matric and having a post-matric qualification oneself. As in the main results using income, this suggests that the lower marginal effects of current SES for those from a high childhood SES background are primarily driven by those at high levels of current SES.

Second, we use an asset index constructed using multiple correspondence analysis. For both women and men, the interaction between high childhood SES and the asset index was negative and significant, again reflecting the pattern of the results using income.

Finally, we use respondents' subjective assessment of their household's current step on a six-step ladder, where households are ranked from poorest to richest. The interaction between this measure and childhood SES was negative and significant for men and women, though only at the 10 percent level for women. Using subjective step at 15 as the measure of childhood SES and subjective step today as the measure of adult SES, the interaction is negative and significant for women but not for men.

Overall, these results suggest that the patterns observed in the main results are robust to using alternative measures of childhood and adult SES.

2.4.3.3 Other robustness checks and analyses

As a robustness check, we restricted the sample to African and coloured[23] respondents (Appendix A.8). Whites and Asians/Indians represent a relatively small subset of the population and of the sample, but may have distinct patterns. Wittenberg (2013) shows that the social gradient in body weight has already reversed for white women, suggesting that this group is already at the next stage of the obesity transition. We therefore investigated whether the main results held when excluding white and Indian respondents. Furthermore, these two groups are historically and persistently more affluent on average, so excluding them isolates the effects for groups that may be more likely to go through economic and health transitions with rising living standards. Results were largely similar to those for the full sample, but with larger p-values. For urban women, high childhood SES was still significantly negatively associated with BMI. For women, the interaction between mother having matric and household income remained negative but was only significant in the combined urban and rural sample, and then only at the 10 percent level. Neither childhood SES nor the interaction with household income were significant for African and coloured men. Again, this may at least partly be explained by the smaller number of observations available for the African and coloured subsample, particularly for those with mothers who completed matric.

Finally, Tables A.9.1 and A.9.2 explore whether the coefficient on childhood SES is attenuated by controlling for current SES and health behaviours. Controlling for current SES makes the childhood SES coefficient markedly more negative, reflecting the positive association between childhood SES and adult SES. Failing to control for adult SES generally results in insignificant estimates of the association between childhood SES and adult BMI, because the coefficient on childhood SES then picks up some of the positive association between adult SES and BMI. Controlling for smoking and an indicator for exercising weekly makes little difference to the coefficient on childhood SES or its interaction with household income. This suggests that these adult health behaviours are not significant transmission mechanisms for the association between childhood SES and adult body weight[24].

[23]NIDS respondents were asked to self-identify their race group as one of 'African', 'coloured', 'Asian/Indian', 'white' or 'other'. These are official South African classifications still used in many surveys and government statistics, based on the system of racial classification under the apartheid regime. The use of these categories acknowledges the lingering effects of the division of South African society, including economic resources and opportunities, along racial lines, and is not an endorsement of the legitimacy of these categories.

[24]We cannot control for adult diet or eating behaviours, which may be transmission mechanisms of the childhood SES-adult BMI association, using the NIDS data. The NIDS only included questions on food consumption in the first wave, and these questions tended to be poorly answered.

Table 2.9: Random effects within-between models for combined urban and rural samples, women

	(1)	(2)	(3)	(4)	(5)
Fixed coefficients					
Mother matric	−1.46**	−1.35**	0.79	0.42	2.31*
	(0.62)	(0.61)	(0.82)	(0.78)	(1.34)
Within coefficients:					
Income (W)	0.01	0.01	−0.00	−0.00	−0.03
	(0.06)	(0.06)	(0.06)	(0.07)	(0.07)
Income sq. (W)		−0.02		−0.01	
		(0.03)		(0.04)	
Urban (W)					0.29
					(0.28)
Mother matric x income (W)			0.20	0.66	
			(0.32)	(0.41)	
Mother matric x income sq. (W)				−0.19	
				(0.13)	
Mother matric x urban (W)					−1.83**
					(0.81)
Employed (W)	−0.08	−0.09	−0.08	−0.09	−0.07
	(0.09)	(0.09)	(0.09)	(0.09)	(0.10)
Age (W)	0.57***	0.57***	0.57***	0.57***	0.56***
	(0.08)	(0.08)	(0.08)	(0.08)	(0.08)
Age sq. (W)	−0.01***	−0.01***	−0.01***	−0.01***	−0.01***
	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
Married/cohabiting (W)	0.39***	0.39***	0.39***	0.39***	0.39***
	(0.14)	(0.14)	(0.14)	(0.14)	(0.14)
Between coefficients:					
Income (B)	1.18***	1.19***	1.32***	1.32***	1.15***
	(0.17)	(0.19)	(0.18)	(0.18)	(0.17)
Income sq. (B)		−0.09		−0.02	
		(0.09)		(0.08)	
Urban (B)					0.80***
					(0.29)
Mother matric x income (B)			−1.75***	0.63	
			(0.47)	(0.80)	
Mother matric x income sq. (B)				−0.90***	
				(0.27)	
Mother matric x urban (B)					−4.60***
					(1.44)
Employed (B)	−0.43	−0.41	−0.39	−0.41	−0.44
	(0.34)	(0.34)	(0.34)	(0.34)	(0.34)
Age (B)	0.59***	0.60***	0.59***	0.59***	0.60***
	(0.10)	(0.10)	(0.10)	(0.10)	(0.10)
Age sq. (B)	−0.01***	−0.01***	−0.01***	−0.01***	−0.01***
	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
Married/cohabiting (B)	0.92***	0.94***	0.95***	0.99***	0.94***
	(0.31)	(0.31)	(0.31)	(0.31)	(0.30)
Constant	14.93***	14.81***	14.68***	14.69***	14.15***
	(2.15)	(2.16)	(2.16)	(2.15)	(2.12)
var(Income (W))	0.87	0.85	0.86	0.85	
var(Intercept)	39.06	39.00	38.83	38.73	34.82
var(Residual)	9.04	9.04	9.04	9.04	9.27
var(Income sq. (W))		0.00		0.00	
var(Urban (within))					6.21
Observations	21889	21889	21889	21889	21889

* p <0.1, ** p <0.05, *** p <0.01. Standard errors in parentheses.

Note: Controls for race group, province and wave included but not displayed.

Table 2.10: Random effects within-between models for combined urban and rural samples, men

	(1)	(2)	(3)	(4)	(5)
Fixed coefficients					
Mother matric	−0.46	−0.68*	0.34	0.52	0.93
	(0.37)	(0.37)	(0.47)	(0.41)	(0.58)
Within coefficients:					
Income (W)	0.27***	0.27***	0.28***	0.28***	0.28***
	(0.06)	(0.06)	(0.06)	(0.06)	(0.06)
Income sq. (W)		0.01		0.02	
		(0.03)		(0.03)	
Urban (W)					−0.07
					(0.22)
Mother matric x income (W)			−0.25	−0.26	
			(0.22)	(0.22)	
Mother matric x income sq. (W)				−0.02	
				(0.08)	
Mother matric x urban (W)					1.66*
					(0.91)
Employed (W)	−0.10	−0.09	−0.09	−0.09	−0.08
	(0.10)	(0.10)	(0.10)	(0.10)	(0.10)
Age (W)	0.39***	0.38***	0.39***	0.38***	0.39***
	(0.07)	(0.07)	(0.07)	(0.07)	(0.07)
Age sq. (W)	−0.00***	−0.00***	−0.00***	−0.00***	−0.00***
	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
Married/cohabiting (W)	0.19	0.19	0.19	0.19	0.18
	(0.15)	(0.15)	(0.15)	(0.15)	(0.15)
Between coefficients:					
Income (B)	1.42***	1.39***	1.48***	1.50***	1.39***
	(0.17)	(0.13)	(0.17)	(0.13)	(0.17)
Income sq. (B)		0.20***		0.25***	
		(0.04)		(0.04)	
Urban (B)					0.30
					(0.22)
Mother matric x income (B)			−0.64	−0.06	
			(0.40)	(0.55)	
Mother matric x income sq. (B)				−0.42*	
				(0.25)	
Mother matric x urban (B)					−1.66**
					(0.72)
Employed (B)	0.54**	0.55**	0.52*	0.48*	0.52*
	(0.27)	(0.27)	(0.28)	(0.27)	(0.27)
Age (B)	−0.07	−0.09	−0.07	−0.08	−0.08
	(0.07)	(0.07)	(0.08)	(0.07)	(0.07)
Age sq. (B)	0.00	0.00*	0.00	0.00*	0.00
	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
Married/cohabiting (B)	2.10***	2.10***	2.11***	2.11***	2.09***
	(0.24)	(0.24)	(0.24)	(0.24)	(0.24)
Constant	23.31***	23.46***	23.17***	23.29***	23.35***
	(1.57)	(1.56)	(1.59)	(1.58)	(1.57)
var(Income (W))	0.28	0.29	0.27	0.29	
var(Intercept)	16.10	15.90	16.06	15.74	14.16
var(Residual)	5.47	5.47	5.48	5.47	5.53
var(Income sq. (W))		0.00		0.00	
var(Urban (within))					2.72
Observations	14771	14771	14771	14771	14771

* p <0.1, ** p <0.05, *** p <0.01. Standard errors in parentheses.

Note: Controls for race group, province and wave included but not displayed.

2.5 Discussion

For both women and men we find that high childhood SES, measured by the respondent's mother having matric, is associated with lower BMI in urban areas, but not in rural areas. This is in contrast to the generally positive association between adult SES and BMI observed in the SA literature and in this study, but is in line with findings on childhood SES in the international literature. The finding that higher childhood SES is associated with lower body weight in women concurs with those from a range of studies from both developed (González et al., 2009; Senese et al., 2009) and developing countries (Aitsi-Selmi et al., 2013; Wagner et al., 2018), including South Africa (Case & Menendez, 2009), though some studies have found no significant association for women (Addo et al., 2009; Ginsburg et al., 2013). For men, some developed country studies have found that higher childhood SES is associated with lower body weight, as in this study, but results have been less consistent than those for women, with many finding no association (González et al., 2009; Senese et al., 2009). In contrast to this study, the handful of developing country studies have found a positive (Addo et al., 2009; Aitsi-Selmi et al., 2013; Wagner et al., 2018) or no (Case & Menendez, 2009) relationship between higher childhood SES and body weight in men. Our finding that higher childhood SES is associated with lower body weight in urban men is thus unusual in both the South African and wider developing country context, though the developing country literature is relatively limited. Another unique aspect of our results is that the association between higher childhood SES and lower adult obesity risk was observed in a context where higher adult SES is associated with higher obesity risk – in a country that remains in stage 2 of the obesity transition. Almost all other studies of which we are aware were conducted in contexts already showing characteristics of stage 3 of the obesity transition, where higher adult SES was associated with lower obesity risk[25], and the associations between childhood and adult SES and body weight were thus in the same direction. It is thus noteworthy that we find an association between higher childhood SES and lower body weight even in a context where adult SES has the opposite association. However, the association between high childhood SES and lower adult obesity risk is driven by those who are likely to be at a more advanced stage of the nutrition transition: urban residents and those with high incomes.

For both men and women we found a negative interaction between childhood SES and adult household income: high childhood SES seems to have a protective effect against high BMI for those with

[25] One exception is the study by Addo et al. (2009) in Ghana, where the associations between both adult and pre-adult wealth and body weight were positive for men, but these associations still had the same sign.

high adult income, but not for those with low adult income. Put differently, among individuals with high adult income, predicted BMI is significantly higher for those from a low childhood SES background. This suggests that upward social mobility may result in an increased obesity risk relative to those who had high SES in childhood and adulthood. These results concur with those of several high-income country studies (Chung et al., 2020; Heraclides & Brunner, 2010; Savitsky et al., 2017; Savitsky et al., 2021), but of these only the Heraclides and Brunner (2010) study conducted separate analyses on men and women. A meta-analysis by Vieira et al. (2019) also finds that for women upward mobility was associated with a higher BMI compared to those who maintained a high SES throughout life, but in contrast to our study finds no significant association for men. In the South African context, our results contrast with those of Case and Menendez (2009), who find no significant interaction between childhood hunger and adult household income in explaining the probability of being obese. This difference may be because the Case and Menendez (2009) study was conducted on a sample in a poor urban suburb of Cape Town, and in our study the interaction was driven by individuals with high levels of income, who may have been underrepresented in the Case and Menendez (2009) study. In the only other South African study on social mobility and BMI of which we are aware, Ginsburg et al. (2013) find that upward social mobility between birth and 15 years of age, compared to those who did not change SES, was associated with higher BMI and obesity among females, but not among males. However, the study by Ginsburg et al. (2013) controls for SES at birth but not adult SES, which could conflate upward social mobility with the effects of having a higher adult SES. Their specification of social mobility is not directly comparable to ours, which controls for adult SES; outcomes at age 15 are also not necessarily comparable with those for adults. Our finding that upward mobility from low childhood SES to high adult SES is associated with increased obesity risk compared to remaining high SES throughout life is thus unique in the South African context.

Our results support the critical/sensitive periods and social mobility models of life course SES and obesity risk. The significant association between childhood SES and BMI remains after controlling for adult SES (indeed, childhood and adult SES work in opposite directions, and the association is only consistently present when controlling for adult SES) and health behaviours, suggesting that childhood, broadly speaking, is a sensitive period in the development of obesity. But the measure of childhood SES used in this study is only a proxy for SES in childhood as a whole, so this study could not assess effects of SES at various points in the life course to investigate whether specific points in childhood or adolescence are critical or sensitive periods in the development of obesity. As discussed previously, our results suggest that among high SES individuals, those who came from a lower SES background are at higher risk of obesity, lending support to the social mobility model. This study does not directly assess the accumulation model. However, our results suggest that though childhood SES and adult SES work in opposite directions, high childhood SES in combination with higher adult SES is associated with lower obesity risk, which could be interpreted as evidence in favour of the accumulation model.

We found that high childhood SES was associated with lower BMI in urban but not in rural areas. This may be partly due to small sample sizes of respondents whose mothers had matric in rural areas, but may also be because the nutrition transition is more advanced in urban areas, yielding greater access to energy-dense processed foods and greater exposure to obesogenic environments in urban areas. The protective effect of higher childhood SES in urban but not rural areas, together with the protective effect of higher childhood SES at high levels of adult income, suggest that environmental mismatch may play a role in obesity in South Africa. Both of these factors suggest that high childhood SES has a protective effect against obesity when adults are faced with an environment of energy abundance, but not one of scarcity. Living in an urban area and having a higher income may both enable greater access to obesogenic environments, processed foods and fast foods. The mismatch hypothesis predicts that an environment of nutritional scarcity in early childhood followed by one of energy abundance in adulthood makes the development of obesity more likely (Gluckman & Hanson, 2008). In light of this hypothesis it would make sense that a higher childhood SES has the most protective effect against obesity for those facing the most obesogenic environments in adulthood – for those who face the greatest potential mismatch between their childhood and adulthood environments. Those who face an environment of abundant energy in early childhood are more likely to develop in such a way that their metabolism is better able to cope with an environment of abundant energy in adulthood. This links to our finding that the estimated marginal effect of adult income was positive for those with low childhood SES, but negative and insignificantly different from zero for those with high childhood SES: additional income was not associated with a higher BMI among those who faced a more favourable childhood background. In the context of a country in stage 2 of the obesity transition, higher adult SES yields greater access to energy-dense processed foods and obesogenic environments, but high childhood SES may help people to be less vulnerable to their obesogenic effects.

Our findings shed light on the possible drivers of the shift from stage 2 to stage 3 of the obesity transition, involving the shift of the social gradient in body weight from positive to negative. The insignificant negative marginal effect of adult income on BMI for women with high childhood SES suggests that the social gradient in BMI is already on the verge of reversing for this group. Though still positive, the marginal effect of adult income is also significantly lower for men with high childhood SES than men with low childhood SES. Furthermore, the social gradient already appears to have reversed among women who experienced a high SES in both childhood and adulthood. This suggests that favourable childhood conditions sustained into adulthood – or alternatively, high SES sustained over more than one generation – are necessary before we see a reversal of the gradient. It may take several generations of adequate childhood nutrition before we see higher SES have a protective effect against obesity. Speculatively, this may also be a precondition for a shift to the hypothetical fourth stage of the obesity transition, where obesity rates start to decline.

The social gradient in BMI, as well as the flatter social gradient in body weight among those with high childhood SES, is driven more by differences in income between individuals than by short-run changes in income within individuals. This may be because both BMI and SES are slow-changing, and social mobility is a long-run process. This suggests that body weight may be more influenced by social mobility over the entire life course than by very short-run mobility over the course of adulthood. This underscores the long-run nature of the obesity transition, and the idea that changes between generations and between childhood and adulthood may help to drive the obesity transition. However, a limitation of this analysis is that only nine years separate the first wave of NIDS from the last wave, which may not be sufficient time to see effects of income changes within individuals. Furthermore, a relatively small proportion of respondents had complete data in all waves, or even complete data for both the first wave and the final wave. Much of the within-individual change in income is thus estimated using consecutive waves, or waves separated by only a few years, rather than the full nine years between waves 1 and 5. The relatively small sample sizes available for estimating within-individual income changes may also have played a role.

This study is the first of which we are aware to explicitly link childhood SES and social mobility to the nutrition and obesity transitions, and specifically to propose that these factors may contribute to the process of reversal of the (adult) social gradient in obesity. It also provides valuable evidence regarding the association between childhood SES, social mobility and BMI in the context of a developing country undergoing the nutrition and obesity transitions. It uses a large nationally representative dataset, and is unique in its exploration of the social gradient in BMI within versus between individuals. However, it is subject to several limitations. First, our measure of childhood SES, maternal education, is based on respondents' reports of their mother's education. Many respondents may not know their mother's education level, or may not recall it accurately, and so this measure is subject to measurement error. While respondent's reports were replaced with the mother's own report of her education where the mother was also present in the NIDS sample, for most respondents this was not available. It would also be useful for future studies to explore these relationships using more nuanced measures of childhood SES, rather than relying on a single binary indicator. Secondly, the subsample sizes of respondents with mothers who had matric were relatively small, particularly in rural areas. This was particularly a problem when assessing whether the relationships held in the African and coloured subsample, though indications are that the main results held for women. Lastly, this study is not able to establish whether the relationship between mother's education and BMI is causal. It is possible that genetic factors may play a role; we were not able to assess this possibility. Better educated mothers may have unobserved characteristics that do not represent SES and are related to their children's BMI in some way, which could bias estimates of the effect of mother's education. Childhood SES is very difficult to manipulate experimentally, and valid quasi-experimental estimates are hard to come by. Nonetheless, future research establishing a causal relationship between childhood SES, social mobility and BMI would be valuable. Research using more refined measures of childhood SES would also be useful, as would studies using SES measures at various points in childhood to isolate critical or sensitive periods in the development of obesity in South Africa. Studies with measures of SES at multiple points in the life course would also be useful to assess the accumulation model, and to explore at what point in the life course higher SES stops being protective against obesity and becomes a risk factor in South Africa.

2.6 Conclusion

This chapter suggests that childhood SES and intergenerational mobility may contribute to the reversal of the social gradient in body weight and the shift to later stages of the obesity transition. Upward social mobility is associated with increased obesity risk in adulthood compared to individuals who had a high SES throughout life, while high childhood SES is associated with lower obesity risk in the face of obesogenic environments. The social gradient in body weight is closer to reversing among individuals from a high SES childhood background, and already appears to have reversed among high-SES women who also experienced a high SES in childhood. This suggests that it may take more than one generation of sustained high SES – or alternatively perhaps of adequate childhood nutrition – before higher adult SES starts to be associated with lower obesity risk, and a corresponding levelling off in obesity rates. The shift to the hypothetical fourth stage of the obesity transition, where obesity rates eventually start to decline, may require the foundation of favourable childhood conditions. Looking ahead, as long as South Africa still has problems of widespread childhood undernutrition, coupled with improving living standards and increasing exposure to obesogenic environments, we can expect obesity rates to continue to rise, particularly among those with low childhood SES. As Big Food companies expand further into rural areas, we can expect rising obesity rates in rural areas too. As the nutrition transition advances in rural areas, better childhood environments may help to protect against future rises in obesity rates among adults. If indeed childhood nutrition is behind the relationship we have observed between maternal education and adult obesity risk (this is plausible but not certain), this adds further evidence to the already large body of evidence that good childhood nutrition is crucial for many elements of health, success and wellbeing in adulthood, and adds even greater urgency to calls to reduce childhood undernutrition in South Africa.

CHAPTER 3

DO GENERATIONAL SHIFTS DRIVE THE OBESITY TRANSITION?

3.1 Introduction

As more countries move through the nutrition transition (a shift from starchy low-fat traditional diets to diets high in fat, sugar and processed foods), the burden of obesity is increasing among developing countries, and shifting increasingly to poor individuals within countries. Obesity rates in developing countries have grown faster than those in developed countries (Popkin & Gordon-Larsen, 2004). Historically, high consumption of fat has been a hallmark of rich societies, and of rich individuals within societies. However, the availability of cheap vegetable oils has weakened the relationship between income and the proportion of fat in diets, increasingly making higher fat consumption available even at low income levels (Drewnowski & Popkin, 1997). The relative prices of other energy-dense processed foods, such as those high in sugar, have also fallen in recent decades (Cawley, 2015; Monsivais et al., 2010), making them available even to people with low income.

Within countries, the relationship between socioeconomic status (SES) and obesity tends to shift from positive to negative as countries develop (Monteiro et al., 2004b). Rich individuals within low-income countries face a higher risk of obesity, while poor individuals within high-income countries tend to experience higher obesity risk. These shifting socioeconomic patterns of obesity have been described as the 'obesity transition', consisting of four stages (Jaacks et al., 2019). In stage 1, obesity is more prevalent in those with high SES, women, and adults. In stage 2, obesity prevalence increases, while the gap between women and men narrows, as do the socioeconomic differentials in obesity prevalence among women. The shift from a positive to a negative socioeconomic gradient in obesity occurs in stage 3, while obesity plateaus among children and high SES women. The theoretical fourth stage involves a decline in obesity prevalence, but there is little evidence of this yet having taken place in any country.

This paper raises the possibility that the obesity transition may not only be a 'macro' tendency that occurs across time and countries' levels of economic development, but can have different 'micro' trajectories across different generations. Examining the social gradient in body weight for an entire population at any one point in time may obscure generational differences in this relationship. As countries develop, they move from stages 1 and 2 of the obesity transition to stage 3. However, within countries some cohorts may remain in an earlier stage, while others may already be in stage 3 or even stage 4. We raise the possibility that the phases of the obesity transition can coexist, but for different generations. Younger generations may already have experienced a shift in the social gradient in body weight or be in the midst of this shift, but are grouped together with older generations, which may be slower to adopt new body size ideals or not adopt them at all. In some countries the shift to the next phase of the obesity transition may thus already be occurring among younger generations, without being observed in aggregate.

Assessing whether there are differences in the SES gradient in body mass index (BMI) and obesity across generations requires us to distinguish between age, period and cohort effects. Age effects in obesity capture the effects of physiological changes as well as other changes across the life course, for example in social roles. Period effects capture fluctuations or trends in obesity across time that affect all age groups and cohorts, such as long-run aggregate economic development and changes in social norms (though this may also be a cohort effect). Cohort effects capture factors affecting the body weight of those born at the same point in time, for example exposure to certain social, economic or nutritional conditions at a particular episode in history, or social norms common to certain generations.

There are several reasons why the relationship between SES and BMI may be different for younger people. First, the SES-BMI relationship may differ across the life course. Social gradients in many health outcomes tend to widen with age (e.g. Currie & Stabile, 2003), possibly reflecting cumulative effects of socioeconomic circumstances across the life course. Alternatively, health concerns may become more salient with age, as the risk of non-communicable diseases (NCDs) increases with age. Higher SES individuals may be better able to make investments to improve their health, and may be more likely to do so at older ages. In the US, Baum and Ruhm (2009) find that social gradients in obesity also widened with age. Among urban civil servants in Ghana, Addo et al. (2009) find that higher asset wealth was positively associated with obesity for women aged 45 and older, but was associated with lower obesity risk for women under 45, though these differences were generally not significant.

Second, the SES-BMI relationship may differ for more recent *cohorts*. Age effects are changes that occur over the life course for all cohorts, while cohort effects are more long-run and permanent patterns affecting members of particular generations (or individuals born in specific years). Cohort effects do not necessarily change as individuals age. The socioeconomic gradient in body weight may change for members of all generations as they age (an age effect), or alternatively may differ for younger generations even as they age (a cohort effect). In line with the 'foetal origins' hypothesis of Barker and colleagues (Hales & Barker, 1992), nutritional environments in early life may have lasting effects on obesity risk, and certain points in the life course may be sensitive periods in the development of obesity. Nutritional environments have changed over time as the nutrition transition has advanced, with expanding availability of fast foods and other energy-dense processed foods. This implies that younger cohorts may have been more exposed to obesogenic environments in childhood or at other sensitive periods in the life course, with potentially lasting effects on the SES-BMI relationship for these cohorts. Furthermore, the SES-BMI relationship may be related to body size ideals, and these may change slowly. It is possible that body size ideals change more across generations than within generations, with body size preferences being relatively fixed once people reach adulthood, but with younger generations adopting different body size preferences to their predecessors. We hypothesise that younger generations may be first to adopt a thinner ideal body size, and so may be the first to experience a shift in the relationship between SES and obesity. In South Africa, while there is evidence that many people prefer a larger body size, particularly for women (Draper et al., 2015), there is also some evidence of a preference for normal weight or thin body shapes rather than obesity among adolescents (Gitau et al., 2014).

There has been very limited exploration of potential age, time and generational or cohort differences in the SES-BMI relationship in the literature to date. Several high-income country studies have investigated age, period and cohort effects on BMI or obesity, but very few have examined these effects with regard to the social gradient in BMI. While most studies find increasing obesity rates over time (period effects), several have also found increased BMI or risk of obesity in more recent cohorts, and that the onset of obesity occurs at increasingly younger ages (Clarke et al., 2009; Dobson et al., 2020; Reither et al., 2009; Robinson et al., 2013; Yang et al., 2021). In the UK, the social gradient in body weight neither widened nor narrowed between the early 1990s and 2000s (White et al., 2007). A handful of studies have found evidence of a widening of SES gradients in BMI with age (until middle age, after which the gaps tend to decline) (Baum & Ruhm, 2009; Clarke et al., 2009; Yang et al., 2021), and Yang et al. (2021) additionally found that educational gradients in BMI have widened with successive cohorts in the US, particularly among women. Some studies have also found a widening of educational (Lynch, 2003) or sex and race (Yang & Lee, 2009) disparities in other health outcomes in more recent cohorts. In contrast, recent evidence suggests that educational gradients in BMI in Indonesia have flattened in more recent cohorts, possibly driven by increases in BMI among the less educated (Liwin, 2022).

Most of the handful of studies considering age and cohort differences in the SES-BMI relationship come from high-income countries that are already in stage 3 of the obesity transition. It is particularly interesting to investigate these differences in countries that are still in stage 2 of the obesity transition and possibly on the cusp of entering stage 3, to explore whether cohort shifts are part of what pushes countries into stage 3 of the transition. South Africa remains in stage 2 of the obesity transition (Jaacks et al., 2019). The shift from a positive to a negative relationship between SES and BMI has been estimated to occur at a gross national product (GNP) per capita of around US$2500 (Monteiro et al., 2004b), and the burden of obesity already appears to have begun to shift towards the poor in some other upper middle-income developing countries such as Brazil and Mexico. In South Africa, however, it is puzzling that the SES-BMI association does not yet appear to have begun to reverse, except among white women (Wittenberg, 2013). A number of studies have shown a positive association between SES and obesity in South Africa among both women and men (Alaba & Chola, 2014; Ardington & Case, 2009; Ardington & Gasealahwe, 2012; Sartorius et al., 2015; Wittenberg, 2013). Indeed, Wittenberg (2013) argues that BMI is positively related to SES to the point that BMI can be considered a measure of economic wellbeing in South Africa. In most cases the relationship is stronger for men, though Case and Menendez (2009) find a relationship for women and no relationship for men. Alaba and Chola (2014), on the other hand, find a positive socioeconomic gradient in obesity for men, but find that the relationship among women is positive but fairly flat. Because South Africa is approaching a new phase of the obesity transition, it is a particularly interesting context in which to study differences between cohort and life cycle aspects of the social gradient in BMI. The context allows for a better understanding of *how* societies move from one phase of the obesity transition to another.

This paper explores whether there is a generational aspect to the obesity transition in South Africa using the longitudinal National Income Dynamics Study (NIDS) data. It asks whether there are any signs of generational differences in the relationship between SES and BMI in South Africa, hypothesising that we may see a flattening of the positive SES gradient in BMI with successive cohorts, in line with recent findings from Indonesia (Liwin, 2022). Uniquely, we use a machine learning algorithm to detect structural breaks in the association between SES and BMI by birth year. We also explore differences in the SES-BMI gradient by age to assess whether the health risks posed by socioeconomic advantage accumulate over the life course, as those posed by disadvantage have been observed to do.

3.2 Data

We use data from all five waves of the nationally representative NIDS, collected in 2008, 2010-11, 2012, 2014-15 and 2017 (Southern Africa Labour and Development Research Unit, 2018). Using data from all five waves of NIDS helps to reduce the collinearity between age and birth year by providing measurements on individuals born in a given year at several ages.

NIDS includes measures of the height, weight and waist circumference of adult respondents. The primary measure of adiposity used in this study is BMI, which is defined as weight in kilograms divided by the square of height in metres. The procedure used to clean the height and weight data is described in the appendix to Chapter 2. To reduce the influence of individuals with extreme BMIs, BMI is winsorised at the first and 99th percentiles, for males and females separately. We also use a binary indicator for being obese as a robustness check[1]. Obesity is defined as having a BMI of 30 or above.

NIDS also includes data on respondents' age, birth year and a range of variables related to socioeconomic status (SES). We use household income as our measure of SES. Household income is deflated to March 2017 values using the deflator files provided with NIDS, and then divided by the square root of household size to adjust for economies of scale within the household (OECD, 2013). We then apply the inverse hyperbolic sine transformation, which is similar to a logarithmic transformation, but allows for the inclusion of zero values (Bellemare & Wichman, 2020).

The sample for this analysis is limited to adults aged 25-64. We use 25 as the lower age limit because increases in height are possible even in the early twenties (Hulanicka & Kotlarz, 2009), which causes issues for the procedure we use to clean the anthropometric data, as it relies on checking consistency in height across waves. We use 64 as the upper limit because the sample sizes of older adults are relatively small, and because height starts to decline later in life (Cline et al.,

[1]We use linear probability models for obesity.

1989; MedlinePlus, n.d.), which also causes problems for cleaning the BMI data.

3.3 Methods

We explore differences in SES gradients by cohort, age and wave using a series of regressions. In line with most of the literature (see e.g. Case & Menendez, 2009; Wittenberg, 2013), we estimate all regressions separately for men and women, as the determinants and correlates of obesity differ for men and women (Case & Menendez, 2009). Mean BMI, as well as the associations between BMI and other variables of interest such as age and income, differ markedly by gender (see Figures 3.1 and 3.2). In addition to household income and the age, cohort and wave variables, we control for a series of other socioeconomic and demographic variables that may be associated with BMI and income: race group (reference group African)[2], education, an indicator for being employed, an indicator for being married or cohabiting, and province. Education is categorised into less than matric (Grade 12, the final year of schooling), matric, and post matric, with less than matric as the reference category. We also include indicators for being a smoker and for exercising weekly. For women we include an indicator for ever having given birth. We estimate some regressions separately for urban and rural areas, but where we do not we also include an indicator for residing in an urban area.

3.3.1 Model-based recursive partitioning

Our main question is whether there are differences in the SES gradient in BMI in younger versus older cohorts. Birth year is highly correlated with age, but not perfectly because each birth year cohort is observed at different stages, either before or after their birthdays, across the five waves of NIDS. We search for a structural break in the relationship between SES and BMI by birth year. However, it is not clear where to split birth year to divide the sample into younger and older cohorts. Instead of choosing an arbitrary split point, we use the model-based recursive partitioning machine learning algorithm, developed by Zeileis et al. (2008), to detect structural breaks in the SES-BMI

[2]NIDS respondents were asked to self-identify their race group as one of 'African', 'coloured', 'Asian/Indian', 'white' or 'other'. These are official South African classifications still used in many surveys and government statistics, based on the system of racial classification under the apartheid regime. We control for race because South African society is still heavily affected by its racially segregated history, and race is still strongly associated with income and other socioeconomic variables. Controlling for race thus implicitly controls for other factors, such as unequal access to services and amenities, that may be correlated with both SES and BMI. Controlling for race does not imply an endorsement of the legitimacy of these categories.

relationship by birth year[3].

The model-based recursive partitioning algorithm fits a parametric model to a dataset and tests for parameter instability in that model across a set of partitioning variables. If overall parameter instability is detected, it splits the model by the variable associated with the highest instability, choosing the split point that minimises the objective function – in the linear case, the sum of squared residuals (Hothorn & Zeileis, 2015; Zeileis & Hothorn, 2015). This procedure is repeated in each of the nodes created by this split, stopping when no more significant instability is found (Zeileis et al., 2008). The result is a form of decision tree with a separate parametric model estimated in each node (instead of a predicted value or binary outcome as in conventional decision trees based on recursive partitioning). In this way we establish separate relationships between SES and BMI for different cohorts.

We implement the model-based recursive partitioning algorithm using the *partykit* package in R (Hothorn & Zeileis, 2015). The parametric model fit in each node is BMI regressed on income and age. Income and age are both centred at their sample means. Gender and birth year are used as splitting variables. In all cases the first split (associated with the highest parameter instability) is by gender, and the second by birth year. We control for age in the parametric models in each node because age and BMI are strongly correlated – BMI increases with age, before levelling out for women in the early fifties, as shown in Figure 3.1 – as are age and income, and we want to detect instability in the SES-BMI association independent of age. We impose a parametric restriction on age, constraining it to have a linear functional form, as opposed to a fully flexible specification of age dummies. This assumption is restrictive, but chosen for the sake of tractability and to ensure that the algorithm focuses on finding cohort instability. However, the algorithm detects splits according to overall parameter instability in the model, which means that the age parameter contributes to overall parameter instability, even though we are interested in instability in the income parameter. As a robustness check, we therefore also run the algorithm without controlling for age (i.e. assuming the coefficient on age is zero, so that all instability is attributed to cohort). Results are available in Appendix B.1. To allow for possible differences between urban and rural areas due to the nutrition transition being more advanced in urban areas, we run the algorithm for the full sample and for

[3]McKenzie (2006) proposed a method to identify structural breaks in age-period-cohort models, but these methods are not appropriate for our question, which involves searching for structural breaks in the relationship between two variables, rather than in an outcome. For example, the method developed by McKenzie (2006) would be appropriate for identifying age, period and cohort effects in BMI or in obesity prevalence, but not in the SES-BMI relationship.

urban and rural areas separately.

Failing to account for clustering of repeated observations on the same individual in a panel dataset could cause the parameter instability tests to over-reject, leading to spurious splits in the tree. The standard errors are therefore clustered by individual (using the 'cluster' option in the *partykit::mob* function). The model is also weighted using the post-stratified survey weights (using the 'weights' option). The significance level for the parameter instability tests is set at 0.05 (the default). The model-based recursive partitioning algorithm will continue growing the tree until no more significant splits can be detected. However, given a fairly large sample this could result in a very large tree. For simplicity[4] we limit the depth of the tree to the first birth year split (by setting the 'maxdepth' argument to 3)[5].

3.3.2 Interactions between SES and age, wave and cohort

We use the model-based recursive partitioning algorithm as a guide to detect structural breaks in the SES-BMI relationship by birth year. Because the algorithm detects overall parameter instability rather than instability in a specific parameter, we could not control for any other variables without them also contributing to parameter instability. The intercept is also a parameter and can contribute to instability, meaning that average BMI changes significantly with birth year, regardless of any differences in the SES-BMI relationship. Even in a model not controlling for any other variables, a significant birth year split detected by the algorithm may not be significant when interacted with SES in a linear regression. We therefore take the splits identified by the algorithm and test them in a statistical model, interacting an indicator for each split with our SES variable in a linear regression model. We explore whether SES gradients vary with age, cohort and wave by interacting these variables with our SES measure in a series of regressions. We estimate the following regressions:

$$BMI_{it} = \beta_0 + \beta_1 SES_{it} + \beta_2 Age_{it} + \beta_3 SES_{it} \times Age_{it} + \beta_4 Birthyear_i + \beta_5 Wave_t + \beta_6 \boldsymbol{X_{it}} + \epsilon_{it} \quad (3.1)$$

[4]Initial models included further splits, but in some cases resulted in trees with single birth years in a node, leading to concerns that outliers were driving the results.

[5]It is usual practice in machine learning to test the performance of an algorithm on unseen data, i.e. to divide the data into training and test or validation datasets, and train the model on the training dataset and test it on the unseen test data to prevent overfitting. However, our goal is not prediction, but rather to use the algorithm as an intermediate step as an input into a regression model. We therefore do not go through the usual steps of splitting the data into training and test splits or of cross-validation.

$$BMI_{it} = \beta_0 + \beta_1 SES_{it} + \beta_2 Birthyear_i + \beta_3 SES_{it} \times Birthyear_i + \beta_4 Age_{it} + \beta_5 Wave_t + \beta_6 \boldsymbol{X_{it}} + \epsilon_{it} \tag{3.2}$$

$$BMI_{it} = \beta_0 + \beta_1 SES_{it} + \beta_2 Wave_t + \beta_3 SES_{it} \times Wave_t + \beta_4 Age_{it} + \beta_5 Birthyear_i + \beta_6 \boldsymbol{X_{it}} + \epsilon_{it} \tag{3.3}$$

Here SES_{it} represents household income for individual i in wave t, Age_{it} represents the respondent's age in wave t, $Wave_t$ is an indicator for the survey wave, and $Birthyear_i$ is an indicator for being born after the birth year split identified by the algorithm. $\boldsymbol{X_{it}}$ is a vector of covariates as described above. In each case β_3 is the main coefficient of interest, reflecting how the SES coefficient varies by age, birth cohort and wave. For simplicity we interact linear age with SES, although the relationship between age and BMI appears to be quadratic for women. Results for a quadratic age specification are available in Appendix B.4, but do not differ markedly from the results using a linear age specification. We also use a linear specification for the SES variable, even though there are signs of non-linearities in the SES-BMI association for men and women (see Figure 3.2), and the other chapters in this dissertation include a quadratic specification for income. We use a linear specification for SES in this chapter for simplicity, as we are interested in how the overall social gradient differs by cohort.

As in the model-based trees, all regressions are weighted using the post-stratified weights provided with NIDS, and standard errors are clustered by individual to account for clustering of repeated observations on the same individual. We run the regressions with and without the controls $\boldsymbol{X_{it}}$ to explore whether these interactions are attenuated by controlling for other sociodemographic variables that are also associated with BMI. As there are significant differences in the social gradient between men and women in urban and rural areas (see seemingly unrelated regressions in Appendix B.5), we run the regressions for the full sample, and for urban and rural areas separately to explore whether different patterns exist in urban and rural areas.

Given the segregated history of South Africa, it is possible that different patterns are present in different race groups. Indeed, Figure 3.3 shows that the SES-BMI relationship has already reversed for white women, but not for other groups, as found by Wittenberg (2013). This suggests that the groups may on average be at different phases in the obesity transition, which may relate to long-run historical differences in SES. We therefore repeat the analysis for the African subsample. We focus on the African subsample because the subsample sizes for the other race groups are relatively small.

Finally, we further explore and illustrate the interactions between SES and the age, cohort and wave variables by plotting the marginal effect of income across various values of these variables (using Stata's *margins* command).

3.4 Results

3.4.1 Descriptive statistics

Figure 3.1 shows loess regressions of BMI on age by gender. For men, the relationship between BMI and age is approximately linear: BMI increases with age across the 25-64 age range. For women, the relationship between BMI and age is quadratic / inverse U-shaped: average BMI increases with age until the early fifties before declining slightly. An inverse U-shaped relationship between age and BMI has also been observed in other studies, for example Wittenberg (2013) in SA and Yang et al. (2021) and Sheehan et al. (2003) in the US.

Figure 3.2 shows loess regressions of BMI on income for urban and rural men and women. For men, BMI increases with income across most of the range of income: the income-BMI curve is relatively flat at low levels of income, before increasing fairly rapidly and then starting to flatten out for urban men. For women, BMI increases with additional income across most of the income distribution, but starts to decrease with income at high income levels. The gradient is steeper in urban than in rural areas for men. Urban women have a flatter gradient than rural women, but tend to be heavier at low levels of SES. The seemingly unrelated regressions in Appendix B.5 show that these differences in the SES gradient are significant.

We also plot the income-BMI relationship by race and gender in Figure 3.3. The relationship between BMI and income is positive for all gender and race groups, except for white women, for whom the relationship between BMI and income has already reversed, as also observed by Wittenberg (2013).

Table 3.1 shows mean BMI by age category, birth year, wave and income quintile. As seen in Figure

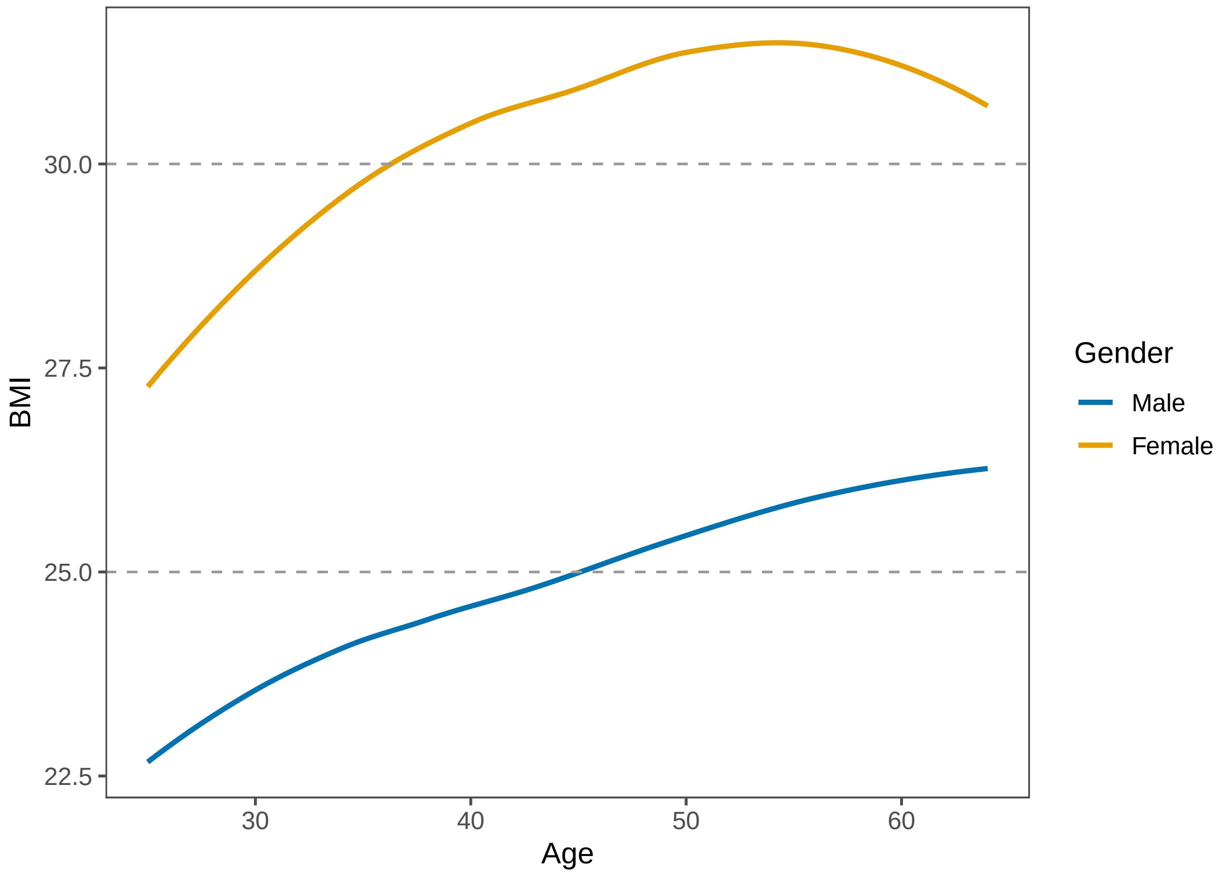


Figure 3.1: Loess regression of BMI on age
Note: Dashed lines indicate overweight and obesity cutoffs.

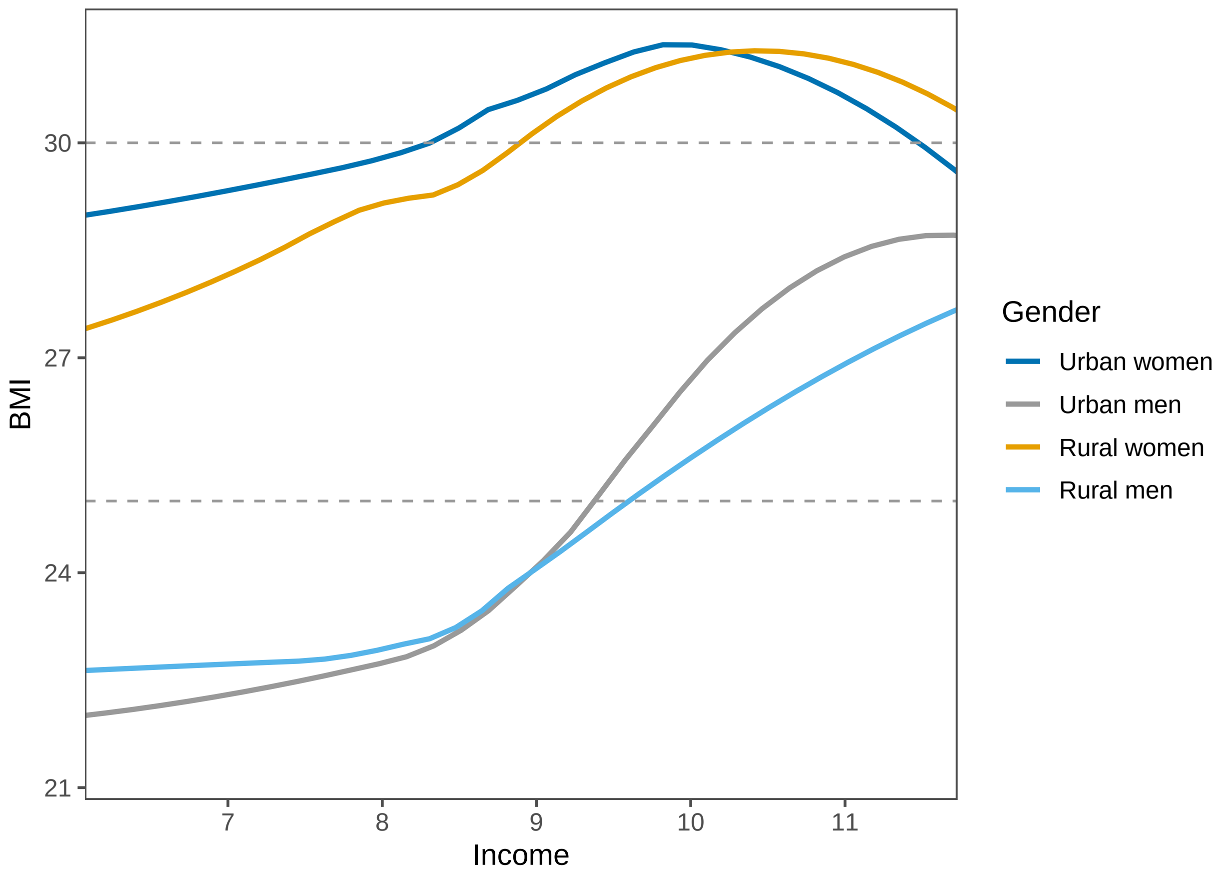


Figure 3.2: Loess regression of BMI on income
Note: Dashed lines indicate overweight and obesity cutoffs.

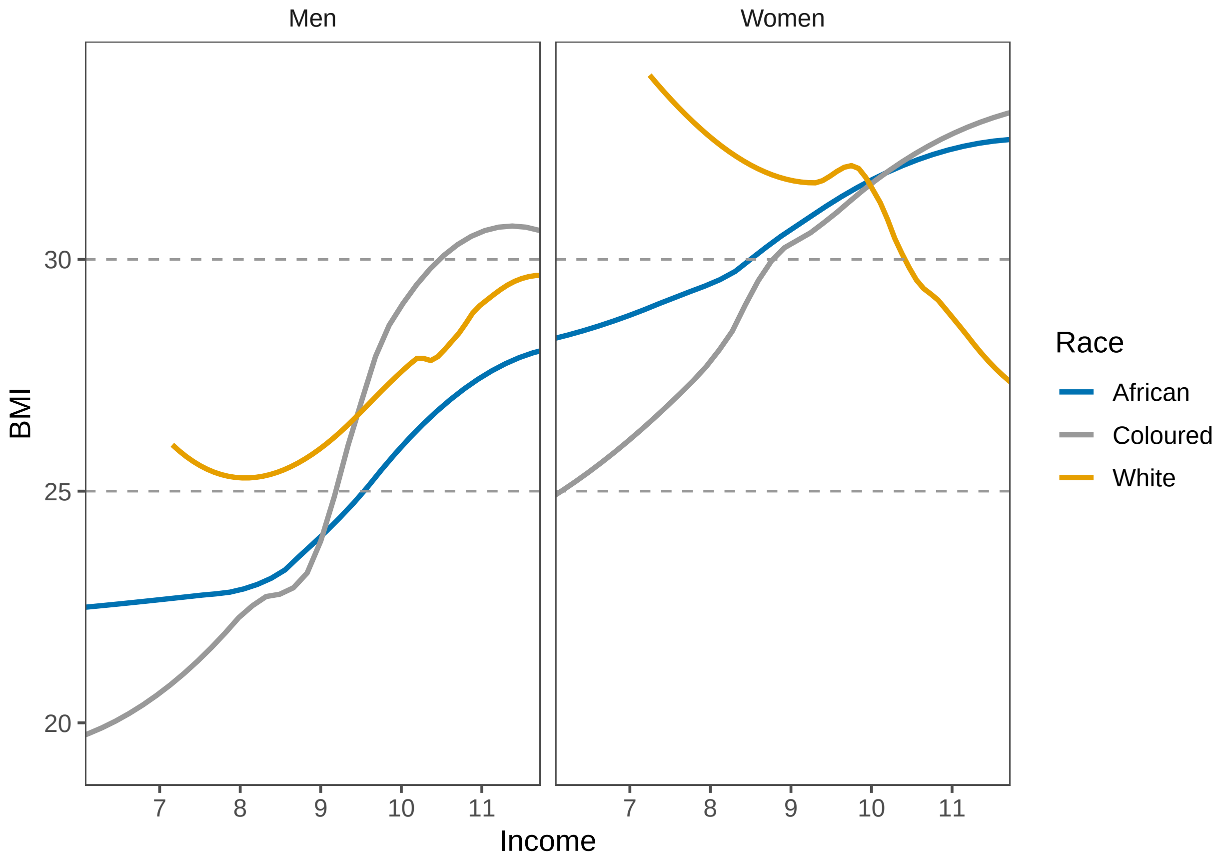


Figure 3.3: Loess regression of BMI on income by race
Note: Dashed lines indicate overweight and obesity cutoffs.

3.1, average BMI increases with age. Conversely, average BMI declines with birth year. This is to be expected as age and birth year are highly inversely correlated (but not perfectly because each birth year cohort is observed at 5 different ages between waves 1 and 5). Mean BMI in our sample also increased slightly after wave 1, before declining slightly (but not significantly) in wave 5. As seen in Figure 3.2, mean BMI increases with income, with a significantly higher mean BMI in quintile 5 than any of the other quintiles.

3.4.2 Main results

Figure 3.4 shows the birth year splits detected by the model-based tree for the full sample. Men are split at 1969, and women at 1953, with a flatter SES gradient for those born after the birth year split. Tables 3.2 and 3.3 (for women and men respectively) show the results of regressions interacting our SES variable with these birth year splits, as well as with age, wave and urban residence. For women, the SES gradient in BMI is not significantly flatter in younger cohorts in the full sample. The SES gradient also does not widen significantly with age. However, the SES gradient strengthens over time for this sample, with a positive and significant interaction between SES and the wave indicator for waves 3, 4 and 5 compared to wave 1. The SES gradient is also significantly flatter for urban than rural women, as also shown in the seemingly unrelated regression results in Appendix B.5.

For men, the SES gradient in BMI is significantly flatter in younger cohorts. The interaction between SES and age is positive, suggesting a widening of the SES gradient in BMI with age. The SES gradient also widens with time, with a significant positive interaction between SES and the wave 4 and 5 indicators. The interaction with urban residence reflects the opposite pattern to that for women: the SES gradient is steeper for men in urban areas. The interactions for men are all significant at the 5 percent level. Tables 3.2 and 3.3 also show the regression results with and without the other sociodemographic controls. The interactions of interest are generally attenuated slightly when including the controls, but remain significant where they were significant in the models without controls.

Tables 3.4 (women) and 3.5 (men) show the results of the interaction regressions stratified by urban or rural area, including the full set of controls. When run separately for urban and rural areas, the model-based recursive partitioning algorithm detected a birth year split of 1957 for urban women,

Table 3.1: Mean BMI by age, birth year, wave and income quintile

	Mean	95% Confidence interval
Age		
25-34	26.0	(25.7—26.2)
35-44	27.6	(27.3—27.9)
45-54	28.6	(28.2—29.0)
55-64	29.1	(28.7—29.4)
Birth year		
1943-59	29.0	(28.7—29.4)
1960-69	28.4	(28.0—28.9)
1970-79	27.1	(26.8—27.5)
1980-92	26.0	(25.7—26.4)
Wave		
Wave 1	27.0	(26.8—27.3)
Wave 2	27.4	(27.1—27.7)
Wave 3	27.6	(27.4—27.9)
Wave 4	27.6	(27.3—27.9)
Wave 5	27.3	(27.1—27.6)
Income quintile		
Quintile 1	26.5	(26.3—26.8)
Quintile 2	26.9	(26.6—27.2)
Quintile 3	27.0	(26.7—27.3)
Quintile 4	27.6	(27.3—28.0)
Quintile 5	29.1	(28.7—29.6)

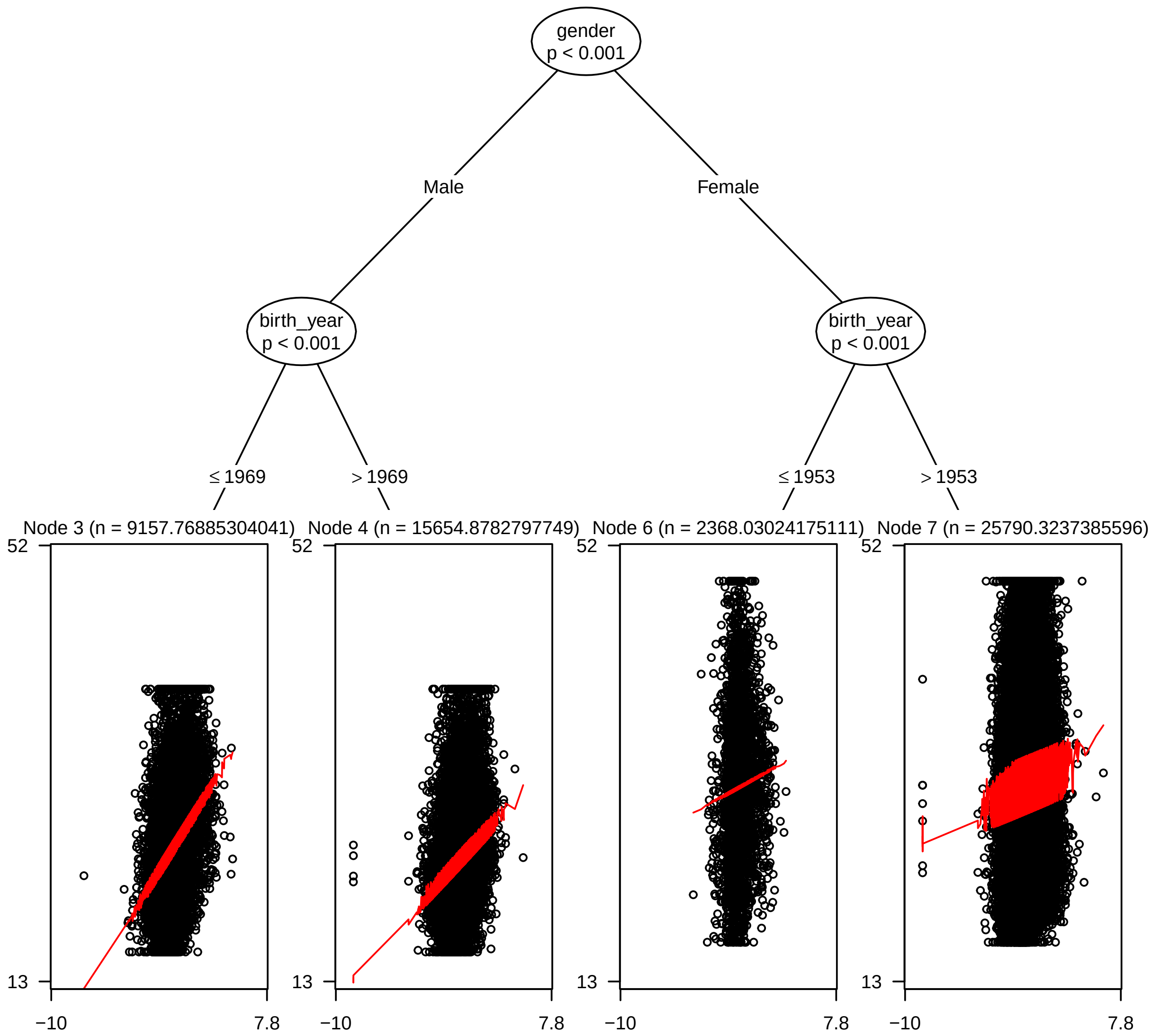


Figure 3.4: Model-based tree to detect parameter instability in SES-BMI relationship for full sample with age control

Source: Results of model-based recursive partitioning algorithm using NIDS.

Table 3.2: SES gradients by age, birth year, wave and urban residence: women

	Age		Cohort		Wave		Urban	
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Age	0.17**	0.13*	0.13***	0.13***	0.13***	0.13***	0.14***	0.13***
	(0.07)	(0.07)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)
Age x Household income	−0.00	−0.00						
	(0.01)	(0.01)						
Born 1954 or later	2.04***	1.82***	2.61	3.86	2.02***	1.81***	2.06***	1.85***
	(0.44)	(0.44)	(2.81)	(2.79)	(0.44)	(0.44)	(0.44)	(0.44)
Born 1954 or later x Income			−0.07	−0.24				
			(0.33)	(0.33)				
Wave 2	0.67***	0.62***	0.67***	0.62***	−0.13	0.14	0.65***	0.60***
	(0.16)	(0.17)	(0.16)	(0.17)	(1.40)	(1.35)	(0.16)	(0.16)
Wave 3	0.76***	0.78***	0.76***	0.77***	−2.63*	−2.44*	0.70***	0.73***
	(0.16)	(0.17)	(0.16)	(0.17)	(1.42)	(1.41)	(0.16)	(0.17)
Wave 4	0.92***	0.93***	0.92***	0.92***	−2.14	−1.86	0.84***	0.87***
	(0.17)	(0.18)	(0.17)	(0.18)	(1.52)	(1.49)	(0.17)	(0.17)
Wave 5	0.73***	0.66***	0.73***	0.65***	−3.18**	−2.52*	0.64***	0.59***
	(0.18)	(0.18)	(0.18)	(0.18)	(1.43)	(1.39)	(0.18)	(0.18)
Wave 2 x Income					0.10	0.06		
					(0.17)	(0.16)		
Wave 3 x Income					0.40**	0.38**		
					(0.17)	(0.17)		
Wave 4 x Income					0.36**	0.33*		
					(0.18)	(0.18)		
Wave 5 x Income					0.45***	0.37**		
					(0.17)	(0.16)		
Urban	0.83***	1.02***	0.82***	1.02***	0.83***	1.03***	7.23***	6.81***
	(0.21)	(0.24)	(0.21)	(0.24)	(0.21)	(0.24)	(1.55)	(1.59)
Urban x Income							−0.76***	−0.69***
							(0.18)	(0.19)
Income	0.52	0.52	0.40	0.73**	0.07	0.28*	0.90***	0.98***
	(0.34)	(0.35)	(0.32)	(0.35)	(0.13)	(0.15)	(0.14)	(0.14)
Constant	17.00***	17.08***	18.09***	15.32***	20.86***	19.12***	13.90***	13.25***
	(2.98)	(3.05)	(2.75)	(3.06)	(1.37)	(1.60)	(1.31)	(1.46)
Observations	31451	31451	31451	31451	31451	31451	31451	31451
Adjusted R^2	0.04	0.08	0.04	0.08	0.05	0.08	0.05	0.08
Controls	No	Yes	No	Yes	No	Yes	No	Yes

* p <0.1, ** p <0.05, *** p <0.01. Standard errors in parentheses.

Note: Standard errors clustered by individual. Controls are race group, education, employed, married/cohabiting, province, smoker, exercises weekly, and ever given birth.

Table 3.3: SES gradients by age, birth year, wave and urban residence: men

	Age		Cohort		Wave		Urban	
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Age	−0.13*	−0.14**	0.07***	0.05***	0.07***	0.05***	0.07***	0.05***
	(0.07)	(0.07)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)
Age x Household income	0.02***	0.02***						
	(0.01)	(0.01)						
Born 1970 or later	−0.20	−0.34	4.52***	4.04***	−0.24	−0.37	−0.20	−0.34
	(0.35)	(0.33)	(1.51)	(1.43)	(0.35)	(0.33)	(0.35)	(0.33)
Born 1970 or later x Income			−0.53***	−0.49***				
			(0.17)	(0.16)				
Wave 2	0.14	0.16	0.13	0.16	−0.46	−0.21	0.16	0.18
	(0.16)	(0.16)	(0.16)	(0.16)	(1.48)	(1.44)	(0.16)	(0.16)
Wave 3	0.23	0.37**	0.23	0.37**	−1.76	−0.94	0.28*	0.40***
	(0.15)	(0.14)	(0.15)	(0.14)	(1.41)	(1.31)	(0.15)	(0.15)
Wave 4	−0.14	0.06	−0.14	0.06	−4.54***	−3.81**	−0.09	0.08
	(0.16)	(0.16)	(0.16)	(0.16)	(1.62)	(1.49)	(0.16)	(0.16)
Wave 5	−0.42**	−0.21	−0.42**	−0.21	−4.58***	−3.68***	−0.37**	−0.19
	(0.17)	(0.16)	(0.17)	(0.16)	(1.40)	(1.32)	(0.17)	(0.17)
Wave 2 x Income					0.07	0.04		
					(0.17)	(0.17)		
Wave 3 x Income					0.23	0.15		
					(0.16)	(0.15)		
Wave 4 x Income					0.50***	0.44**		
					(0.19)	(0.17)		
Wave 5 x Income					0.47***	0.39***		
					(0.16)	(0.15)		
Urban	0.20	0.22	0.20	0.22	0.24	0.26	−4.49***	−2.36*
	(0.15)	(0.17)	(0.15)	(0.17)	(0.15)	(0.17)	(1.27)	(1.22)
Urban x Income							0.55***	0.30**
							(0.15)	(0.14)
Income	0.47	0.09	1.73***	1.23***	1.17***	0.72***	1.03***	0.72***
	(0.32)	(0.31)	(0.15)	(0.15)	(0.13)	(0.13)	(0.11)	(0.11)
Constant	17.30***	21.55***	6.01***	11.30***	11.13***	15.88***	12.20***	15.77***
	(2.85)	(2.69)	(1.46)	(1.51)	(1.30)	(1.31)	(1.10)	(1.15)
Observations	20958	20958	20958	20958	20958	20958	20958	20958
Adjusted R^2	0.15	0.23	0.15	0.23	0.15	0.23	0.15	0.23
Controls	No	Yes	No	Yes	No	Yes	No	Yes

* p <0.1, ** p <0.05, *** p <0.01. Standard errors in parentheses.

Note: Standard errors clustered by individual. Controls are race group, education, employed, married/cohabiting, province, smoker, and exercises weekly.

1955 for rural women, 1976 for urban men and 1961 for rural men. Results for the model-based trees are available in Figures B.1.1 and B.1.2 in Appendix B.1.1. The results are similar to those for the combined urban and rural sample. The SES gradient is not significantly different in younger cohorts of women in rural or urban areas. For rural women, the SES gradient widens with age, but the interaction is not significant for urban women.

For men, the SES gradient is significantly flatter among younger cohorts in both urban and rural areas. For urban men, the SES gradient is weaker for those born in 1976 or later, while for rural men the SES gradient is weaker for those born in 1961 or later, but in the latter case is only significant at the 10 percent level. The SES gradient widens with age in both urban and rural areas, though in urban areas the interaction is only significant at the 10 percent level. The SES gradient also appears to have widened over successive waves of NIDS for men and women in urban areas.

We illustrate these results by plotting the marginal effect of income by age, birth year split and wave in Figures 3.5 to 3.7. Figure 3.6 shows a lower marginal effect of income in younger cohorts of men and rural women, but a higher marginal effect in younger cohorts of urban women (though the regressions results show that the interaction is not significant for women). Figure 3.5 shows a generally increasing marginal effect of income with age for men and rural women, but a slight (insignificant) downward trend in the marginal effect of income with age for urban women. Figure 3.7 shows an upward trend in the marginal effect of income with successive waves for men and urban women, but no trend for rural women.

The results for the African subsample are available in Appendix B.2. In the combined urban and rural sample, the SES gradient is significantly flatter for younger cohorts – for women born after 1961 and for men born after 1969. For women these results are driven by those in urban areas, while for men the gradient is significantly flatter among younger cohorts in both urban and rural areas. The SES gradient also widens with age for African men and women. The SES gradient is flatter in urban areas for women and steeper in urban areas for men. As in the full sample, the SES gradient widens with successive waves for urban residents, but not for rural residents. These interactions are again illustrated by plotting the marginal effect of income at various values of age, birth year split and wave (Figures B.2.4 to B.2.6 in Appendix B.2).

Results for obesity are available in Appendix B.3. Unlike for BMI, the SES gradient in obesity is significantly flatter among younger cohorts of women, though only at the 10 percent level. As for

Table 3.4: SES gradients by age, birth year and wave, stratified by urban residence: women

	Urban			Rural		
	(1) Age	(2) Cohort	(3) Wave	(4) Age	(5) Cohort	(6) Wave
Income	0.67	−0.03	0.04	0.06	1.29***	0.96***
	(0.46)	(0.40)	(0.20)	(0.44)	(0.34)	(0.20)
Age	0.20**	0.14***	0.14***	−0.02	0.14***	0.14***
	(0.10)	(0.02)	(0.02)	(0.08)	(0.01)	(0.01)
Age x Household income	−0.01			0.02*		
	(0.01)			(0.01)		
Born 1957 or later	1.54**	−2.71	1.53**			
	(0.60)	(3.58)	(0.60)			
Born 1957 or later x Income		0.47				
		(0.39)				
Born 1955 or later				2.07***	6.05**	2.10***
				(0.47)	(2.86)	(0.47)
Born 1955 or later x Income					−0.48	
					(0.35)	
Wave 2	0.73***	0.73***	−0.71	0.40**	0.39**	3.83**
	(0.24)	(0.24)	(1.84)	(0.19)	(0.19)	(1.89)
Wave 3	0.79***	0.80***	−3.75**	0.63***	0.61***	0.05
	(0.24)	(0.24)	(1.87)	(0.19)	(0.19)	(1.92)
Wave 4	1.03***	1.04***	−3.80*	0.57***	0.55***	3.01
	(0.25)	(0.25)	(2.03)	(0.20)	(0.20)	(2.25)
Wave 5	0.76***	0.77***	−3.51*	0.25	0.24	−0.70
	(0.25)	(0.25)	(1.85)	(0.23)	(0.23)	(2.17)
Wave 2 x Income			0.16			−0.43*
			(0.21)			(0.24)
Wave 3 x Income			0.51**			0.07
			(0.22)			(0.24)
Wave 4 x Income			0.54**			−0.30
			(0.23)			(0.28)
Wave 5 x Income			0.48**			0.11
			(0.21)			(0.27)
Constant	16.75***	23.12***	22.34***	20.51***	10.41***	13.11***
	(4.20)	(3.79)	(2.05)	(3.82)	(3.02)	(2.04)
Observations	16222	16222	16222	15229	15229	15229
Adjusted R^2	0.07	0.07	0.08	0.10	0.10	0.10
Controls	Yes	Yes	Yes	Yes	Yes	Yes

* p <0.1, ** p <0.05, *** p <0.01. Standard errors in parentheses.

Note: Standard errors clustered by individual. Controls are race group, education, employed, married/cohabiting, province, smoker, exercises weekly, and ever given birth.

Table 3.5: SES gradients by age, birth year and wave, stratified by urban residence: men

	Urban			Rural		
	(1) Age	(2) Cohort	(3) Wave	(4) Age	(5) Cohort	(6) Wave
Income	0.23	1.23***	0.78***	−0.01	1.02***	0.54**
	(0.40)	(0.15)	(0.17)	(0.33)	(0.21)	(0.23)
Age	−0.13	0.06***	0.06***	−0.14**	0.01	0.01
	(0.09)	(0.02)	(0.02)	(0.07)	(0.01)	(0.01)
Age x Household income	0.02*			0.02**		
	(0.01)			(0.01)		
Born 1976 or later	−0.52	3.71**	−0.50			
	(0.36)	(1.75)	(0.36)			
Born 1976 or later x Income		−0.46**				
		(0.19)				
Born 1961 or later				−1.02***	2.47	−0.96**
				(0.38)	(1.93)	(0.38)
Born 1961 or later x Income					−0.41*	
					(0.23)	
Wave 2	0.09	0.08	−0.89	0.37	0.36	0.86
	(0.21)	(0.21)	(1.85)	(0.24)	(0.24)	(2.69)
Wave 3	0.41**	0.41**	−1.11	0.48**	0.47**	−0.19
	(0.19)	(0.19)	(1.66)	(0.22)	(0.22)	(2.22)
Wave 4	0.21	0.21	−4.08**	0.01	−0.00	−2.25
	(0.21)	(0.21)	(2.00)	(0.21)	(0.21)	(2.22)
Wave 5	−0.11	−0.10	−3.79**	−0.05	−0.07	−3.41
	(0.22)	(0.22)	(1.72)	(0.24)	(0.24)	(2.14)
Wave 2 x Income			0.11			−0.06
			(0.21)			(0.34)
Wave 3 x Income			0.17			0.09
			(0.18)			(0.27)
Wave 4 x Income			0.47**			0.27
			(0.22)			(0.27)
Wave 5 x Income			0.40**			0.40
			(0.19)			(0.26)
Constant	20.43***	11.22***	15.33***	24.34***	15.69***	19.55***
	(3.54)	(1.58)	(1.63)	(2.86)	(2.00)	(2.05)
Observations	11896	11896	11896	9062	9062	9062
Adjusted R^2	0.25	0.25	0.24	0.16	0.16	0.16
Controls	Yes	Yes	Yes	Yes	Yes	Yes

* p <0.1, ** p <0.05, *** p <0.01. Standard errors in parentheses.

Note: Standard errors clustered by individual. Controls are race group, education, employed, married/cohabiting, province, smoker, and exercises weekly.

BMI, the gradient is significantly flatter for younger cohorts of men. The SES gradient in obesity widens slightly with age for men, but not for women. Unlike for BMI, the interactions between SES and wave are not significant.

3.5 Discussion

We find that the socioeconomic gradient in BMI in South Africa is flatter among younger cohorts of men. For women, the socioeconomic gradient in BMI is not significantly different in younger cohorts in the whole sample, but the gradient is flatter in younger cohorts among African women, particularly in urban areas. There are also indications that the gradient in obesity (rather than BMI) is flatter among younger cohorts of women. These results provide support for our hypothesis that the association between SES and BMI may be weaker in younger cohorts.

We also find that the positive socioeconomic gradient in BMI in South Africa is flatter among men and women at younger *ages*, particularly in the African subsample, suggesting that the gradient may widen with age. This is consistent with research from developed countries showing a widening of the SES gradient in BMI with age, even though in those contexts the SES gradient in BMI is negative. The SES gradient has also widened between the first wave of NIDS in 2008 and the most recent waves conducted in 2012, 2014-15 and 2017; this seems to be driven mainly by a widening of the gradient in urban areas. We cannot say conclusively whether the different SES gradients are due to age or cohort effects, particularly given the relatively short time range covered by NIDS (a maximum of nine years). Age-period-cohort models separating out the effects of these three variables are notoriously difficult to identify, because of the exact linear relationship between age, time period and cohort: age is equal to period minus cohort. This identification problem has been the subject of a decades-long methodological debate (see e.g. Bell & Jones, 2014a, 2014b, 2018; Fienberg, 2013; Yang & Land, 2006). It is possible that the SES gradient will widen for current younger cohorts as they age, or alternatively the flatter SES gradient may represent a longer-term shift, and their SES gradient may remain flatter than that of preceding cohorts even as they age. This paper presents tentative evidence that the flattening of the SES gradient is due in part to a cohort effect, at least among men, though we would need more research with data observing individuals in the same birth cohort at a wider range of ages to confirm that it is indeed a cohort rather than an age effect.

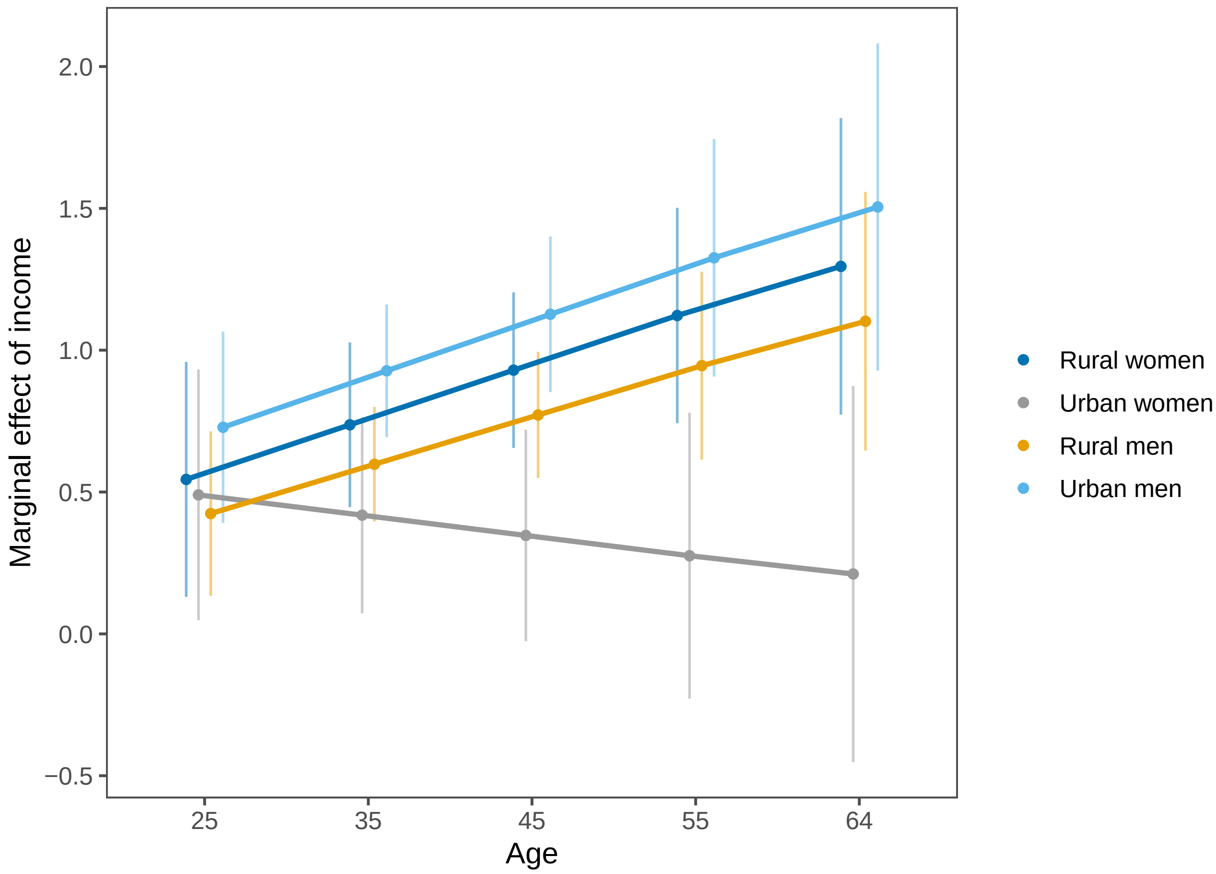


Figure 3.5: Marginal effect of income by age

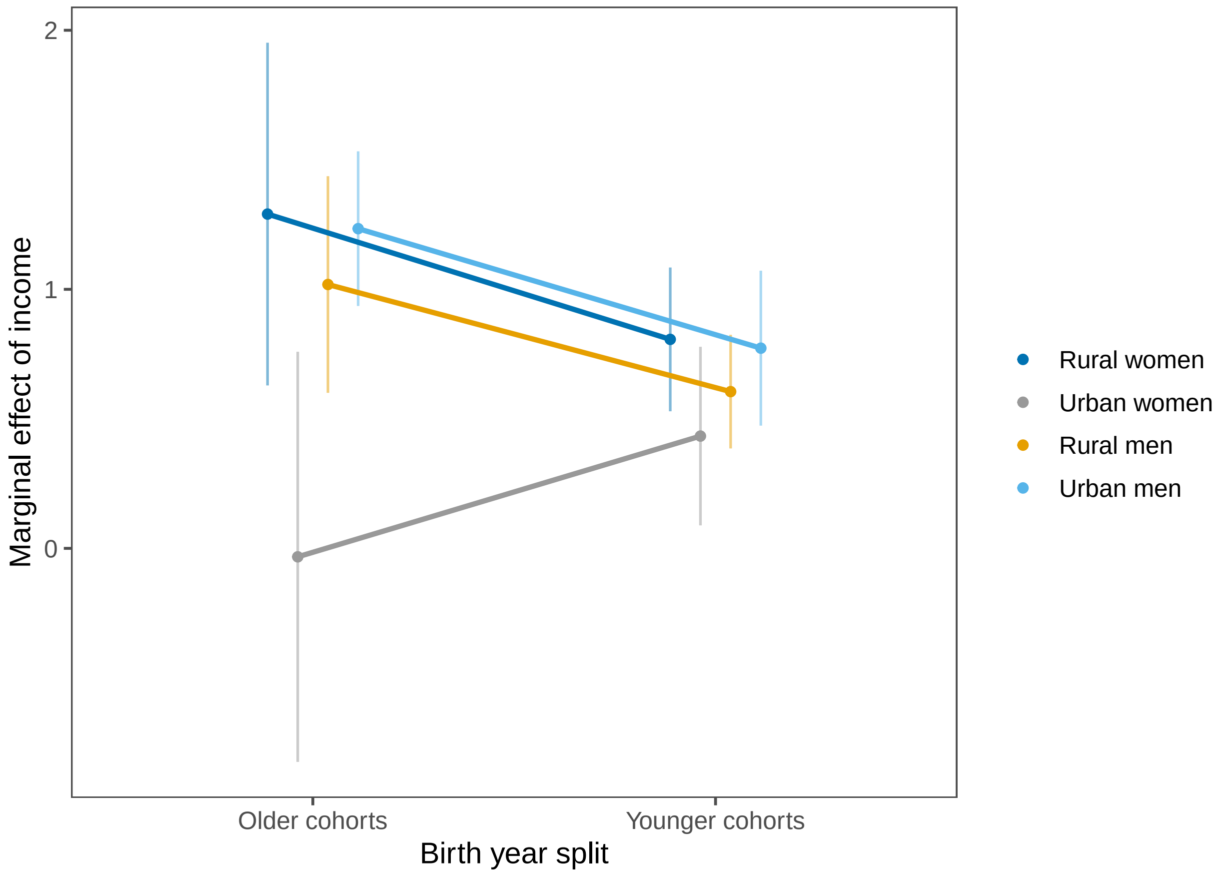


Figure 3.6: Marginal effect of income by cohort based on birth year splits detected by model-based trees

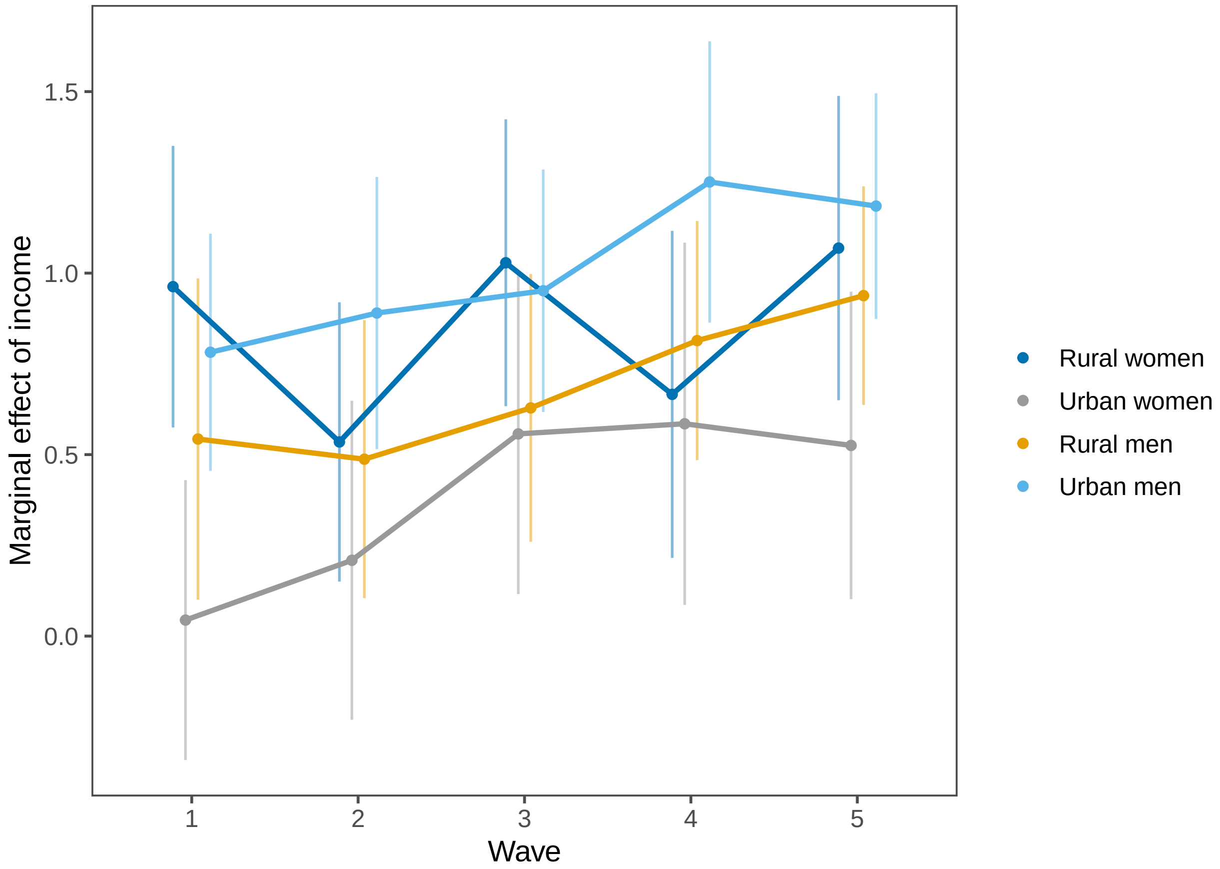


Figure 3.7: Marginal effect of income by wave

Speculatively, one reason for the flatter SES gradient in younger cohorts may be improvements in child nutrition and living conditions in early childhood over time, so that successive cohorts have on average experienced increasingly better nutrition in childhood. Mpeta et al. (2018) document a slow increase in the heights of black South African men in the second half of the twentieth century, while other groups showed a larger growth. The increase in average height suggests an improvement in living standards, particularly in early life. As shown in Chapter 2, the SES gradient is flatter for individuals from a higher childhood SES background, and the results for adult height also support the interpretation that this is due to better childhood nutrition. Another reason may be increasing urbanisation in the wake of the abolition of apartheid-era influx controls. Influx controls restricting black South Africans from living and working in urban areas were abolished in 1986. This may have caused a distinct break in economic life. The timing of the structural breaks identified by the model-based partitioning algorithm corresponds loosely to the relaxation of these controls during young adulthood, particularly for men. Wang et al. (2020) found that the social gradient in obesity was positive for rural-to-urban migrants in China who were aged 20 or older when they arrived in an urban area, but was negative among migrants who were aged below 20 on arrival. It is possible that the removal of influx controls allowed younger generations of black South Africans to migrate to urban areas at a younger age, and that this was associated with a flattening of the social gradient in body weight among these cohorts. Urbanisation may also be associated with greater access to energy-dense processed foods for individuals with lower incomes, resulting in increases in body weight among less affluent individuals and thus a flattening of the social gradient.

A limitation of this study is that the relationship between SES and BMI may be bidirectional. The implied direction of causality in this study is from SES to BMI, but causality may also run in the opposite direction: there is evidence from high-income countries of a negative causal effect of obesity on wages and employment (for a review see Cawley, 2015), and Henry and Kollamparambil (2017) provides evidence of an inverse U-shaped relationship between BMI and wages and employment in South Africa. Furthermore, it is possible that age or cohort effects differ by SES, rather than the other way around. For example, certain cohorts may have been exposed to different early life circumstances, such as different nutritional environments, which may have a lasting effect on BMI and obesity risk later in life. It is possible that the effect of these factors may differ by SES. For example, those from a higher SES background may have been relatively shielded from nutritional shocks common to certain cohorts. Factors such as the expansion of processed foods and fast foods may also have different effects for different SES groups. This paper does not estimate causal effects of SES on BMI, and does not attempt to disentangle the direction of the association between SES and BMI.

Another possible source of confounding is mortality selection. Obesity increases the risk of chronic health problems and increases mortality risk (Abdelaal et al., 2017; Angelantonio et al., 2016), which means that severely obese people may be more likely to drop out of the sample through death. Higher SES may have a protective effect against mortality, for example through access to better medical care (Bassuk et al., 2002; Kabudula et al., 2017). This may confound the relationship between SES and BMI, as well as the interactions between SES and age or cohort interaction: it is possible that at older ages the higher SES obese are more likely to be alive than their lower SES counterparts, and therefore to be observed in the sample. This could result in wider SES-BMI gradients for older people, as we observe in this study. We do not address this possibility here.

In spite of these limitations, this paper has several strengths. First, instead of creating arbitrary birth year splits, we used an innovative machine learning algorithm to detect structural breaks in the SES-BMI relationship by birth year. This paper illustrates the potential of the model-based recursive partitioning algorithm for detecting changes in phases of the obesity transition, though ideally this would require additional long-run data. Second, this paper brings a dynamic generational and life course perspective to the relationship between SES and BMI in South Africa; previous research has only studied this relationship from a static perspective.

3.6 Conclusion

We show that the social gradient in BMI is flatter among younger cohorts of South African men, and find some indications of a flatter gradient in body weight for younger cohorts of women too. We cannot say definitively whether these differences are due to age or cohort effects, but the possibility that the SES-BMI relationship is flattening in younger cohorts is important to consider when looking ahead to the future of the obesity transition in South Africa, and in other countries in stage 2 of the obesity transition. It suggests that, while the SES gradient in body weight remains positive for most groups, we may see a reversal in this gradient in younger people first. This paper highlights the need for more research on cohort and life course trajectories in BMI and in health more broadly in South Africa, as well as on how socioeconomic factors are related to BMI across birth cohorts, over the life course, and over time. However, the true potential of such research can only be realised with longitudinal data following the same individuals over a longer period than we currently have available in South Africa. This study introduces a new way of thinking about the nutrition and obesity transitions, raising the possibility that shifts in the socioeconomic patterning of obesity may happen not only over time, but at different starting points for different generations. Perceptions of body weight, attitudes to food consumption and health awareness may change across generations, resulting in shifts in the social gradient in body weight across successive generations. As consecutive younger generations adopt new norms early in their lives, this may shift the population closer towards the next phase of the obesity transition. Though the social gradient in body weight remains positive, there are signs that the burden of obesity may already have begun to shift towards the poor among younger generations. The timing of the structural breaks corresponds loosely to the relaxation of apartheid-era influx controls during young adulthood, subsequent urbanisation and widespread labour market entry. Speculatively, this suggests that this period may have represented a distinct break in economic life, which may be associated with increases in body weight, especially among less affluent households. It is unlikely that this trajectory will change rapidly, and South Africa's already high rates of obesity are likely to increase as younger generations continue on this path. This calls for public health interventions that can help the country navigate these changes. This paper was one of the first to investigate the social gradient in body weight at a time of transition, and highlights the importance of exploring generational differences in these relationships. Younger generations are likely to drive future trends, and provide a lens to study possible future changes in the prevalence and socioeconomic patterning of obesity.

CHAPTER 4

THE ASSOCIATION BETWEEN ECONOMIC INSECURITY AND OBESITY MAY DEPEND ON ACCESS TO EXCESS CALORIES: EVIDENCE FROM SOUTH AFRICA

4.1 Introduction

Economic insecurity – or the anxiety produced by exposure to subjectively significant potential economic losses or adverse economic events (Bossert & D'Ambrosio, 2013; Osberg, 1998) – and various closely related concepts have been found to affect a range of physical and mental health outcomes in developed countries. The concept of economic insecurity is distinct from that of poverty or vulnerability to poverty that has dominated the discourse in developing countries (Dercon, 2006; Osberg, 2010), in that it covers a wider range of potentially negative economic events beyond just the risk of falling into poverty, and – crucially – in that it may affect even the middle class and the affluent (Osberg, 2010). The well-known Whitehall II studies, which studied the health outcomes of British civil servants in a department facing the threat of privatisation and thus greater job insecurity, found that job insecurity and work-related stress affected self-rated health (Ferrie et al., 1995), cardiovascular health (Ferrie et al., 2013) and BMI (Ferrie et al., 1998). Economic insecurity has also been found to impact mental health (Kopasker et al., 2018).

Economic insecurity has also been posited as a possible cause of obesity through its effect on stress (Smith et al., 2009; Wisman & Capehart, 2010). Anxiety is central to the concept of economic insecurity as defined by Osberg (1998). Wisman and Capehart (2010) argue that economic insecurity in an environment where fatty and energy-dense foods are readily available may have contributed to the rise in obesity in recent decades, particularly in the US but also worldwide. They observe that the dramatic rise in obesity levels in the US coincided with a rise in economic insecurity since the 1980s. Across developed countries, economic insecurity has been found to be a stronger predictor of obesity rates than the increasing accessibility and fall in relative prices of fast foods and energy-dense processed foods (Offer et al., 2010).

As outlined in the literature review in Chapter 1, there is evidence that stress may promote obesity. Smith et al. (2009) argue that body fat provides a form of 'insurance' against starvation – body fat acts as an energy reserve that can be drawn upon to prevent starvation when food is scarce. As such, overeating may be a rational response to uncertainty in food-scarce environments. Though most modern humans in developed countries do not face the threat of starvation, and so this response is no longer optimal, it has become hard-wired into the body's response to stress. Even in energy-abundant environments such as those faced by most developed country populations, stress may cause the body to act as if it is facing food scarcity, even if starvation is not a realistic threat. This can be seen as an extension of the evolutionary mismatch hypothesis (outlined in Chapter 1). Stress triggers the body's 'fight-or-flight' response, stimulating the production of stress hormones called glucocorticoids, such as cortisol. This results in the temporary suppression of appetite and digestion. However, the chronic activation of this stress response may promote overeating and weight gain (Wisman & Capehart, 2010). Glucocorticoids have been found to promote cravings for and consumption of energy-dense 'comfort' foods, a form of 'self-medication' against the effects of stress (Dallman et al., 2005). These 'comfort' foods stimulate pleasure centres in the brain, reducing the negative emotions produced by stress. It is thus possible that, in energy-abundant food environments, the stress created by economic insecurity may promote weight gain and excess body fat.

There is evidence that economic insecurity in various forms may contribute to obesity[1], but this comes almost exclusively from developed countries (the one exception being a study by Staudigel (2016) on Russia in transition). It is probable that economic insecurity only contributes to obesity in environments where sufficient energy is readily available and accessible. This may mean that in many developing countries, where a substantial proportion of the population does not have access to sufficient calories to meet their nutritional needs, economic insecurity does not show the relationship with body mass index (BMI) observed in developed countries. Based on comparisons of associations between food insecurity and body weight observed in more developed and less developed countries, it has been proposed that food insecurity specifically (as opposed to economic insecurity) only becomes a risk factor for obesity when the nutrition transition – the shift from traditional diets high in starch and fibre to diets higher in fat, sugar and processed foods – reaches a stage where energy-dense foods are available and affordable (Farrell et al., 2018; Kac et al., 2012). However,

[1] There is also some literature on food insecurity in developed and some developing countries (Farrell et al., 2018; Pan et al., 2012). This is related to that on economic insecurity, as the effects of food insecurity may trigger the same stress response as economic insecurity. However, while food insecurity could be seen as a form of economic insecurity, the concept of economic insecurity is broader than that of food insecurity, considering a potentially wider range of economic risks. This study concerns itself with economic insecurity rather than food insecurity.

to our knowledge no study has examined whether the association between economic insecurity and body weight differs between high-income and low-income groups within the same country, which may have differing access to energy-dense processed foods and be at different stages of the nutrition transition.

Popkin and Gordon-Larsen (2004) and Popkin (2006) outline five patterns or stages of the nutrition transition, of which stages 3 to 5 are relevant here. Stage 3 involves receding famine, and is characterised by starchy, low variety traditional diets. Nutritional deficiencies such as stunting tend to have a relatively high prevalence in this stage. Stage 4 involves a shift towards increased fats, sugar and processed foods, with associated problems of obesity and degenerative disease. Stage 5 involves behavioural change towards healthier diets and increased leisure-time physical activity, resulting in reduced obesity. We hypothesise that economic insecurity is more likely to be associated with higher body weight among individuals in stages 4 or 5 of the nutrition transition, as is the case for developed country populations who are already in stages 4 or 5. Individuals in stage 3 are less likely to be able to access sufficient calories and energy-dense processed foods to be able to accumulate excess body fat.

Using the South African National Income Dynamics Study (NIDS) panel data, we investigate whether the association between economic insecurity and body weight differs by income level. We explore whether there is any evidence of a stronger association between economic insecurity and BMI among higher-income individuals, who would be expected to have greater access to excess calories and energy-dense processed foods. South Africa makes an interesting case study because it straddles stages 3 to 5 of the nutrition transition, with segments of the population in each of the three stages. This is in part due to South Africa's very high levels of income inequality, and (relatedly) in part due to its history of unequal and segregated spatial development, resulting in many rural former homelands with a very different level of development and also a different food landscape to that in more affluent urban areas. Many South Africans are still at the receding famine stage (stage 3), experiencing high levels of childhood stunting and struggling to obtain sufficient quantity and quality of calories to meet their nutritional needs, as evidenced by high levels of hunger (Van der Berg et al., 2021). On the other hand, relatively well-off South Africans, and to some extent those residing in urban areas, tend to be at a more advanced stage of the nutrition transition, in stages 4 or 5, able to access excess calories and energy-dense processed foods. South Africa's high levels of unemployment and limited social safety net would also be expected to yield high levels of economic insecurity, and the penetration of supermarkets and fast food ('Big Food') in the South African food market mean that relatively cheap energy-dense processed foods are fairly widely and increasingly available and affordable (Igumbor et al., 2012), though not affordable for all. Reliance on subsistence farming has declined; households are increasingly likely to buy food on the market rather than produce their own (Baiphethi & Jacobs, 2009). Proximity to supermarkets and fast food outlets has been linked to increased risk of obesity in South Africa (Otterbach et al., 2021). South Africa also has high rates of obesity, particularly among women: South African women have one of the highest average body mass indexes (BMI) in the world (NCD Risk Factor Collaboration, 2016).

The remainder of this paper proceeds as follows. Section 4.2 defines and discusses the measurement of economic insecurity, and reviews the literature on the association between economic insecurity and BMI or obesity. Section 4.3 outlines our economic insecurity measures and methodology. Section 4.4 presents our results, Section 4.5 discusses our findings, and Section 4.6 concludes.

4.2 Economic insecurity and obesity

4.2.1 Definition of economic insecurity

In an influential paper, Osberg (1998) defines economic insecurity as "the anxiety produced by a lack of economic safety, i.e. by an inability to obtain protection against subjectively significant potential economic losses". Bossert and D'Ambrosio (2013) define it as "the anxiety produced by the possible exposure to adverse economic events and by the anticipation of the difficulty to recover from them". Though many definitions have been proposed, Osberg (2015) argues that the definitions in the most prominent academic articles have in common the idea that "individuals feel economically insecure when they perceive a significant and unavoidable downside economic risk – i.e. a hazard or danger – looming in their future" (for a review of the concept and proposed definitions see Osberg, 2015). Key to this concept is that it is subjective, depending on individual perceptions of economic hazards. Individual attitudes to risk may influence the degree to which an individual feels economically insecure. This also implies that even the relatively well-off could be negatively affected by insecurity, which could have important consequences for their health and wellbeing. This concept concerns future hazards, rather than current or past events. Though past experiences may influence individuals' assessment of future risks, the concept is thus inherently forward-looking (Osberg, 2015). Furthermore, the concept of economic insecurity is focused on downside risks, or negative events. This distinguishes the concept from that of economic volatility, which increases with both positive and negative shocks (Osberg, 2015).

4.2.2 Measuring economic insecurity

A considerable and growing literature has developed in recent years around the measurement of economic insecurity, focused on developed countries. This literature has not yet reached a consensus on the best way to measure economic insecurity. This section outlines some of the approaches to measuring economic insecurity.

A first distinction can be made between macro- and micro-level measures of economic insecurity. The former are best suited to measuring trends in economic insecurity across time or countries. An example is the Index of Economic Well-Being (IEWB) Economic Security Index, which measures four specific economic risks at the national level, namely unemployment, sickness, widowhood, and old age (Osberg & Sharpe, 2002, 2014). Given that this paper explores the association between economic insecurity and BMI within South Africa, we take a micro-level approach.

A second distinction can be made between objective and subjective measures. Examples of subjective measures include self-assessed job security, financial satisfaction, ability to raise emergency funds (Rohde et al., 2015), inability to deal with unexpected expenses, and changes in respondents' assessment of their ability to go on holiday (Cantó et al., 2020; Romaguera-de-la-Cruz, 2020). As the concept of economic insecurity concerns subjective perceptions and individual anxieties, an argument can be made that survey questions eliciting subjective assessments of economic insecurity or economic risks are best able to capture these individual anxieties regarding one's economic future. Individuals differ in their degree of risk aversion and predisposition to anxiety, so two individuals may experience different levels of anxiety when faced with the same objective risk. Subjective measures are better able to capture these differences. Interestingly, Rohde et al. (2016) found that subjective measures were more strongly predictive of mental health than objective measures[2] based on past income streams. However, questions on subjective feelings of anxiety or economic

[2]Such as the income drop or Bossert and D'Ambrosio (2013) measures based on past income streams, or those based on the predicted probability of negative future states, as discussed in more detail below.

insecurity may be subject to translation issues[3], and responses may be influenced by cultural biases and transient events or life circumstances (Osberg, 2015). In practice, the literature has largely used objective measures, partly due to a lack of availability of subjective measures in most of the household surveys commonly used to assess economic insecurity.

A further choice has to be made between forward-looking and backward-looking measures. Examples of forward-looking measures include the predicted probabilities of future unemployment or expenditure distress (Rohde et al., 2015; Romaguera-de-la-Cruz, 2020) or of falling into poverty, and a series of measures proposed by Rohde et al. (2020) based on the unpredictability of future incomes after filtering out predictable variations in income. Examples of backward-looking measures include the income drop index (Hacker et al., 2014; Rohde et al., 2015), the index proposed by Bossert and D'Ambrosio (2013), and measures based on income volatility (Rohde et al., 2017a; Smith et al., 2009; Staudigel, 2016). The income drop index classifies as insecure individuals experiencing a drop of 25 percent or more in household income from year to year[4]. The Bossert and D'Ambrosio (2013) index consists of a buffer stock of current wealth as well as a weighted sum of past gains and losses in wealth; losses receive higher weight than gains to account for loss aversion and changes in wealth are discounted so that more recent changes receive a higher weight than changes further in the past[5]. Backward-looking measures based on past income or wealth streams assume that the experience of insecurity in the past is a good proxy for current or future insecurity, as it would be expected to influence current perceptions of economic risk. However, instability in past income streams represents a realised risk rather than the risk itself (Hacker et al., 2014). Feelings of insecurity in the present are arguably influenced more by the probability of negative events happening in the future than by the experience of economic insecurity in the past (Osberg, 2015; Romaguera-de-la-Cruz, 2020). Using retrospective data originally appears to have been seen as the

[3]For example, it may be difficult to translate words such as 'anxious' or 'worried' into different languages; the words may have different nuances of meaning in other languages.

[4]This results in a binary insecurity measure, where x_{it} refers to household income for individual i in time t, defined as:

$$ID_{it} = \begin{cases} 1 & \text{if } x_{it} < 0.75 \times x_{it-1} \text{ and } x_{it} < \bar{x}_{it} \\ 0 & \text{otherwise} \end{cases}$$

[5]The Bossert and D'Ambrosio (2013) index was adapted by Bossert et al. (2019) to use income instead of wealth and exclude the buffer stock of wealth, and is defined as:

$$I^T(x) = l_0 \sum_{t \in \{1,\dots,T\}: x_{-t} > x_{-(t-1)}} \delta^{t-1}(x_{-t} - x_{-(t-1)}) + g_0 \sum_{t \in \{1,\dots,T\}: x_{-t} < x_{-(t-1)}} \delta^{t-1}(x_{-t} - x_{-(t-1)})$$

l_0 and g_0 refer to losses and gains respectively, x refers to income and δ is a discount factor.

only viable option, but a handful of more recent papers have attempted to develop forward-looking measures based on predicted probabilities of negative future states such as unemployment or large drops in income (Rohde et al., 2015). Furthermore, measurement error in income may result in measurement error in economic insecurity measures based on past income streams. This may be compounded in a developing country context where income has been shown to be measured with considerable error (Burger et al., 2016). For these reasons, in this paper we use a series of forward-looking measures of economic insecurity based on predicted probabilities of negative future states. This approach also corresponds most closely with the vulnerability literature (see e.g. Dercon, 2006; Schotte et al., 2018).

4.2.3 Economic insecurity and BMI or obesity

On the whole, the relatively small literature to date suggests an effect of economic insecurity on BMI, obesity and weight gain, but the results are not always significant for men and women or across other subgroups. In a cross-country study of 11 developed countries, Offer et al. (2010) find that the IEWB economic security index (Osberg & Sharpe, 2002) has a large and consistently negative association with obesity rates for both genders (i.e. higher economic insecurity is associated with higher obesity). Furthermore, economic security was a much stronger predictor of obesity rates than what they call the 'fast-food shock' – the increasing accessibility and fall in relative prices of fast foods and energy-dense processed foods – with an effect nearly three times as large as the food shock. At the demographic group level, Smith et al. (2017) find significant effects of the Hacker et al. (2014) economic insecurity index on obesity rates in the US, and that increased economic insecurity over the period 1988 to 2012 explained a significant proportion of the rise in obesity at the national level.

Studies at the individual level are more numerous, but the measures of economic insecurity used are fairly ad hoc, based on what could be constructed with available data, and are not consistent across studies. In the well-known Whitehall II study, the threat of job loss, which can be seen as a facet of economic insecurity, increased BMI among British civil servants in a department facing privatisation (Ferrie et al., 1998). Smith et al. (2009) find that economic insecurity, measured by the probability of unemployment, income volatility, the number of income drops experienced, and the probability of poverty, led to weight gain in US men. Rohde et al. (2017a) find consistently positive effects of lagged insecurity on BMI in Australia, using an index composed of income volatility and subjective measures of job security, financial satisfaction and ability to raise emergency funds. Using the predicted probability of a 25 percent or greater drop in household income, Watson (2018) finds that insecurity significantly increases BMI for men, but has no significant effect for women in Canada. Watson et al. (2016) find a significantly greater change in BMI between survey waves for men with high school education or less who became unemployed after a Canadian policy change that reduced unemployment benefits, which would be expected to have caused an exogenous increase in economic insecurity. No significant effects were found for females or men with higher education levels. On the other hand, Watson et al. (2020) find that the Great Recession increased the effects of becoming economically insecure for Canadian women and older men. Becoming insecure is argued to be more stressful during a major recession than becoming insecure during non-recessionary times. Women and men aged 45-64 who became economically insecure (became unemployed or experienced a decrease in subjectively-rated job security) during the Great Recession experienced a significantly larger increase in BMI than those who became insecure prior to the Great Recession. Staudigel (2016) uses income volatility, the probability of falling into poverty, the number of years with unpaid bills, and the number of years with wage arrears as measures of insecurity in Russia, finding mixed results. Insecurity was found to increase consumption of sugary foods, but decrease the proportion of fat in energy intake. Insecurity decreased weight gain between 1994 and 2005 for women, though results were not significant across all insecurity measures. For men, some variables had a positive effect on weight gain, but results were insignificant or had the opposite sign for some variables. The author suggests that these mixed results may be due to relatively low availability of energy-dense foods in Russia during the transition period.

It is important to distinguish between the effects of income and economic insecurity. While income and economic insecurity tend to be correlated, the two are conceptually distinct. Furthermore, any effects may not work in the same direction. As discussed in Section 4.2.2, several of the measures of insecurity proposed in the literature are based on realised income streams, though insecurity has been conceptualised as the anxiety concerning potential future shocks rather than realised shocks. A realised negative income shock reduces available income for consumption, as well as potentially inducing anxiety. This blurs the lines between insecurity and income. The effect of a negative income shock could be divided into two effects: the direct effect of reduced income, and a psychological effect. A similar issue exists if using a measure such as job loss to get at the effect of economic insecurity – job loss involves a loss of income (in the absence of a social security system or insurance that fully replaces income) as well as a potential psychological effect. It is possible that the income and psychological effects work in opposite directions. Similarly, income and economic insecurity may work in opposite directions, a possibility raised by Smith et al. (2009). A lower income in itself may lower money available for food consumption and thus lower BMI. The stress of economic insecurity may, on the other hand, increase BMI. Most of the studies outlined above control for income, attempting to separate out the effects of economic insecurity from those of income. This suggests that when significant effects of economic insecurity are found, they are not being driven by a correlation between income and insecurity: they are not merely picking up an effect of low income or poverty. Rohde et al. (2017a) find that when controlling for both economic insecurity and income, insecurity is a significant predictor of BMI while income is not. Smith et al. (2017) find a significantly positive but concave relationship between income and obesity for US men, while economic insecurity is also positively related to obesity rates. This suggests that except at higher levels of income, the effects of income and economic security may move in opposite directions. Smith et al. (2009) find that income is insignificant across most of their specifications when also controlling for insecurity.

The evidence outlined above comes from a handful of developed countries, and Russia in transition. As economic insecurity is strongly correlated with low income, some of these authors note that greater economic insecurity among the poor may help to explain the higher obesity rates observed among the poor in developed countries. It is possible that the role, if any, of economic insecurity in obesity may differ in developing countries. In developed countries, even the poor are comparatively unlikely to suffer from hunger to the extent that they cannot obtain their daily calorie requirements. In a developing country context, on the other hand, a fairly large segment of the population may struggle to meet their calorie requirements. It is possible then that economic insecurity may only have an effect on weight outcomes above a certain level of income: one that allows households to obtain their daily energy requirements. Only once households can afford to consume enough calories to meet their energy requirements can individuals overeat as a self-medicating coping response to stress. The idea that economic insecurity may only lead to weight gain and obesity above a certain level of income is implied by the economic insecurity hypothesis outlined above. Wisman and Capehart (2010) note that stress in the absence of the availability of energy-dense foods does not lead to weight gain in mice. Neither chronic stress nor a high-sugar, high-fat diet on its own led to significant weight gain, but only a combination of the two. The same would be expected in humans. Stress, and economic insecurity, would only be expected to lead to weight gain in the presence of the availability of surplus calories, and particularly energy-dense foods. In light of this, it is likely that in a developing country with a high level of poverty, economic insecurity may have little effect on weight outcomes for a fairly large segment of the population. Insecurity may even decrease BMI or obesity if it results in these households cutting back on food consumption. On the other hand, in a developing country with high levels of inequality such as South Africa, parts of the population have similar access to energy-dense processed foods and excess calories to those in developed countries. For these individuals we may see the association between economic insecurity and higher body weight observed in many developed country studies. We would thus expect to see the relationship between economic insecurity and body weight observed in some developed country studies only for households with a sufficient income to meet their energy requirements.

In light of the issues raised above, this chapter explores whether economic insecurity in South Africa is only associated with higher body weight among higher-income individuals, who are likely to have greater access to excess calories.

4.3 Methods

We use all five waves of the nationally representative National Income Dynamics Study (NIDS) data, collected between 2008 and 2017 (Southern Africa Labour and Development Research Unit, 2018). NIDS contains anthropometric data, data on household income and expenditure, as well as adult (aged 15 and above) household members' employment status[6].

NIDS includes measures of the height, weight and waist circumference of adult respondents. Our primary measure of adiposity is BMI, which is defined as weight in kilograms divided by the square of height in metres. The procedure used to clean the height and weight data is described in Appendix A.2. To reduce the influence of individuals with extremely high or low BMIs, BMI is winsorised at the first and 99th percentiles, for males and females separately. We also use a binary indicator for being obese (a BMI of 30 or above) as a robustness check (results are available in

[6] NIDS also included questions asking respondents to rate the position of their household on a six-step ladder; today, in two years' time, and in five years' time. Expected downward mobility on the ladder could be used as an indicator of subjective economic insecurity, and initial analyses explored using this and variations thereof as insecurity measures. However, as reported by Burns (2009), expectations of mobility were overwhelmingly positive, with very few households expecting to move downward on the ladder. In our sample less than two percent of respondents expected to move downward. These measures were therefore excluded from further analysis.

Appendix C.2).

This paper uses objective forward-looking measures of economic insecurity, namely the predicted probability of unemployment and of poverty in the following wave. Because BMI is slow-changing and backward-looking measures of insecurity are based on past income streams, backward-looking measures may result in a spurious correlation between insecurity and BMI: a correlation between BMI and insecurity may result from a correlation between current BMI and income in previous periods. We therefore opt to use forward-looking measures of economic insecurity in this paper. Forward-looking measures are also closer to the conception of economic insecurity as anxiety regarding future adverse events as outlined in Section 4.2.1. Using the predicted probability of falling into poverty also corresponds with the approach taken by the vulnerability literature, as for example by Schotte et al. (2018).

The probability of experiencing a large income drop in future was initially included as an additional measure. This measure has been found to be associated with body weight (Watson, 2018). However, because of the unreliability of income data, insecurity measures based on income streams could be problematic in the South African context and the developing country context more broadly. Burger et al. (2016) find that roughly 20 percent of the variation in reported household income in NIDS is due to measurement error, leading to the extent of income mobility being overestimated. Measures based on income volatility, as used in some studies (Rohde et al., 2017a; Smith et al., 2009; Staudigel, 2016), are subject to the same potential issues. Measures based on the predicted probabilities of poverty or unemployment may be a better option in the developing country context, as these may be less prone to measurement error[7]. Furthermore, the pseudo R-squared in the probit regression predicting an income drop (see Table C.1.1 in Appendix C.1) was very low (0.02). This measure was therefore excluded from subsequent analyses.

Individuals are defined as being unemployed according to the broad definition of unemployment (including discouraged workers). Households are defined as poor if their per capita household expenditure falls below the Stats SA poverty line. For the sake of robustness we use both the food and upper-bound poverty lines. We predict the probability of unemployment and of being poor in the following wave using a series of probit regressions (available in Appendix C.1). Unemployment

[7]Household expenditure data, on which poverty classification is based, is also subject to measurement error, but measurement error is likely to be less of a problem when considering wellbeing at a single point in time than when looking at changes across time

in the following wave is predicted using current employment status, education (less than primary, primary, incomplete secondary, matric or tertiary, with no schooling as the reference category), employment type if employed (employee, self-employed, casual or subsistence), and indicators for being a union member, for having a permanent contract, and for being formally employed. The labour market variables are defined similarly to those in Schotte et al. (2018). Following Schotte et al. (2018), formal employment is defined for employees as having a written employment agreement, medical aid deduction from one's salary, or a pension or provident fund deduction from one's salary. Self-employed individuals are defined as formally employed if their businesses are registered for income tax and/or VAT. Casual and subsistence work is defined as informal. Additionally, we include the share of public sector employment in the individual's sector of employment as a predictor. The probability of future poverty is based on household expenditure, and is therefore predicted using the above characteristics of the household head. All household members therefore have the same probability of future poverty, while each household member may have a different value for the probability of future unemployment. All regressions also include controls for gender, race group, number of employed household members, number of children in the household, whether the household receives grants, area type (traditional area, urban or farms), province, and an indicator for the survey wave.

Some international studies use the predicted probability of unemployment only for those who are currently employed (see e.g. Rohde et al., 2017b; Smith et al., 2009). However, because of South Africa's high rates of unemployment, this would result in this measure being available only for the employed, leaving out a large proportion of South Africa's population. We therefore opt to predict the probability of unemployment and poverty respectively regardless of current unemployment or poverty status.

We assess whether these measures of economic insecurity are related to BMI using a series of linear regressions. BMI is regressed separately on each of the economic insecurity measures, as well as on a series of controls: income and its square, age and age squared, education (less than matric, matric or post-school), race group, province, and indicators for being married or cohabiting, being a smoker, and exercising weekly. Household income is deflated to March 2017 values using the deflator files provided with NIDS. It is then divided by the square root of household size to adjust for economies of scale within the household (OECD, 2013). We apply the inverse hyperbolic sine transformation, which is similar to a logarithmic transformation, but allows for the inclusion of zero values (Bellemare & Wichman, 2020). All regressions also include indicators for the survey wave. For the sake of comparability, the sample is restricted to observations with complete data for all insecurity measures. All regressions are weighted using the post-stratified weights provided with NIDS, and clustered to account for the fact that repeated observations on the same individual would be more similar than observations drawn from a random sample of different individuals. The standard errors have been bootstrapped using a cluster bootstrap to account for the fact that the predicted probabilities of unemployment and poverty are generated regressors. The sample for this analysis is limited to adults aged 25-64. Restricting the sample to those aged 25-64 helps to avoid issues around employment instability among youth and partly avoids picking up fluctuations due to retirement among older individuals (Watson et al., 2016). The sample size of older adults is also relatively small, so excluding older adults helps to avoid sample size issues.

We explore whether the association between economic insecurity and BMI differs by level of income by interacting our insecurity measures with transformed household income. We illustrate these interactions by plotting the marginal effects of our insecurity measures across levels of household income.

4.4 Results

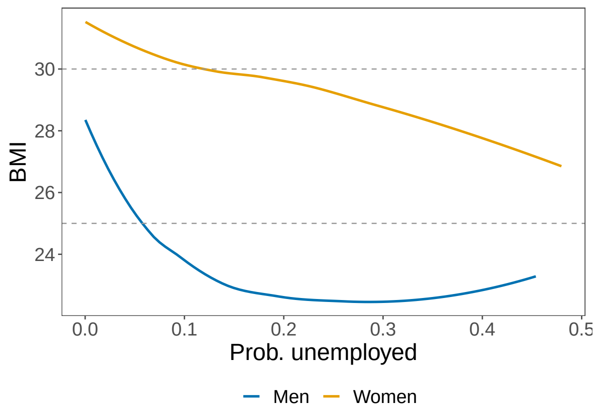
4.4.1 Descriptive statistics

Figure 4.1 shows non-parametric loess regressions of BMI on the economic insecurity measures. BMI is negatively related to economic insecurity. For women, BMI decreases with a higher probability of future unemployment and a higher probability of future poverty (for both the food poverty and upper-bound poverty lines). Similarly, for men BMI decreases with a higher probability of future poverty, at least up to a certain point, and decreases with the probability of future unemployment initially before increasing slightly at higher future unemployment probabilities. The relationship between economic *security* and BMI is thus similar to that between current income and BMI, as found in Chapters 2 and 3 and for example by Wittenberg (2013): BMI increases with current income and with economic *security*, or a lower probability of unemployment or poverty.

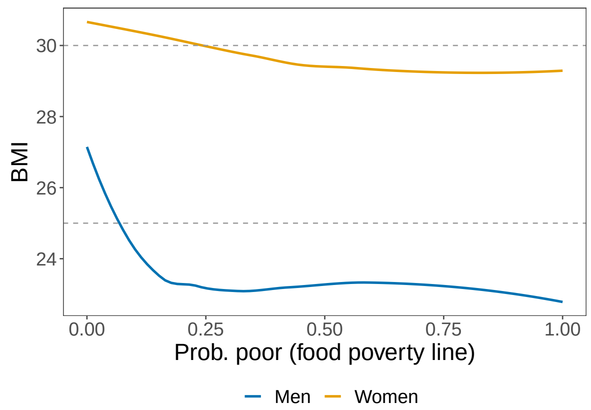
Furthermore, income and economic *in*security are negatively correlated, as shown by the correlations in Table C.1.2 in Appendix C.1, and the measures decline monotonically with income, as shown in non-parametric loess regressions of the insecurity measures on income in Figure C.1.1 in Appendix C.1. The fact that income and the economic insecurity measures are correlated raises the question: are economic *security* and income merely acting as alternative measures of economic wellbeing? However, while the insecurity measures are fairly strongly correlated with income, as shown in Table C.1.2, the correlations are not perfect (they range from -0.49 to -0.68). Furthermore, the variance inflation factors (VIFs) on the insecurity measures from regressions controlling for income and other covariates without interactions (regressions shown in Tables C.4.1 and C.4.2 in Appendix C.4), as shown in Table C.4.3 in Appendix C.4, are fairly low (all below 5). This suggests that our economic insecurity measures and income are not completely interchangeable measures of the same phenomenon or of economic wellbeing more generally.

4.4.2 Interactions between economic insecurity and income

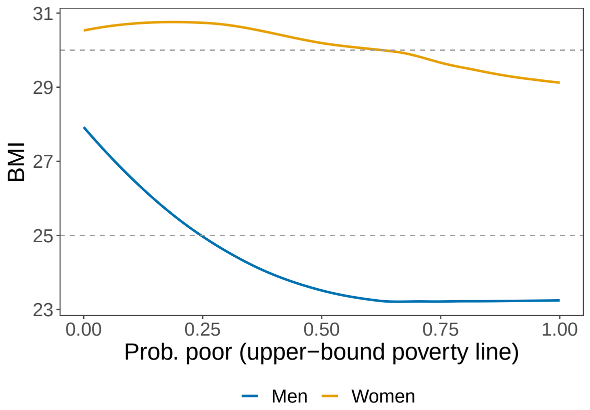
We move next to the question of whether a stronger association between economic insecurity and BMI exists for individuals with higher incomes, who would be expected to have greater access to excess calories. Tables 4.1 and 4.2 show the results of regressions interacting the insecurity measures with income and income squared, and Figure 4.2 illustrates these interactions by plotting the marginal effects of each insecurity measure by income level. The interactions between income and the insecurity measures are significant for all three measures for both men and women, and the negative sign on the income interaction term and positive sign on the income squared interaction term suggest a U-shaped association between income and the marginal effects of the insecurity measures. The marginal effects initially decline with income, which is puzzling. A possible reason for the positive marginal effects at very low levels of income is that low-wage employment is more likely to involve intense physical activity, and therefore at lower levels of income a loss of employment is more likely to involve a reduction in physical activity on the job. Male workers in particular may be in unstable seasonal jobs that involve intense physical activity. This may offset any BMI-increasing effects of insecurity. For women, the marginal effects increase and become significantly positive at higher levels of income. For men, the marginal effects of the future poverty measures are negative in the middle of the income distribution, but insignificant at higher income levels. For the unemployment measure, the marginal effects continue to decline with income, becoming more negative at higher income levels. The association between economic insecurity and lower BMI



(a) Unemployment



(b) Poverty (food)



(c) Poverty (upper-bound)

Figure 4.1: Loess regressions of BMI on economic insecurity measures
Note: Dashed lines indicate overweight and obesity cutoffs.

for men at some parts of the income distribution may be because of an inability to smooth food consumption in the face of potential shocks.

Results for obesity are shown in Appendix C.2, and for women are similar to those for BMI. For men, the interactions between income and the economic insecurity measures are not significant in the obesity regressions.

Appendix C.4 shows the results of regressions of BMI on the insecurity measures without interactions. For women, none of the insecurity measures are significantly associated with BMI overall, while for men all three measures are negatively associated with BMI. Results for systolic and diastolic blood pressure as well as an indicator for having high blood pressure (having a systolic or diastolic reading above their respective cut-offs, or being on medication for high blood pressure) are available in Section C.3 in the appendix. The interaction between income and the insecurity measures is positive and significant for systolic blood pressure and the high blood pressure indicator for women, and for diastolic blood pressure at the 10 percent level. It is generally not significant for men. As hypertension is known to be associated with stress, these results support the idea that economic insecurity may operate through stress.

Results for regressions interacting the insecurity measures with an indicator for urban residence are shown in Appendix C.6. The probability of future poverty is more positively associated with BMI in urban than in rural areas, but the marginal effects plots show that the marginal effects of the insecurity measures are not significantly different from zero in urban areas.

4.4.3 Robustness checks

4.4.3.1 Negative household events and job loss

As a robustness check, we explore the interactions between household income and negative events experienced in the previous 24 months. Negative household events were classified into seven categories, defined following Burger et al. (2017): death of a non-resident relative or friend who provided financial assistance, serious illness or injury of a household member, a negative agricultural event (major crop failure or widespread death and/or disease of livestock), a negative employment event (reduction in work hours or job loss of person providing financial assistance), a negative transfer event (a cut-off or decrease in remittance or grant income), a loss of property event (theft, fire or destruction of household property), and the death of a resident household member. Addition-

Table 4.1: Regressions of BMI on insecurity measures with interactions: women

	(1)	(2)	(3)
Prob. unemployed	181.97***		
	(46.01)		
Prob. unemployed x Income	−42.34***		
	(10.81)		
Prob. unemployed x income sq.	2.44***		
	(0.63)		
Prob. poverty (upper-bound)		65.24***	
		(18.88)	
Prob. poverty (upper-bound) x Income		−15.38***	
		(4.20)	
Prob. poverty (upper-bound) x income sq.		0.90***	
		(0.24)	
Prob. poverty (food)			53.26**
			(23.16)
Prob. poverty (food) x Income			−13.04**
			(5.50)
Prob. poverty (food) x income sq.			0.79**
			(0.33)
Income	7.16***	10.16***	5.17**
	(2.05)	(3.05)	(2.02)
Income sq.	−0.37***	−0.54***	−0.26**
	(0.12)	(0.17)	(0.12)
Urban	0.97***	0.95***	0.89***
	(0.29)	(0.30)	(0.30)
Constant	−18.45*	−31.36**	−8.49
	(9.45)	(14.24)	(9.23)
Observations	16307	16307	16307
Adjusted R^2	0.09	0.10	0.09

* p <0.1, ** p <0.05, *** p <0.01. Standard errors in parentheses.

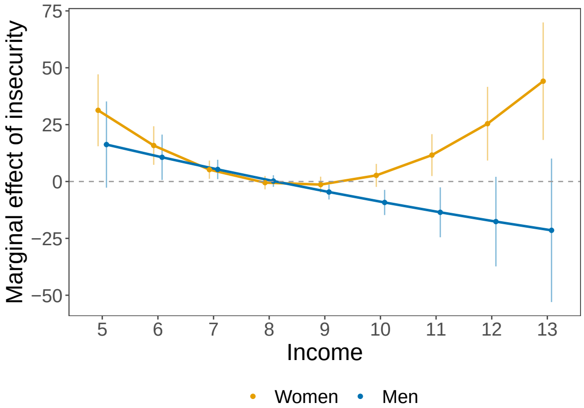
Note: Bootstrapped standard errors clustered by individual. It was not possible to bootstrap the standard errors for the food poverty results. Bootstrapped standard errors are shown for the upper-bound poverty and unemployment results, but for these variables the conclusions did not change when using bootstrapped standard errors rather than uncorrected standard errors. Controls are age, race group, education, employed, married/cohabiting, province, smoker, exercises weekly, and ever given birth.

Table 4.2: Regressions of BMI on insecurity measures with interactions: men

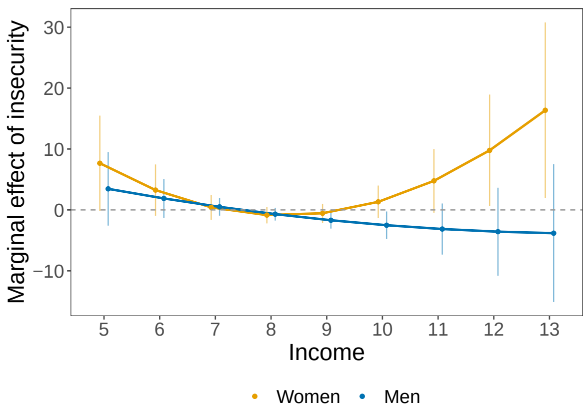
	(1)	(2)	(3)
Prob. unemployed	48.00		
	(55.64)		
Prob. unemployed x Income	−6.98		
	(13.07)		
Prob. unemployed x income sq.	0.13		
	(0.77)		
Prob. poverty (upper-bound)		23.52	
		(17.38)	
Prob. poverty (upper-bound) x Income		−5.16	
		(4.10)	
Prob. poverty (upper-bound) x income sq.		0.27	
		(0.24)	
Prob. poverty (food)			14.17
			(18.13)
Prob. poverty (food) x Income			−2.62
			(4.31)
Prob. poverty (food) x income sq.			0.09
			(0.26)
Income	1.12	1.21	−0.52
	(2.31)	(2.20)	(1.67)
Income sq.	0.03	0.01	0.10
	(0.13)	(0.12)	(0.10)
Urban	0.37*	0.26	0.29
	(0.22)	(0.23)	(0.23)
Constant	9.81	10.75	18.42**
	(10.21)	(9.62)	(7.41)
Observations	10294	10294	10294
Adjusted R^2	0.25	0.25	0.24

* p <0.1, ** p <0.05, *** p <0.01. Standard errors in parentheses.

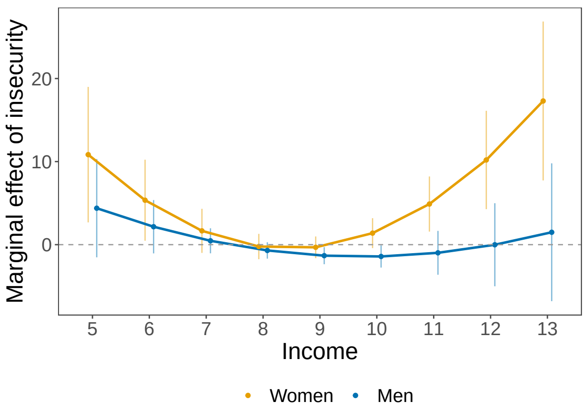
Note: Bootstrapped standard errors clustered by individual. It was not possible to bootstrap the standard errors for the food poverty results. Bootstrapped standard errors are shown for the upper-bound poverty and unemployment results, but for these variables the conclusions did not change when using bootstrapped standard errors rather than uncorrected standard errors. Controls are age, race group, education, employed, married/cohabiting, province, smoker, and exercises weekly.



(a) Unemployment



Poverty (food)



(c) Poverty (upper-bound)

Figure 4.2: Marginal effects of economic insecurity measures on BMI by income level

ally, we include becoming unemployed since the previous wave (using both the broad and narrow definitions of unemployment), defined as being employed in the previous wave and unemployed in the current wave. These negative events are all backward-looking rather than forward-looking measures, reflecting realised shocks rather than potential future risks, and do not all necessarily reflect economic insecurity as defined previously. However, these are all events that are likely to involve significant stress, financial or otherwise.

Results are available in Section C.5.1 in Appendix C.5. For women, the death of a household member, death of a non-resident friend or family member who provided financial assistance, a negative agricultural event (at the 10 percent level), and becoming unemployed since the previous wave all showed a significant positive interaction with income. The interactions were not significant for the serious illness or injury of a household member, a negative employment event, a negative transfer event, or a loss of property event. For men, the interactions were either negative or insignificant. These results reflect the general patterns of those using our insecurity measures: the marginal effects of these stressful life events on BMI tend to be negative or insignificant for men, and for women at lower levels of the income distribution, but positive for women at higher levels of income.

4.4.3.2 Alternative definitions of unemployment

The results in the previous section use the predicted probability of unemployment defined according to the broad definition of unemployment, which includes people not actively searching for a job. If individuals are voluntarily unemployed, they may not necessarily be worse off economically than individuals in precarious employment. We therefore test the robustness of the results to using the predicted probability of unemployment defined according to the narrow definition of unemployment, as well as to predicting the probability of becoming unemployed (using the broad and narrow definitions) only for those who are currently employed. The results are similar to the results for the main predicted probability of unemployment measure, with the marginal effects showing a U-shaped relationship with income for women, and a generally declining relationship with income for men.

4.4.3.3 Limiting the sample to individuals aged below 60

Including individuals up to and including age 64 in the sample implies that some of these individuals may already be receiving the older person's grant (old-age pension), for which individuals become eligible at age 60. We test the robustness of the results to excluding these individuals by estimating the main results for individuals aged below 60. Results are shown in Table C.5.4, and are similar to the main results.

4.5 Discussion

Though the results for the future unemployment measure were conflicting, taken together the results suggest that the positive association between economic insecurity and BMI observed in a number of developed country studies is on the whole more likely to be present in South Africa among high-income women. While on the whole there tends to be no association between economic insecurity and BMI for women with low to average incomes, for women at the upper end of the income distribution economic insecurity is positively associated with BMI. The results for women are on the whole consistent with our hypothesis – that economic insecurity would be positively associated with BMI at levels of income that are sufficient to obtain excess energy. For men our results do not support this hypothesis: if anything, economic insecurity is negatively associated with BMI among men.

The fact that in South Africa we are more likely to see the positive association between economic insecurity and BMI observed in many developed countries among high-income women may be because a fairly large proportion of South Africans are still at stage 3 of the nutrition transition as described by Popkin and Gordon-Larsen (2004) and Popkin (2006), unable to obtain enough calories to meet their energy requirements, let alone to consume excess calories. Despite high rates of overweight and obesity in SA, hunger is still prevalent. Close to 40 percent of South Africans report that their household ran out of money to buy food the previous month, while close to 20 percent report that someone in their household went hungry in the past week because there was not enough food (Van der Berg et al., 2021). In these circumstances, economic insecurity may even be associated with reduced BMI for those who are unable to smooth food consumption in the face of potential shocks. This may explain the negative association between economic insecurity and BMI among men on aggregate, driven by men in the middle of the income distribution.

The burgeoning literature on economic insecurity has found that economic insecurity is in many cases associated with worse mental and physical health, including higher BMI and an increased risk of obesity. However, this literature comes almost exclusively from developed countries. This paper has shown that economic insecurity does not have the same association with BMI as that generally observed in developed countries, highlighting the need for more research on the health and other effects of economic insecurity in developing countries. However, the lack of consensus on how best to measure economic insecurity, even in developed countries, and the difficulties in applying measures proposed in the developed country context to developing countries is a barrier. The developing country literature has focused on vulnerability to poverty (see e.g. Dercon, 2006; Schotte et al., 2018) and there has been little linkage between the developed country literature on economic insecurity and the developing country vulnerability literature (Osberg, 2010). This study suggests that economic insecurity may have health consequences for the relatively well-off, even if they are at relatively low risk of falling into poverty. It is an open question whether economic insecurity or vulnerability to poverty is the more appropriate concept in developing countries. This is an area ripe for further conceptual and methodological work.

Aside from the lack of consensus and difficulties in measuring economic insecurity, a limitation of this paper is that it cannot identify a causal association between economic insecurity and BMI. Unobserved individual characteristics may play a role in generating both economic insecurity and obesity. A bidirectional relationship is also possible; those with higher BMI may face greater economic insecurity due to health issues associated with obesity or due to labour market discrimination. Indeed, Henry and Kollamparambil (2017) found evidence of discrimination against obese individuals in both the probability of employment and wages in South Africa, particularly for women. While this paper has taken a two-stage regression approach in predicting the probabilities of future poverty and unemployment in a first stage and then entering these predicted probabilities into regressions predicting BMI as a second stage, this is not a traditional two-stage least squares instrumental variables approach. We could not identify any instrumental variables that influence BMI only through their effect on economic insecurity. This approach therefore does not allow us to infer causality. The instrumental variables approach has been used in the international economic insecurity literature: Smith et al. (2009), Staudigel (2016) and Watson (2018) use state- or region-level unemployment rates and means or medians of economic insecurity measures as instruments for economic insecurity. However, given the high levels of spatial inequality in South Africa, as well as differences in food environments that may be associated with this spatial inequality[8], the crucial assumption that these district-level variables would only affect individual BMI through their effect on individual insecurity is dubious in the South African context.

4.6 Conclusion

Economic insecurity has been proposed as a driver of obesity in developed countries, and a number of studies have found an association between economic insecurity and higher BMI or obesity. This relationship may not exist where people do not have access to excess energy or energy-dense processed foods, as is the case for many people in developing countries. We show that this relationship is not in general present in South Africa: indeed, among men our measures of economic insecurity are associated with lower BMI. However, we show that among women economic insecurity tends to be more positively associated with BMI among those with higher incomes, suggesting that for women economic insecurity is more likely to result in higher BMI and contribute to obesity when coupled with increased access to excess calories. This paper also highlights the difficulties in measuring economic insecurity, particularly in a developing country context, and highlights the need for more work on conceptualising and measuring economic insecurity and its effects in developing countries.

[8]For example, the availability of fast food outlets and supermarkets is generally greater in urban centres than in former 'homelands', and unemployment rates tend to be higher in former 'homelands' (Kwenda et al., 2021). Proximity to fast food outlets and supermarkets has been shown to increase overweight and obesity in South Africa (Otterbach et al., 2021). District-level unemployment rates may thus be associated with BMI through their association with food environments, rather than through their impact on economic insecurity, causing the exclusion restriction to be violated.

CHAPTER 5

CONCLUSION

As countries move through the nutrition transition, the burden of obesity tends to shift from the rich to the poor. While the reversal of the socioeconomic gradient in obesity usually occurs at a lower level of economic development than South Africa's, and has already occurred for women in some other upper middle-income countries, the gradient in South Africa remains positive for men and women. This dissertation has explored several aspects of the relationship between SES and obesity in South Africa, investigating several nuances in this relationship that have not previously been explored, and whether there are any signs of this relationship beginning to reverse. Most of the literature, both South African and international, has viewed the nutrition and associated obesity transitions as a process that occurs for the whole population at once (though generally first for women and urban residents).

This dissertation has raised the possibility that there may also be life course or intergenerational and generational aspects to the nutrition and obesity transitions, which are obscured when examining the social gradient in obesity for the entire population. Chapter 2 highlighted life course and intergenerational aspects of the nutrition and obesity transitions. It showed that SES has a different association with obesity at different stages of life: higher adult SES is associated with higher obesity risk, while higher childhood SES is associated with reduced obesity risk. It further showed that, compared to high SES individuals who also came from a high SES childhood background, upwardly socially mobile individuals – those who came from a low SES childhood background but moved to a high SES in adulthood – are at increased risk of obesity. Finally, it showed that the social gradient is closer to reversing for those who came from a high SES childhood background, i.e. for those whose mothers had a high SES. This suggests that the progression to later stages of the nutrition and obesity transitions is also an intergenerational phenomenon, and that more than one generation of high SES is needed before higher SES is associated with reduced obesity risk. Chapter 3 proposed that there may be a generational aspect to the onset and progression of the nutrition and obesity transitions: the transitions may happen first (or only) in younger generations. Using a novel application of a machine learning algorithm, it showed that the social gradient in body weight is flatter in younger generations of South African men, and possibly also in younger generations of women. Chapter 4 explored the interplay between economic insecurity and SES in South Africa, proposing that the positive association between economic insecurity and BMI observed in developed countries would only be seen among those with relatively high income in a developing country such as South Africa. It showed that in South Africa economic insecurity is more positively associated with BMI among high-income women.

The following section discusses the findings of each chapter in greater detail, highlighting the contributions and potential for future research offered by each chapter. Section 5.2 draws out some implications and policy recommendations of this dissertation.

5.1 Summary of findings

5.1.1 Social mobility, childhood SES and the obesity transition: the social gradient in body weight is flatter among individuals with high childhood SES

Chapter 2 investigated the social gradient in BMI across the life course. It examined the interaction between childhood SES, proxied by the individual's mother having completed school (Grade 12 or 'matric'), and current SES among adults. It used random effects within-between models to explore the interaction between childhood SES and adult SES, decomposing adult SES into changes in household income within individuals over time, and between-individual differences in average household income.

It found that upward social mobility increases obesity risk relative to those who retain a high SES throughout life. It showed also that the marginal effect of income is flatter for adults from a high childhood SES background, and for women may already be negative. These results were driven by individuals at the upper end of the income distribution. The social gradient in BMI appears to have become negative for high income women who also experienced high childhood SES, and has flattened out for high income men with high childhood SES. The social gradient in BMI remains positive across the income distribution for adults who experienced low childhood SES. This suggests that the social gradient in BMI is reversing first among high SES individuals who also experienced high SES in early life, or put differently, whose parents also had high SES. This implies that we need to see two generations of sustained high SES before higher SES is associated with reduced BMI: one's mother needs to have had a high SES, and one's own SES in adulthood also needs to be high.

The random effects within-between models show that the association between adult income and BMI, and its interaction with childhood SES, is driven by differences in income between individuals, rather than changes in income within an individual's life. This suggests that the SES-BMI association may be driven more by long-run factors related to SES than short-run changes. BMI is slow-changing, and may be more influenced by social mobility over the entire life course than by very short-run mobility. However, it must be noted that at most nine years of data are available in NIDS, which may be behind the lack of significant associations for changes in income within individuals – changes in income over a longer time period may be more likely to be significant.

A further finding of interest is that high childhood SES is associated with lower adult BMI, even in a context where higher adult SES is associated with higher BMI, suggesting that in this context the effects of SES are not cumulative over the whole life course. This is in contrast to most of the international literature, where adult SES and childhood SES work in the same direction.

Furthermore, this association was only present in urban areas. This may be related to the nutrition transition being more advanced in urban areas. Childhood SES is associated with lower adult obesity risk where obesity risk is highest: where cheap energy-dense processed foods are most readily available. This interpretation is supported by the more negative association between childhood SES and BMI for those with higher adult incomes (i.e. the negative interaction between childhood SES and adult income): childhood SES is most strongly associated with a lower adult obesity risk for those who are most able to afford excess calories, putting them at greater risk of obesity.

This study is the first to explore the relationship between body weight and either childhood SES or social mobility in South Africa using a nationally representative dataset. Indeed, it is one of the few studies on this topic internationally to use nationally representative data. A couple of previous studies (Case & Menendez, 2009; Ginsburg et al., 2013) have explored related issues, but used small urban samples, and in the latter case only examined body weight in adolescence. This study is also the first to explicitly link childhood SES and social mobility to the nutrition transition and the obesity transition, specifically the process of reversal of the (adult) SES gradient in obesity. It is also the first study to explore whether these relationships differ between urban and rural areas, which may be at different stages of the nutrition transition. This study also strengthens the methodological applications typically used in this literature by decomposing the SES gradient into differences between individuals and changes within individuals over time using random effects within-between models, a method that has not to my knowledge been applied to the study of the social gradient in body weight. Finally, in contrast to almost all of the literature, which is almost entirely from developed countries, we look at these relationships in a context where the association between adult SES and BMI is still mainly positive. This is thus the first study of which I am aware to show an association between childhood SES and BMI in the opposite direction to that for adult SES.

This study relies on individuals' recall of their parents' education, which may be unreliable. It would be useful for future research to explore these issues using more reliable and more nuanced measures of childhood SES, preferably not based on recall. An example would be using cohort data where SES is measured in childhood. Studies using cohort data would also be able to examine the relationship between adult BMI and SES at specific points in childhood, to see whether certain periods in childhood are particularly sensitive for the development of adult obesity. It would also be interesting to explore at what point in childhood or adolescence the association between SES and adult BMI becomes positive rather than negative. Furthermore, this study could not show a causal impact of childhood SES and social mobility on BMI. Future research attempting to estimate the causal impact of childhood SES and social mobility on adult BMI, for example using instruments for parents' education, would be valuable. Finally, this study attempted to separate the adult SES gradient into between-individual differences in adult income and changes in income within an individual's adult life. However, this exercise was limited by the fact that NIDS covers at most nine years. Panel data covering a longer period would allow for a deeper exploration of life course dimensions of the social gradient in BMI. Apart from small cohort studies, long-run longitudinal data are rare in most countries – and especially so for nationally representative samples. Despite its shortcomings, using anthropometric data in the NIDS is exceptional in balancing long-run analysis with adequate population-level granularity.

5.1.2 Generational change in the social gradient of obesity risk: the gradient may be flatter in younger generations

Chapter 3 raises the possibility that looking at the social gradient in body weight at one point in time obscures generational differences in this relationship, and that the shift towards a negative gradient may already be happening for younger generations. Instead of arbitrarily splitting the sample into older and younger cohorts, it used the model-based recursive partitioning algorithm to find structural breaks in the social gradient in BMI by birth year. It then interacted the birth year split chosen by the algorithm with household income to test whether the social gradient differs significantly in younger cohorts. It found that the SES gradient in BMI is flatter among younger cohorts of men. For women, the SES gradient in BMI was not significantly different in younger cohorts in the whole sample, but when looking at obesity rather than BMI there were indications of a flatter gradient for younger cohorts. There were also indications of a flatter SES gradient in BMI in younger cohorts among urban African women. There is thus some evidence for our hypothesis that the association between SES and BMI may be weaker in younger cohorts.

This is the first study of which I am aware to apply the model-based recursive partitioning algorithm in an age-period-cohort analysis. Though this analysis would also have benefitted from data covering a longer time period, this study illustrates the potential of the model-based recursive partitioning algorithm for detecting changing stages of the obesity transition. More broadly, this study is an example of how machine learning techniques can be used in explanatory or descriptive[1] analyses. Conceptually, this study introduces a new way of thinking about the nutrition and obesity transitions. It is the first study of which I am aware to raise the possibility that shifts in the socioeconomic patterning of obesity prevalence may happen at different starting points for various generations rather than only across time. It removes the emphasis from macro-level determinants of the obesity transition, and looks at factors that relate to specific groups. In particular, perceptions of body weight and attitudes to food consumption and health awareness may be generation-specific determinants of obesity. As consecutive younger generations adopt new norms early in their lives, the population-level momentum to move to a later stage of the obesity transition accelerates, even if older generations do not make the full shift.

While this study does control for age, it is notoriously difficult to convincingly disentangle age, cohort and period effects. This study provides tentative evidence that the social gradient in BMI is weaker in younger cohorts, but given the relatively short time period covered by NIDS, more research is required to confirm that this is indeed a cohort effect and not an age effect. Panel data covering a longer time period, allowing us to observe each cohort across a wider range of ages, would enable us to better disentangle cohort, age and period effects. Future research could also

[1] In other words where the goal is not to predict an outcome, but rather to study the association between an outcome and specific explanatory variables.

apply novel age-period-cohort identification methods to this question.

5.1.3 Economic insecurity and obesity risk in South Africa: the association between economic insecurity and body weight is dependent on income

Chapter 4 asked whether economic insecurity is more positively associated with increased body weight for those with higher incomes, who would be expected to have greater access to excess calories. Though there were some conflicting results depending on the measure used, it found that the positive association between economic insecurity and higher BMI observed in developed countries is on the whole more likely to be present for higher-income South African women. For men, however, the results were conflicting. The results for women provide evidence for the hypothesis that economic insecurity is more likely to contribute to obesity when excess energy is accessible, but the results for men did not support this hypothesis.

This study is the first to explore whether economic insecurity is more likely to be associated with obesity at higher levels of income, which is a proxy for access to energy-dense food. Other authors have hypothesised that economic insecurity increases body fat *in the presence of energy-dense foods* (Wisman & Capehart, 2010), but none have tested this empirically in humans. The vast majority of people in developed countries have easy access to energy-dense foods and more than enough calories to meet their nutritional needs. This makes a highly unequal developing country such as South Africa an interesting case study, as pockets of the population have access to excess calories while many others go hungry. This heterogeneity provides support for the external validity of the hypothesis that the availability of unhealthy foods in high SES settings is a primary mechanism for driving the transition into obesity. As Chapter 2 shows, the path out of obesity requires reinforcing the benefits of favourable economic circumstances sustained over more than one generation to provide long-run economic stability that counters the short-run health disadvantages resulting from improving SES. This is also the first study to explore economic insecurity and its effects on health outcomes in South Africa.

This study highlights the potential for research on the effects of economic insecurity on other health outcomes and other aspects of wellbeing in South Africa and other developing countries. Despite a burgeoning international literature on economic insecurity, it has not been studied in South Africa, and has been very little studied in other developing countries. The developing country literature has tended to focus specifically on vulnerability to poverty (see e.g. Dercon, 2006; Schotte et al., 2018). However, this study suggests that economic insecurity may have health consequences for the relatively well-off, even if they are at relatively low risk of falling into poverty. It is an open question whether economic insecurity or vulnerability to poverty is the more appropriate concept in developing countries. Future research could explore how the class schema developed by Schotte et al. (2018), based on the probabilities of entering or exiting poverty, relates to the measures of economic insecurity proposed in the international literature, and how class membership relates to BMI.

This study also highlights the need for further consideration of how best to measure economic insecurity in developing countries. Measures used in the international literature were adapted for the South African context: due to the high rates of unemployment and poverty in South Africa, it opted to use the probability of unemployment and of poverty rather than the probability of becoming unemployed or falling into poverty, which would have resulted in these measures being unavailable for those who were currently unemployed or poor. However, whether measures of economic insecurity created for developed countries are appropriate for developing countries is an area ripe for further conceptual and methodological discussion. Lastly, this study could not show whether economic insecurity has a causal effect on BMI. Even in the international literature, there are relatively few studies on the causal effects of economic insecurity. This is an area that invites future research.

5.2 Implications and policy recommendations

These findings have important implications. They highlight that the transition to later stages of the obesity transition, where we see a levelling out and eventually a reduction in obesity rates, occurs slowly. The wealthiest parts of society take two generations; it is not yet possible to observe the speed of this transition for the poor and economically immobile. Alleviating the frictions that keep large populations stuck in a stage of the obesity transition where improved living conditions are likely to come with increases in obesity will require public intervention that goes beyond a reliance on a natural progression to the final stages of the transition.

Although the SES gradient in BMI shows signs of flattening out for younger generations and for those who came from a high SES childhood background, the gradient has not yet reversed for the population as a whole. Furthermore, we do not see the association between economic insecurity and higher BMI observed in developed countries, except for high-income women. Both of these findings may be because many South Africans still do not have access to sufficient resources to meet their nutritional needs. Many South Africans remain in stage 3 of the nutrition transition, and hunger remains a significant problem in South Africa. For example, in recent surveys nearly 40 percent of respondents reported that their household had run out of money to buy food in the previous month, while nearly 20 percent reported that someone in their household had gone hungry in the previous week (Van der Berg et al., 2021).

However, Chapter 2 suggests that the continued prevalence of undernutrition in South Africa may itself pose an obesity risk, particularly when undernutrition is experienced in childhood and is followed by upward mobility. The 'double burden of malnutrition' may occur not only within the country as a whole and within households, but also within individuals across the course of their lives. This adds further urgency to the need to ensure sufficient nutrition for all, particularly in early life. This suggests that policies to combat obesity need to be seen in broad terms: policies encouraging a reduction in calories consumed or an increase in physical activity in adulthood are only part of the puzzle.

This dissertation also emphasises that the process of accumulation of body fat and of shifting socioeconomic patterns of obesity are long-run processes. Chapter 2 suggests that short-run changes in socioeconomic conditions over the course of adulthood may not have much of an effect, while long-run changes such as those between childhood and adulthood may. We may even need to take a multigenerational perspective: we may need to see two generations of sustained high SES before greater resources are associated with decreased obesity risk. Chapter 3 suggested that shifts may happen over generations. This suggests that shifts in the socioeconomic patterning of obesity happen over a long time horizon.

Chapter 2 found that high childhood SES was more strongly associated with lower BMI for those with higher incomes and residents of urban areas, and Chapter 4 found that among women economic insecurity is associated with higher BMI only among those with higher incomes. These results suggest that factors that are associated with increased obesity risk are more important for those with greater access to excess energy. In other words, upward economic mobility has initial disadvantages. For high SES to be protective against obesity requires two generations of reinforcement. This suggests that we need policies to reduce consumption of energy-dense foods, particularly for those who have higher access to these foods. Promoting favourable childhood environments and greater economic security may become more important for containing obesity as access to excess calories and energy-dense foods increases, either through rising incomes, through urbanisation, or through the expansion of Big Food retailers into rural areas. However, we know less of what to do when the multi-generational benefits of high SES are absent, and how to help the urbanising poor to circumvent the transition into obesity.

Looking ahead, the results of this dissertation suggest several speculative conclusions about future trajectories and patterns of obesity in South Africa. Firstly, given that the SES gradient remains positive, we can expect obesity rates to continue to rise if South Africa achieves a sustained increase in living conditions. This is particularly the case if childhood malnutrition remains widespread, and is coupled with improving living standards across the life course. As the SES gradient is largest for those with low childhood SES, we can expect the greatest rises in obesity prevalence with rising incomes among individuals from low-SES backgrounds. Given relatively high rates of child malnutrition in rural areas (Otterbach & Rogan, 2017), we can also expect further increases in obesity rates in rural areas as the Big Food industry expands further into these areas, increasing the availability of energy-dense processed foods. Finally, if the social gradient in BMI is weaker in younger cohorts, as suggested by Chapter 3, we would expect to see the gradient reverse first in younger cohorts, with the burden of obesity shifting increasingly towards the poor among these cohorts. We can also expect that – without intervention – these obesity trends will not resolve within the next two generations. South Africa is unlikely to transition to the final phases of the obesity transition, where we see a plateau or even a reduction in obesity rates, without clear policies to counteract the trajectory. While my conclusions do not directly lend themselves to clear policy actions, I nevertheless present a discussion of possible routes that could be followed to minimise the impacts of changes in SES on obesity. The next section discusses some suggestions for specific policies to address rising obesity rates.

5.2.1 Policies to reduce the consumption of unhealthy foods

Taxes on unhealthy foods could lead to reductions in consumption of these foods, and may induce manufacturers to reformulate their products to reduce their sugar, fat and salt contents. South Africa has already implemented a tax on sugar-sweetened beverages, and there is evidence that the tax may have been effective in reducing sugar and energy intake from these beverages, in large part due to behaviour change and in lesser part due to reformulation of beverages by manufacturers (Essman et al., 2021). South African policymakers could consider taxing other 'junk' foods too. Hungary and Mexico introduced taxes on energy-dense processed foods in 2011 and 2014 respectively; these taxes were associated with small reductions in purchases or consumption of taxed foods (Batis et al., 2016; Bíró, 2015). However, it is worth noting that all three of the above studies were either conducted among low-SES groups (Essman et al., 2021), or found that reductions were concentrated among low-SES groups (Batis et al., 2016; Bíró, 2015). The incidence and socio-economic distribution of the effects of possible 'junk' food taxes requires careful consideration in the South African context.

Another policy that may help to tackle rising obesity rates is to introduce requirements for simplified front-of-pack (FOP) labelling. South African regulations currently require a list of ingredients, but nutritional information is only mandatory when a nutritional claim has been made (Department of Health, 2010; Dlamini et al., 2021), and evidence suggests nutritional information is poorly understood (Jacobs et al., 2011; Mandle et al., 2015). The draft amendment to the regulations relating to the labelling and advertising of foods (R. 429, published in 2014) proposed voluntary simplified FOP labelling according to a 'traffic light' system, where risky nutrient and energy values are labelled in green, yellow or red according to whether they are within ranges considered healthy (Department of Health, 2014), but these regulations have yet to come into law. The South African Department of Health is reportedly considering introducing mandatory FOP labelling for packaged foods, possibly including warning labels on foods high in sugar, saturated fat or salt (Cullinan, 2019). Evidence on the effectiveness of FOP labelling in encouraging healthier food purchases is mixed, though many studies have shown positive results (An et al., 2021). Evidence suggests that simplified labelling schemes such as 'traffic light' systems may be more effective in increasing the selection of healthier food options than more complicated nutritional information (Cecchini & Warin, 2016), and may be better understood among South African consumers (Hutton & Gresse, 2021). A further option is to extend food labelling requirements to fast food and to alcoholic beverages. Many fast food restaurants do not provide any nutritional information, despite many fast food meals exceeding recommended daily fat, sugar and carbohydrate intakes (Dlamini et al., 2021). Alcohol consumption, particularly heavy drinking, is also associated with weight gain (Traversy & Chaput, 2015).

Restrictions on the marketing of 'junk foods' are a further policy option worth considering. The UK is set to implement a ban on the advertising of foods high in fat, salt and sugar online and on television before 9pm from 2023 (Sweney & business correspondent, 2021). Tobacco advertising bans have been implemented in many countries, and have been shown to reduce tobacco consumption (Blecher, 2008; Saffer & Chaloupka, 2000). Given that junk food advertising bans have not yet been widely implemented, evidence on their effectiveness is still limited, but some evidence (mainly based on simulations) suggests they may be effective in reducing purchases of junk food (Chou et al., 2008; Dhar & Baylis, 2011; Dubois et al., 2018).

5.2.2 Policies to improve childhood nutrition

We cannot say whether the association of childhood SES with lower adult obesity in obesogenic environments is due to poor nutrition associated with lower childhood SES (a biological mechanism) or to eating behaviours and preferences associated with lower childhood SES (a behavioural mechanism). The policy implications differ depending on which mechanism is at play, though there are likely to be elements of both.

If the mechanism linking low childhood SES to adult obesity in obesogenic environments is poor childhood nutrition, this calls for policies to improve nutrition during pregnancy and childhood. The first 1000 days of life – from conception to two years – are particularly crucial for child development, including the development of adult obesity and NCD risk (Black et al., 2013). Nutrition education and counselling during pregnancy (Girard & Olude, 2012; Hossain et al., 2017); access to water, sanitation and hygiene (Bridgman & Von Fintel, 2022; Fink et al., 2011); multiple-micronutrient supplementation during pregnancy (Keats et al., 2019); and exclusive breastfeeding (Scherbaum & Srour, 2016) have all been linked to improved maternal and/or child nutrition outcomes. Breastfeeding is also directly associated with a lower risk of obesity and diabetes later in life (Victora et al., 2016). The National Integrated Early Childhood Development (NIECD) Policy adopted in 2015 includes commitments to provide or support these and other essential Early Childhood Development (ECD) services, but gaps in implementation remain (Thorogood et al., 2020). South Africa's Child Support Grant (CSG) has also been found to improve child nutrition (Agüero et al., 2007; Coetzee, 2013). Nonetheless, uptake of the grant – particularly in the first year of life – could be improved, for example through enabling registration for the CSG during pregnancy as recommended in the NIECD Policy (Thorogood et al., 2020).

Beyond the first two years of life, ECD centres play an important role in providing nutrition to children. A subsidy is available to poor children attending registered ECD centres, 40 percent of which is meant to be allocated to food. However, the means test threshold is much lower than that for the CSG, excluding many poor children (Thorogood et al., 2020). The registration requirements for ECD centres are also prohibitive, particularly for ECD centres in poor areas. The majority of children attending ECD centres are attending unregistered centres, and are thus unable to access the subsidy – only 10 percent of children aged 0-5 years receive the subsidy (Thorogood et al., 2020). Less prohibitive ECD centre registration requirements and making all CSG recipient children automatically eligible for the ECD subsidy would allow more children to access the subsidy, helping more centres to provide nutritious meals to young children.

Interventions and policies supporting optimum nutrition in early childhood should be followed up by supportive environments in later childhood to have maximum impact. One way to do this may be through expanding or complementing the school feeding scheme, or National School Nutrition Programme (NSNP). The NSNP already feeds more than nine million of the poorest learners at least one meal daily. One potential option to improve nutrition among children would be to expand the NSNP to include breakfast, as is already done in some provinces (Moncho-Maripane, 2022). The NSNP currently only provides meals on school days – roughly 200 days of the year – leaving learners uncovered for roughly 165 days of the year. Another possible option would be to establish complementary programmes to provide food to children on weekends and school holidays.

While the nutritional pathway linking poor childhood nutrition to adult obesity is well-established, the association between high childhood SES and lower risk of adult obesity in obesogenic environments may be due in part to a behavioural rather than a biological mechanism. Food preferences learned in childhood tend to persist into adulthood (Venter & Harris, 2009). Preliminary evidence suggests a role for a behavioural channel linking scarcity of a food in childhood to higher consumption of that food in adulthood (Adamopoulou et al., 2021), and adults with low childhood SES are more likely to experience problems with regulation of food intake and eat even when not hungry (Hill et al., 2016). Food preferences are important determinants of diets (Allcott et al., 2019; Atkin, 2016; Dubois et al., 2014), and diets do not tend to change significantly (Hut & Oster, 2022; Oster, 2018), at least in adulthood. This suggests a role for encouraging healthy eating and reducing demand for energy-dense processed foods in childhood, when eating behaviours and preferences are likely to be more malleable. While food preferences are likely largely shaped within the home, schools may have a role to play in promoting healthy eating among children. A school curriculum that includes healthy eating, physical activity and body image is one of the strategies commonly included in interventions found to prevent childhood obesity (Waters et al., 2011). South Africa has already recognised the importance of nutrition education in schools: one of the goals of the NSNP is to teach good eating and lifestyle habits (Department of Basic Education, n.d.), and the Integrated School Health Programme (ISHP) introduced in 2012 provides for nutrition education (Department of Health & Department of Basic Education (South Africa), 2012). However, there is probably room for improving the nutrition education provided to South African children. Nutrition education in schools is the responsibility of Life Orientation teachers, but in some South African schools Life Orientation teachers have been found to have poor nutritional knowledge (Okeyo et al., 2020). Nutrition education interventions in South African schools have had mixed results, showing an improvement in nutrition knowledge (Kupolati et al., 2019; Oldewage-Theron & Egal, 2012), but a failure to improve diet quality (Kupolati et al., 2019; Oosthuizen et al., 2011; Steyn et al., 2015). This suggests that though there is scope for improving nutritional knowledge through nutrition education in South African schools, we cannot be too optimistic that this will lead to large dietary shifts. Further research on effective nutrition education interventions is needed. A further option for promoting healthier food preferences in children may be restricting or banning advertising of junk food to children, as is being implemented in the United Kingdom from 2023. As children model their eating preferences on those of adults around them (Venter & Harris, 2009), this may also be an argument for banning junk food marketing across the board.

5.2.3 Policies to reduce economic insecurity

Economic insecurity has been proposed as a driver of obesity in developed countries. Chapter 4 found that economic insecurity is associated with higher BMI among women at the upper end of the income distribution. At the macroeconomic level, policies to support the creation of stable employment opportunities are likely to reduce economic insecurity. Social protection may also help to reduce economic insecurity and its potential negative effects on health. A policy such as the proposed Basic Income Grant (if it is of sufficient magnitude) may help to alleviate economic insecurity even for those individuals who are not at immediate risk of falling into poverty. However, while the CSG has been found to be associated with improved child nutrition, it is worth noting that the impact of cash transfers on adult body weight is unclear. It is possible that increased income from cash transfers may increase food consumption and also obesity for lower-income individuals. Case and Menendez (2009) find that receipt of the CSG is associated with increased odds of obesity, as well as with a higher reported sugar intake, among women in a Cape Town township. D'Agostino et al. (2018) find that the CSG leads to a significant increase in household food expenditure, and increases expenditure on carbohydrates without increasing dietary diversity. The Supplemental Nutrition Assistance Program (formerly the Food Stamp Program) in the US and the *Oportunidades* conditional cash transfer programme in Mexico have also been found to be associated with higher adult obesity (Fernald et al., 2008; Gibson, 2003; Webb et al., 2008). It is thus worth exploring ways to provide the benefits of cash transfers without increasing obesity, and to encourage the use of cash transfers to purchase more nutritious foods. For example, in the US there have been proposals to make sugar-sweetened beverages and junk foods ineligible for purchase with SNAP funds (see e.g. Shenkin & Jacobson, 2010), though such a proposal may not be administratively feasible or desirable in the South African context. Other options that have been proposed to encourage healthier purchases with SNAP funds include giving cash back on purchases of healthy foods with SNAP funds (Shenkin & Jacobson, 2010) and offering prizes through lottery for buying healthy foods (Richards & Sindelar, 2013). Nutrition education programmes, such as SNAP-Ed linked to the SNAP programme in the US (Shenkin & Jacobson, 2010), are another option.

5.3 Final remarks

This dissertation has investigated several nuances in the social gradient in body weight in South Africa, showing that this relationship may differ depending on one's start in life, and may also differ for younger generations. The social gradient in body weight has not yet reversed for South African men or women, but may show signs of flattening in younger generations and among the children of high-SES parents. This calls for us to consider that the nutrition and obesity transitions may happen not only over time, but over the life course and generations. The nutrition transition is a process that affects different groups differently at different times. Without urgent action, obesity rates are likely to continue to rise, particularly for those from low childhood SES backgrounds who experience social mobility over the life course. This calls for policies to attempt to reduce consumption of unhealthy foods at all ages, such as simplified front-of-pack food labelling requirements and restrictions on the marketing of unhealthy foods. It also calls for policies to improve nutrition in childhood, particularly in the earliest years of life. It remains to be seen whether any of these policies will be effective in reversing South Africa's persistently high obesity rates, but without intervention these trends are unlikely to resolve in the near future. This dissertation has provided evidence that a shift to a stage of the obesity transition where we may eventually see a decline in obesity prevalence is likely to take several generations at the least, and South Africa will have to deal with the health consequences of high and possibly rising obesity rates for decades to come.

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Appendices

APPENDIX A

CHILDHOOD SOCIOECONOMIC STATUS, SOCIAL MOBILITY AND THE OBESITY TRANSITION IN SOUTH AFRICA

A.1 Survey weighting of random effects models

Appropriate survey weighting of RE models requires weights to be specified at both the observation (level 1) and the individual level (level 2). The level 2 weight w_i is the inverse of the probability of the individual being included in the sample, and the level 1 weight $w_{t|i}$ is the inverse of the probability that the individual was observed in each wave, given that they were included in wave 1 (Pfeffermann et al., 1998; Rabe-Hesketh & Skrondal, 2006; Skinner & Holmes, 2003). The weights included with many panel surveys, including NIDS, are a final weight w_{it}, which is the product of the inverse of the inclusion probabilities at both level 1 and level 2: $w_{it} = 1/\pi_i * 1/\pi_{t|i}$, or equivalently $w_{it} = w_i * w_{t|i}$. This means that the appropriate level 1 weights for multilevel models using panel data could be calculated given the wave 1 design or calibrated weight and the panel weight as $w_{t|i} = w_{it}/w_{i1}$, where w_{i1} is the wave 1 calibrated weight. The level 1 weight for wave 1 is then simply 1 (Skinner & Holmes, 2003, p. 211).

The NIDS panel weights are the product of the wave 1 calibrated weight and an attrition weight, which is the inverse of the probability of being re-interviewed at each of the following waves, conditional on being a wave 1 sample member (Branson & Wittenberg, 2019; Brophy et al., 2018). The NIDS wave 1 calibrated weight and panel weights could thus hypothetically be used to calculate attrition weights for each of the following waves, as outlined above. However, the NIDS panel weights are also rescaled so that they sum to the population total for the survey year. This means that simply dividing the panel weight by the wave 1 calibrated weight will not yield the exact attrition weight. In order to calculate appropriate attrition weights, we re-estimated the sample retention probabilities $\pi_{t|i}$ for each wave using the same procedure used to produce the original panel weights. The probability of being re-interviewed in each wave was estimated using a probit model on the full sample using a set of wave 1 individual and household characteristics, listed in Branson and Wittenberg (2019, p. 5). The attrition weights are the inverse of these probabilities: $\pi_{t|i}^{-1}$.

The original NIDS panel weights are only available for continuing sample members (CSMs) who were included in wave 1. As Brophy et al. (2018, p. 68) note, it is possible to calculate attrition weights for top-up sample members (TSMs), but this is conceptually tricky. TSMs cease to be sample members when they cease to live with a CSM, so their process of attrition is different to that of CSMs. We thus decided to limit the sample to individuals who were successfully interviewed in wave 1. For the sake of comparability, the sample was limited to the same individuals for the OLS regressions.

A further issue in survey weighting multilevel models is the scaling of the level 1 weights. In the case of small level 1 sample sizes (in this case, a small number of observations per individual), both estimates of the variance of the random effects and the regression coefficients may be biased. Several methods are available to scale weights in an attempt to prevent this (Pfeffermann et al., 1998; Rabe-Hesketh & Skrondal, 2006). In this paper, level 1 weights are scaled so that they sum to the level 2 sample size, i.e. to the number of observations for each individual (using the *pwscale(size)* option in Stata's *mixed* command).

A.2 Cleaning of anthropometric data

NIDS includes up to three weight and height measurements. Fieldworkers were required to take two height and weight measurements, and a third if the first two measurements were more than one centimetre apart in the case of height and one kilogram apart in the case of weight (Brophy et al., 2018). Following the procedure for calculating z-scores for children in NIDS (Brophy et al., 2018, p. 64), we use the average of the first two measurements for weight and height respectively if the difference between the measurements is below the aforementioned thresholds. If the two measurements differed by more than the 1 cm for height and 1 kg for weight, we took the third measure if it was available. In cases where one of the first two measurements were missing but a third was available, the average of the two non-missing measurements was taken.

A.2.1 Cleaning of height data

We used the five waves of NIDS available to identify suspicious measurements in the anthropometric data. This cleaning procedure was only run for adults 25 years or older, as it relies on individuals having reached their maximum height and having a stable height across waves[1]. Heights were measured using a portable stadiometer (Finn & Ranchhod, 2017). For adults who have reached their maximum height, height would not be expected to change significantly between waves. Older adults tend to lose height as they age, but this should not result in large changes. Most height measurements differed by at least a small amount between waves, but except in the case of very old or very young adults (who are excluded from the sample in any case), this is unlikely to reflect true variation in height. Given that height is squared in the BMI formula, small measurement errors in height could result in relatively large changes in BMI. Adults' height was therefore set to be equal across waves, so that any change in BMI reflects a change in weight rather than measurement error in height. For individuals with height data in more than two waves, suspicious height values were identified by identifying large deviations from the mean for that individual. For each observation, we calculated the absolute value of the difference between that observation and the mean of other observations for that individual (excluding that observation). The observation with the maximum deviation from the mean of other observations was identified, and if that difference was greater than 2 cm, then that observation was set to the mean of other observations for that individual. If

[1] An argument could also be made for excluding older adults from this cleaning procedure, as their height might decline between waves, but my analysis focuses on working age adults, so older adults are excluded from my analysis.

an individual had only two non-missing height observations (and therefore it was impossible to see which value was more likely to be correct), both values were set to missing if these values differed by more than 20 cm. Once suspicious values were recoded or removed in this way, each height observation was set to the mean of height observations for that individual.

A.2.2 Cleaning of weight data

Weights were measured using digital scales accurate to 0.1kg (Finn & Ranchhod, 2017). A possible source of measurement error in weight identified by Finn and Ranchhod is that the scales could accidentally have been set to pounds rather than kilograms. Given that 1 kilogram is equal to 2.20462 pounds, this would result in measurements 2.2 times higher than they should be. If a weight observation was more than two times (not 2.2, to allow for possible downward fluctuation in weight between waves) greater than the values in both the preceding and subsequent waves, that observation was divided by 2.20462.[2] In order to deal with this issue for the first and last observation on an individual (where either the lagged or leading value are not available), a weight observation was also divided by 2.20462 where the weight measurement was more than double the mean of the other observations on that individual (excluding the observation in question). Furthermore, where more than one household member in a single wave had a weight measurement at least two times the preceding or subsequent value, we took this as further evidence that the scale was set incorrectly, and used slightly less strict criteria for cleaning these observations. In this case, weight was assumed to be in pounds and divided by 2.20462 if weight was greater than 1.5 times the mean of the other observations for that individual, or was more than two times either the lagged or the leading weight value.

Once the weight data were cleaned in this way, 67 observations still have a weight measurement more than double either the previous or the subsequent measurement. Unfortunately, it is less clear whether these cases are due to measurement error or to true fluctuations in weight. In some cases a weight observation is less than half most of the other observations for that individual. It is possible that the interviewer erroneously set the scale to pounds in all waves except one for a particular individual, but this seems less likely than this error occurring in only one wave. We were

[2]This strategy assumes that if weight at least doubles between waves before shrinking back to near its original value this is more likely due to the scale accidentally being set to pounds than to a true fluctuation in weight. Large fluctuations in weight are possible due to factors such as yo-yo dieting or regaining weight after weight loss, but doubling in weight within 2 years and then losing it again seems highly unlikely, so this seems to be a reasonable assumption.

thus hesitant to assume that the one relatively low value was due to incorrect setting of the scale in all other waves and that the low value is therefore the true value and that the other measurements need to be converted into kilograms.

BMI was not calculated for pregnant women or for those with an adult height at or below the cutoff for dwarfism (147 cm), as BMI is not a valid measure for pregnant women or for people with dwarfism (Schulze et al., 2013).

A.3 Descriptive statistics

Table A.3.1: Weighted versus unweighted sample proportions

	Unweighted		**Weighted (estimation)**		**Weighted (full)**	
	Prop.	**S.E.**	**Prop.**	**S.E.**	**Prop.**	**S.E.**
Female	0.60	0.00	0.53	0.01	0.51	0.01
Male	0.40	0.00	0.47	0.01	0.49	0.01
Mother less than matric	0.96	0.00	0.93	0.01	0.92	0.01
Mother matric	0.04	0.01	0.07	0.01	0.08	0.01
Age 25-34	0.34	0.00	0.36	0.01	0.38	0.01
Age 35-44	0.26	0.00	0.28	0.01	0.28	0.01
Age 45-54	0.23	0.00	0.21	0.01	0.20	0.00
Age 55-64	0.17	0.00	0.15	0.01	0.14	0.00
African	0.81	0.00	0.81	0.02	0.77	0.02
Coloured	0.14	0.00	0.09	0.02	0.10	0.02
Asian/Indian	0.01	0.01	0.03	0.01	0.03	0.01
White	0.03	0.01	0.08	0.01	0.10	0.01
Income quintile 1	0.20	0.00	0.17	0.01	0.16	0.01
Income quintile 2	0.22	0.00	0.17	0.01	0.17	0.01
Income quintile 3	0.23	0.00	0.20	0.01	0.20	0.01
Income quintile 4	0.20	0.00	0.23	0.01	0.23	0.01
Income quintile 5	0.14	0.00	0.22	0.02	0.25	0.02
Less than matric	0.69	0.00	0.61	0.01	0.58	0.01
Matric	0.14	0.00	0.16	0.01	0.18	0.01
Post matric	0.17	0.00	0.23	0.01	0.24	0.01
Not employed	0.48	0.00	0.41	0.01	0.42	0.01
Employed	0.52	0.00	0.59	0.01	0.58	0.01
Rural	0.48	0.00	0.35	0.03	0.32	0.03
Urban	0.52	0.00	0.65	0.03	0.68	0.03
Not married/cohabiting	0.55	0.00	0.53	0.01	0.52	0.01
Married/cohabiting	0.45	0.00	0.47	0.01	0.48	0.01

A.4 Alternative outcomes

Table A.4.1: Probit regressions for obesity, women

	(1)	(2)	(3)	(4)	(5)
Mother matric	−0.25**	−0.23*	0.12	0.03	0.20
	(0.12)	(0.12)	(0.14)	(0.14)	(0.19)
Household income	0.09***	0.10***	0.11***	0.11***	0.09***
	(0.03)	(0.03)	(0.03)	(0.03)	(0.03)
Income sq.		−0.03*		−0.01	
		(0.01)		(0.01)	
Urban	0.09*	0.09*	0.08*	0.08*	0.10**
	(0.05)	(0.05)	(0.05)	(0.05)	(0.05)
Mother matric x Household income			−0.26***	0.12	
			(0.09)	(0.15)	
Mother matric x income sq.				−0.14**	
				(0.06)	
Mother matric x Urban					−0.54**
					(0.22)
Matric	−0.00	0.00	−0.02	−0.01	−0.01
	(0.07)	(0.07)	(0.07)	(0.07)	(0.07)
Post matric	0.13**	0.15**	0.13**	0.14**	0.13**
	(0.06)	(0.06)	(0.06)	(0.06)	(0.06)
Observations	21889	21889	21889	21889	21889
Pseudo R^2	0.05	0.05	0.05	0.05	0.05

* p <0.1, ** p <0.05, *** p <0.01. Standard errors in parentheses.
Note: Full set of covariates included but not displayed.

Table A.4.2: Probit regressions for obesity, men

	(1)	(2)	(3)	(4)	(5)
Mother matric	−0.46***	−0.48***	0.01	−0.09	−0.13
	(0.16)	(0.16)	(0.17)	(0.18)	(0.23)
Household income	0.27***	0.23***	0.29***	0.24***	0.27***
	(0.04)	(0.03)	(0.04)	(0.03)	(0.04)
Income sq.		0.03**		0.04***	
		(0.01)		(0.01)	
Urban	0.13*	0.12*	0.12*	0.12*	0.14**
	(0.07)	(0.07)	(0.07)	(0.07)	(0.07)
Mother matric x Household income			−0.27**	0.04	
			(0.12)	(0.20)	
Mother matric x income sq.				−0.12*	
				(0.07)	
Mother matric x Urban					−0.38
					(0.27)
Matric	0.27***	0.28***	0.26***	0.25***	0.27***
	(0.09)	(0.09)	(0.09)	(0.09)	(0.09)
Post matric	0.17**	0.15*	0.17**	0.14*	0.17**
	(0.08)	(0.08)	(0.08)	(0.08)	(0.08)
Observations	14771	14771	14771	14771	14771
Pseudo R^2	0.12	0.12	0.13	0.13	0.12

* p <0.1, ** p <0.05, *** p <0.01. Standard errors in parentheses.
Note: Full set of covariates included but not displayed.

Table A.4.3: Probit regressions for obesity stratified by urban residence, women

	Urban				Rural			
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Mother matric	−0.31**	−0.29**	0.04	−0.17	0.10	0.10	0.22	0.36*
	(0.14)	(0.14)	(0.18)	(0.18)	(0.19)	(0.19)	(0.19)	(0.20)
Household income	0.08**	0.10***	0.10***	0.10***	0.14***	0.14***	0.14***	0.15***
	(0.03)	(0.04)	(0.03)	(0.03)	(0.03)	(0.04)	(0.03)	(0.04)
Income sq.		−0.03		−0.01		0.01		0.02
		(0.02)		(0.02)		(0.02)		(0.02)
Mother matric x			−0.23**	0.32			−0.23	−0.11
Household income			(0.10)	(0.19)			(0.16)	(0.19)
Mother matric x				−0.18***				−0.17
income sq.				(0.07)				(0.11)
Matric	−0.11	−0.10	−0.12	−0.12	0.22***	0.22***	0.22***	0.22***
	(0.09)	(0.09)	(0.09)	(0.09)	(0.08)	(0.08)	(0.08)	(0.08)
Post matric	0.06	0.08	0.06	0.07	0.35***	0.34***	0.35***	0.34***
	(0.08)	(0.08)	(0.08)	(0.08)	(0.08)	(0.08)	(0.08)	(0.08)
Observations	10942	10942	10942	10942	10947	10947	10947	10947
Pseudo R^2	0.05	0.05	0.05	0.05	0.06	0.06	0.06	0.06

* p <0.1, ** p <0.05, *** p <0.01. Standard errors in parentheses.

Note: Full set of covariates included but not displayed.

Table A.4.4: Probit regressions for obesity stratified by urban residence, men

	Urban				Rural			
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Mother matric	−0.48***	−0.50***	0.03	−0.18	−0.19	−0.20	0.02	−0.00
	(0.18)	(0.18)	(0.20)	(0.26)	(0.25)	(0.25)	(0.27)	(0.30)
Household income	0.29***	0.23***	0.32***	0.24***	0.20***	0.19***	0.20***	0.20***
	(0.05)	(0.05)	(0.06)	(0.05)	(0.05)	(0.05)	(0.05)	(0.05)
Income sq.		0.03*		0.06***		0.01		0.01
		(0.02)		(0.02)		(0.02)		(0.02)
Mother matric x Household income			−0.28**	0.20			−0.19	−0.22
			(0.13)	(0.29)			(0.20)	(0.18)
Mother matric x income sq.				−0.17*				0.02
				(0.09)				(0.11)
Matric	0.18*	0.19*	0.16	0.16	0.52***	0.52***	0.51***	0.51***
	(0.11)	(0.11)	(0.11)	(0.11)	(0.13)	(0.13)	(0.13)	(0.13)
Post matric	0.10	0.08	0.09	0.06	0.33***	0.33***	0.33***	0.33***
	(0.10)	(0.10)	(0.10)	(0.10)	(0.11)	(0.11)	(0.11)	(0.11)
Observations	8212	8212	8212	8212	6559	6559	6559	6559
Pseudo R^2	0.13	0.13	0.13	0.14	0.09	0.09	0.09	0.09

* p <0.1, ** p <0.05, *** p <0.01. Standard errors in parentheses.
Note: Full set of covariates included but not displayed.

Table A.4.5: Waist circumference OLS regressions stratified by urban residence, women

	Urban				Rural			
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Mother matric	−3.70**	−3.55**	−0.52	−1.88	2.72	2.69	3.77	5.70
	(1.65)	(1.63)	(2.01)	(1.93)	(2.88)	(2.90)	(3.17)	(3.67)
Household income	0.90**	0.98**	1.06**	1.10**	1.69***	1.72***	1.76***	1.82***
	(0.41)	(0.43)	(0.43)	(0.43)	(0.35)	(0.39)	(0.35)	(0.39)
Income sq.		−0.16		−0.06		0.07		0.14
		(0.17)		(0.17)		(0.17)		(0.17)
Mother matric x			−2.06**	1.59			−2.15	−0.57
Household income			(1.04)	(1.70)			(1.81)	(1.68)
Mother matric x				−1.20**				−2.21**
income sq.				(0.49)				(1.10)
Matric	−2.19**	−2.14**	−2.28**	−2.28**	0.94	0.93	0.92	0.87
	(1.09)	(1.09)	(1.09)	(1.09)	(0.95)	(0.95)	(0.95)	(0.95)
Post matric	−1.03	−0.89	−1.04	−0.99	3.73***	3.67***	3.72***	3.66***
	(0.97)	(0.97)	(0.96)	(0.97)	(0.92)	(0.93)	(0.91)	(0.92)
Observations	10721	10721	10721	10721	10710	10710	10710	10710
Adjusted R^2	0.09	0.09	0.09	0.10	0.11	0.11	0.11	0.11

* p <0.1, ** p <0.05, *** p <0.01. Standard errors in parentheses.

Note: Full set of covariates included but not displayed.

Table A.4.6: Waist circumference OLS regressions stratified by urban residence, men

	Urban				Rural			
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Mother matric	−1.84	−2.00	1.73	1.85	−1.15	−1.40	−1.14	−1.87
	(1.23)	(1.23)	(1.31)	(1.13)	(1.42)	(1.41)	(1.40)	(1.72)
Household income	3.42***	3.11***	3.66***	3.26***	1.78***	1.94***	1.79***	1.95***
	(0.38)	(0.32)	(0.40)	(0.36)	(0.34)	(0.33)	(0.35)	(0.34)
Income sq.		0.33***		0.52***		0.24***		0.24***
		(0.12)		(0.14)		(0.07)		(0.07)
Mother matric x Household income			−2.50***	−0.45			−0.02	−1.26
			(0.81)	(1.34)			(1.09)	(1.12)
Mother matric x income sq.				−1.01**				0.84
				(0.47)				(0.82)
Matric	2.81***	2.76***	2.71***	2.53***	3.84***	3.71***	3.84***	3.68***
	(0.86)	(0.86)	(0.87)	(0.87)	(1.14)	(1.14)	(1.14)	(1.15)
Post matric	1.84**	1.57*	1.79**	1.35	4.09***	3.80***	4.09***	3.80***
	(0.84)	(0.86)	(0.84)	(0.86)	(0.92)	(0.92)	(0.93)	(0.92)
Observations	8012	8012	8012	8012	6395	6395	6395	6395
Adjusted R^2	0.25	0.25	0.26	0.26	0.16	0.16	0.16	0.16

* p <0.1, ** p <0.05, *** p <0.01. Standard errors in parentheses.

Note: Full set of covariates included but not displayed.

Table A.4.7: Waist-height ratio OLS regressions stratified by urban residence, women

	Urban				Rural			
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Mother matric	−0.03**	−0.02**	−0.00	−0.01	0.02	0.02	0.02	0.03
	(0.01)	(0.01)	(0.01)	(0.01)	(0.02)	(0.02)	(0.02)	(0.02)
Household income	0.01**	0.01**	0.01**	0.01***	0.01***	0.01***	0.01***	0.01***
	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
Income sq.		−0.00		−0.00		0.00		0.00
		(0.00)		(0.00)		(0.00)		(0.00)
Mother matric x			−0.02**	0.01			−0.01	−0.01
Household income			(0.01)	(0.01)			(0.01)	(0.01)
Mother matric x				−0.01***				−0.01*
income sq.				(0.00)				(0.01)
Matric	−0.01**	−0.01**	−0.02**	−0.02**	0.01	0.01	0.01	0.01
	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)
Post matric	−0.01	−0.01	−0.01	−0.01	0.02***	0.02***	0.02***	0.02***
	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)
Observations	10845	10845	10845	10845	10793	10793	10793	10793
Adjusted R^2	0.10	0.10	0.10	0.11	0.13	0.13	0.13	0.13

* p <0.1, ** p <0.05, *** p <0.01. Standard errors in parentheses.

Note: Full set of covariates included but not displayed.

Table A.4.8: Waist-height ratio OLS regressions stratified by urban residence, men

	Urban				Rural			
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Mother matric	−0.01**	−0.01**	0.01	0.01	−0.00	−0.01	−0.01	−0.01
	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)
Household income	0.02***	0.02***	0.02***	0.02***	0.01***	0.01***	0.01***	0.01***
	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
Income sq.		0.00*		0.00***		0.00***		0.00***
		(0.00)		(0.00)		(0.00)		(0.00)
Mother matric x			−0.01***	0.00			0.00	−0.00
Household income			(0.00)	(0.01)			(0.01)	(0.01)
Mother matric x				−0.01**				0.01
income sq.				(0.00)				(0.01)
Matric	0.02***	0.02***	0.02***	0.02***	0.02***	0.02***	0.02***	0.02***
	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)
Post matric	0.01*	0.01	0.01*	0.01	0.02***	0.02***	0.02***	0.02***
	(0.00)	(0.00)	(0.00)	(0.00)	(0.01)	(0.01)	(0.01)	(0.01)
Observations	8088	8088	8088	8088	6453	6453	6453	6453
Adjusted R^2	0.23	0.23	0.23	0.23	0.15	0.15	0.15	0.15

* p <0.1, ** p <0.05, *** p <0.01. Standard errors in parentheses.
Note: Full set of covariates included but not displayed.

A.5 Sensitivity of obesity results to using ethnic-specific BMI cut-points

Table A.5.1: Probit regressions for obesity using Kruger (2017) ethnic-specific cut-point for black respondents

	Women				Men			
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Mother matric	−0.21*	−0.19	0.11	0.03	−0.27**	−0.30**	0.12	0.16
	(0.12)	(0.12)	(0.14)	(0.14)	(0.12)	(0.12)	(0.15)	(0.15)
Household income	0.09***	0.09***	0.10***	0.10***	0.25***	0.24***	0.27***	0.26***
	(0.03)	(0.02)	(0.03)	(0.03)	(0.03)	(0.03)	(0.03)	(0.03)
Income sq.		−0.02		−0.01		0.04***		0.05***
		(0.01)		(0.01)		(0.01)		(0.01)
Urban	0.06	0.06	0.05	0.05	0.02	0.01	0.01	−0.00
	(0.05)	(0.05)	(0.05)	(0.05)	(0.06)	(0.06)	(0.06)	(0.06)
Mother matric x			−0.23**	0.16			−0.29***	−0.08
Household income			(0.09)	(0.14)			(0.10)	(0.12)
Mother matric x				−0.15***				−0.12**
income sq.				(0.05)				(0.05)
Matric	−0.01	−0.00	−0.02	−0.02	0.36***	0.35***	0.35***	0.33***
	(0.07)	(0.07)	(0.07)	(0.07)	(0.07)	(0.07)	(0.07)	(0.07)
Post matric	0.13**	0.15**	0.13**	0.14**	0.24***	0.20***	0.24***	0.19***
	(0.06)	(0.06)	(0.06)	(0.07)	(0.07)	(0.07)	(0.07)	(0.07)
Observations	21889	21889	21889	21889	14771	14771	14771	14771
Pseudo R^2	0.05	0.05	0.05	0.05	0.14	0.15	0.15	0.15

* p <0.1, ** p <0.05, *** p <0.01. Standard errors in parentheses.

Note: Full set of covariates included but not displayed.

Table A.5.2: Probit regressions for obesity using Peer (2016) ethnic-specific cut-point for black respondents

	Women				Men			
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Mother matric	−0.29**	−0.25**	0.03	−0.11	−0.27**	−0.29**	0.03	−0.03
	(0.13)	(0.13)	(0.15)	(0.16)	(0.13)	(0.13)	(0.15)	(0.15)
Household income	0.09***	0.12***	0.11***	0.12***	0.27***	0.25***	0.29***	0.26***
	(0.03)	(0.02)	(0.03)	(0.02)	(0.03)	(0.03)	(0.03)	(0.03)
Income sq.		−0.04***		−0.02		0.03***		0.05***
		(0.01)		(0.01)		(0.01)		(0.01)
Urban	0.13**	0.13**	0.13**	0.12**	0.08	0.07	0.07	0.07
	(0.05)	(0.05)	(0.05)	(0.05)	(0.06)	(0.05)	(0.05)	(0.05)
Mother matric x			−0.22**	0.24			−0.21**	0.19
Household income			(0.09)	(0.18)			(0.10)	(0.14)
Mother matric x				−0.16**				−0.17***
income sq.				(0.06)				(0.05)
Matric	−0.01	−0.00	−0.02	−0.01	0.34***	0.34***	0.33***	0.31***
	(0.07)	(0.07)	(0.07)	(0.07)	(0.07)	(0.07)	(0.07)	(0.07)
Post matric	0.14**	0.17**	0.14**	0.16**	0.27***	0.24***	0.27***	0.23***
	(0.07)	(0.07)	(0.07)	(0.07)	(0.07)	(0.07)	(0.07)	(0.07)
Observations	21889	21889	21889	21889	14771	14771	14771	14771
Pseudo R^2	0.05	0.06	0.06	0.06	0.12	0.12	0.12	0.12

* p <0.1, ** p <0.05, *** p <0.01. Standard errors in parentheses.

Note: Full set of covariates included but not displayed.

Table A.5.3: Probit regressions for obesity using Caleyachetty (2021) ethnic-specific cut-point for black respondents

	Women				Men			
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Mother matric	−0.21*	−0.19	0.11	0.04	−0.36**	−0.38***	0.04	−0.07
	(0.12)	(0.12)	(0.14)	(0.14)	(0.14)	(0.14)	(0.15)	(0.17)
Household income	0.09***	0.09***	0.10***	0.10***	0.27***	0.23***	0.29***	0.24***
	(0.03)	(0.02)	(0.03)	(0.03)	(0.04)	(0.03)	(0.04)	(0.03)
Income sq.		−0.02		−0.01		0.03**		0.05***
		(0.01)		(0.01)		(0.01)		(0.01)
Urban	0.07	0.07	0.06	0.06	0.14**	0.13**	0.13**	0.12**
	(0.05)	(0.05)	(0.05)	(0.05)	(0.06)	(0.06)	(0.06)	(0.06)
Mother matric x Household income			−0.23***	0.16			−0.25**	0.15
			(0.09)	(0.14)			(0.11)	(0.19)
Mother matric x income sq.				−0.14***				−0.16**
				(0.05)				(0.07)
Matric	−0.01	−0.01	−0.02	−0.02	0.29***	0.29***	0.27***	0.27***
	(0.07)	(0.06)	(0.07)	(0.06)	(0.08)	(0.08)	(0.08)	(0.08)
Post matric	0.13**	0.15**	0.13**	0.14**	0.24***	0.22***	0.24***	0.21***
	(0.06)	(0.06)	(0.06)	(0.07)	(0.07)	(0.08)	(0.07)	(0.07)
Observations	21889	21889	21889	21889	14771	14771	14771	14771
Pseudo R^2	0.05	0.05	0.05	0.05	0.11	0.11	0.11	0.11

* p <0.1, ** p <0.05, *** p <0.01. Standard errors in parentheses.

Note: Full set of covariates included but not displayed.

A.6 Alternative measures of childhood SES

A.6.1 Father education

Table A.6.1: OLS regressions of BMI on father education stratified by urban residence, women

	Urban				Rural			
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Father matric	−1.81***	−1.75**	−0.96	−1.35*	−0.88	−0.88	−0.47	−0.22
	(0.70)	(0.69)	(0.74)	(0.71)	(0.98)	(0.98)	(0.91)	(1.09)
Household income	0.58***	0.62***	0.64***	0.66***	0.94***	0.94***	0.95***	0.95***
	(0.22)	(0.21)	(0.22)	(0.21)	(0.16)	(0.18)	(0.17)	(0.18)
Income sq.		−0.08		−0.05		0.00		0.01
		(0.06)		(0.06)		(0.08)		(0.08)
Father matric x Household income			−0.59	0.67			−0.62	−0.46
			(0.47)	(0.76)			(0.63)	(0.69)
Father matric x income sq.				−0.42*				−0.22
				(0.24)				(0.39)
Matric	−0.67	−0.65	−0.73	−0.71	0.97**	0.97**	0.97**	0.97**
	(0.53)	(0.53)	(0.53)	(0.53)	(0.48)	(0.49)	(0.49)	(0.49)
Post matric	0.11	0.18	0.09	0.13	2.26***	2.26***	2.27***	2.27***
	(0.51)	(0.51)	(0.51)	(0.51)	(0.53)	(0.53)	(0.53)	(0.53)
Observations	9786	9786	9786	9786	10472	10472	10472	10472
Adjusted R^2	0.08	0.08	0.08	0.08	0.10	0.10	0.10	0.10

* p <0.1, ** p <0.05, *** p <0.01. Standard errors in parentheses.

Note: Full set of covariates included but not displayed.

Table A.6.2: OLS regressions of BMI on father education stratified by urban residence, men

	Urban				Rural			
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Father matric	−1.01*	−1.08*	0.43	0.48	−0.89	−1.00*	−1.09**	−1.01*
	(0.56)	(0.57)	(0.79)	(0.72)	(0.55)	(0.55)	(0.51)	(0.56)
Household income	1.14***	1.06***	1.23***	1.13***	0.74***	0.80***	0.73***	0.80***
	(0.16)	(0.14)	(0.16)	(0.15)	(0.12)	(0.13)	(0.12)	(0.13)
Income sq.		0.09*		0.17***		0.11***		0.11***
		(0.05)		(0.05)		(0.03)		(0.03)
Father matric x			−0.96**	−0.45			0.23	0.10
Household income			(0.40)	(0.70)			(0.43)	(0.62)
Father matric x				−0.28				−0.04
income sq.				(0.23)				(0.30)
Matric	1.06***	1.04***	0.99***	0.91**	1.56***	1.50***	1.56***	1.50***
	(0.36)	(0.36)	(0.36)	(0.36)	(0.46)	(0.46)	(0.46)	(0.46)
Post matric	0.93***	0.85**	0.95***	0.81**	1.45***	1.32***	1.44***	1.32***
	(0.36)	(0.37)	(0.36)	(0.37)	(0.35)	(0.36)	(0.36)	(0.36)
Observations	7421	7421	7421	7421	6164	6164	6164	6164
Adjusted R^2	0.19	0.19	0.19	0.20	0.11	0.11	0.11	0.11

* p <0.1, ** p <0.05, *** p <0.01. Standard errors in parentheses.

Note: Full set of covariates included but not displayed.

Table A.6.3: OLS regressions of BMI on father education for combined urban and rural samples, women

	(1)	(2)	(3)	(4)	(5)
Father matric	−2.04***	−1.95***	−1.01	−1.27**	−0.57
	(0.65)	(0.64)	(0.66)	(0.63)	(0.96)
Household income	0.71***	0.73***	0.76***	0.77***	0.71***
	(0.15)	(0.15)	(0.16)	(0.15)	(0.15)
Income sq.		−0.09*		−0.05	
		(0.05)		(0.06)	
Urban	0.80***	0.82***	0.78***	0.79***	0.84***
	(0.30)	(0.30)	(0.30)	(0.30)	(0.30)
Father matric x Household income			−0.74*	0.35	
			(0.43)	(0.63)	
Father matric x income sq.				−0.37*	
				(0.22)	
Father matric x Urban					−1.61
					(1.10)
Matric	−0.11	−0.09	−0.16	−0.14	−0.12
	(0.39)	(0.39)	(0.39)	(0.39)	(0.39)
Post matric	0.71*	0.79**	0.70*	0.76*	0.71*
	(0.40)	(0.40)	(0.40)	(0.40)	(0.40)
Observations	20258	20258	20258	20258	20258
Adjusted R^2	0.08	0.08	0.08	0.08	0.08

* p <0.1, ** p <0.05, *** p <0.01. Standard errors in parentheses.

Note: Full set of covariates included but not displayed.

Table A.6.4: OLS regressions of BMI on father education for combined urban and rural samples, men

	(1)	(2)	(3)	(4)	(5)
Father matric	−0.99**	−1.09**	0.04	0.11	−0.54
	(0.49)	(0.49)	(0.63)	(0.56)	(0.54)
Household income	1.04***	0.99***	1.09***	1.05***	1.04***
	(0.12)	(0.11)	(0.12)	(0.11)	(0.12)
Income sq.		0.11***		0.17***	
		(0.04)		(0.03)	
Urban	0.34	0.31	0.34*	0.30	0.36*
	(0.21)	(0.21)	(0.21)	(0.21)	(0.21)
Father matric x Household income			−0.73**	−0.21	
			(0.35)	(0.58)	
Father matric x income sq.				−0.30	
				(0.21)	
Father matric x Urban					−0.53
					(0.73)
Matric	1.22***	1.18***	1.17***	1.09***	1.22***
	(0.30)	(0.30)	(0.30)	(0.30)	(0.30)
Post matric	1.15***	1.04***	1.18***	1.02***	1.15***
	(0.28)	(0.29)	(0.28)	(0.29)	(0.28)
Observations	13585	13585	13585	13585	13585
Adjusted R^2	0.17	0.18	0.18	0.18	0.17

* p <0.1, ** p <0.05, *** p <0.01. Standard errors in parentheses.
Note: Full set of covariates included but not displayed.

A.6.2 Subjective income step at 15

Table A.6.5: Subjective income step at 15 as alternative measure of childhood SES

	Women				Men			
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Step at 15	0.25***	0.26***	0.37***	0.51***	0.09	0.08	0.13*	0.19***
	(0.09)	(0.09)	(0.08)	(0.09)	(0.06)	(0.06)	(0.07)	(0.07)
Household income	0.63***	0.65***	1.43***	1.16***	1.09***	1.03***	1.25***	1.22***
	(0.15)	(0.14)	(0.23)	(0.23)	(0.11)	(0.10)	(0.16)	(0.15)
Income sq.		−0.10*		0.25***		0.13***		0.22***
		(0.05)		(0.07)		(0.04)		(0.05)
Step at 15 x income			−0.38***	−0.18*			−0.07	−0.09*
			(0.10)	(0.09)			(0.06)	(0.05)
Step at 15 x income sq.				−0.15***				−0.03**
				(0.04)				(0.01)
Observations	21526	21526	21526	21526	14530	14530	14530	14530
Adjusted R^2	0.08	0.08	0.08	0.08	0.19	0.19	0.19	0.19

* p <0.1, ** p <0.05, *** p <0.01. Standard errors in parentheses.
Note: Full set of covariates included but not displayed.

A.6.3 Adult height

Table A.6.6: Adult height as a proxy for childhood SES

	Women				Men			
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Height (10cm)	−0.75***	−0.75***	−0.64***	−0.68***	−0.25	−0.27*	−0.26*	−0.17
	(0.22)	(0.22)	(0.22)	(0.25)	(0.16)	(0.16)	(0.15)	(0.17)
Household income	0.68***	0.70***	7.27**	7.76***	1.13***	1.06***	0.77	2.34
	(0.15)	(0.14)	(3.06)	(2.73)	(0.11)	(0.10)	(2.66)	(2.05)
Income sq.		−0.10*		−0.64		0.14***		0.84
		(0.05)		(1.48)		(0.04)		(1.34)
Height (10cm) x income			−0.41**	−0.44**			0.02	−0.07
			(0.19)	(0.17)			(0.16)	(0.12)
Height (10cm) x income sq.				0.03				−0.04
				(0.09)				(0.08)
Observations	21889	21889	21889	21889	14771	14771	14771	14771
Adjusted R^2	0.08	0.08	0.08	0.08	0.18	0.19	0.18	0.19

* p <0.1, ** p <0.05, *** p <0.01. Standard errors in parentheses.
Note: Full set of covariates included but not displayed.

A.7 Alternative measures of adult SES

A.7.1 Own education

Table A.7.1: Interaction regressions with own education as alternative measure of adult SES

	Women		Men	
	(1)	(2)	(3)	(4)
Mother matric	0.56	0.53	−0.63	−0.65
	(1.23)	(1.22)	(0.52)	(0.51)
Matric	−0.04	−0.02	1.30***	1.26***
	(0.38)	(0.38)	(0.30)	(0.30)
Post matric	0.94**	1.01***	1.08***	0.94***
	(0.38)	(0.39)	(0.26)	(0.27)
Mother matric x Matric	−1.47	−1.38	−1.18	−1.25
	(1.50)	(1.51)	(0.88)	(0.88)
Mother matric x Post matric	−2.86**	−2.72**	−0.32	−0.44
	(1.30)	(1.30)	(0.69)	(0.69)
Household income	0.70***	0.72***	1.14***	1.08***
	(0.14)	(0.14)	(0.11)	(0.10)
Income sq.		−0.08		0.15***
		(0.05)		(0.03)
Observations	21889	21889	14771	14771
Adjusted R^2	0.08	0.08	0.19	0.19

* p <0.1, ** p <0.05, *** p <0.01. Standard errors in parentheses.
Note: Full set of covariates included but not displayed.

A.7.2 Asset index

Table A.7.2: Asset index as alternative measure of adult SES

	Women		Men	
	(1)	(2)	(3)	(4)
Mother matric	−1.47**	−0.17	−1.03**	0.00
	(0.72)	(0.78)	(0.42)	(0.36)
Asset index	1.16***	1.31***	1.00***	1.15***
	(0.13)	(0.13)	(0.10)	(0.11)
Mother matric x		−1.42***		−1.35***
asset index		(0.43)		(0.25)
Observations	21878	21878	14763	14763
Adjusted R^2	0.09	0.09	0.18	0.18

* p <0.1, ** p <0.05, *** p <0.01. Standard errors in parentheses.
Note: Full set of covariates included but not displayed.

A.7.3 Subjective income step today

Table A.7.3: Subjective income step as alternative measure of adult SES

	Women		Men	
	(1)	(2)	(3)	(4)
Mother matric	−1.52**	1.20	−0.84**	2.23***
	(0.70)	(1.55)	(0.41)	(0.70)
Step today	0.52***	0.58***	0.60***	0.69***
	(0.10)	(0.10)	(0.07)	(0.08)
Mother matric x		−0.83*		−0.98***
income step		(0.45)		(0.23)
Observations	21593	21593	14571	14571
Adjusted R^2	0.08	0.08	0.17	0.17

* p <0.1, ** p <0.05, *** p <0.01. Standard errors in parentheses.
Note: Full set of covariates included but not displayed.

Table A.7.4: Subjective income step as alternative measure of adult SES and subjective income step at 15 as alternative measure of childhood SES

	Women		Men	
	(1)	(2)	(3)	(4)
Step at 15	0.07	0.86***	−0.10	0.03
	(0.10)	(0.21)	(0.07)	(0.16)
Step today	0.46***	1.10***	0.65***	0.75***
	(0.12)	(0.20)	(0.09)	(0.14)
Step at 15 x step today		−0.28***		−0.05
		(0.07)		(0.05)
Observations	21451	21451	14481	14481
Adjusted R^2	0.07	0.08	0.17	0.17

* p <0.1, ** p <0.05, *** p <0.01. Standard errors in parentheses.
Note: Full set of covariates included but not displayed.

A.8 African and coloured subsample

Table A.8.1: BMI OLS regressions stratified by urban residence, African and coloured women

	Urban				Rural			
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Mother matric	−1.71*	−1.80**	−1.36	−1.23	1.45	1.44	1.73	2.82
	(0.88)	(0.88)	(1.03)	(1.06)	(1.81)	(1.81)	(1.74)	(2.42)
Household income	0.99***	1.00***	1.01***	1.02***	0.93***	0.96***	0.95***	1.00***
	(0.18)	(0.18)	(0.18)	(0.18)	(0.16)	(0.18)	(0.16)	(0.18)
Income sq.		0.13***		0.13***		0.07		0.08
		(0.05)		(0.05)		(0.07)		(0.07)
Mother matric x			−0.38	−0.48			−1.15	−1.00
Household income			(0.62)	(0.79)			(1.02)	(0.87)
Mother matric x				−0.06				−1.22
income sq.				(0.33)				(1.08)
Matric	0.34	0.31	0.33	0.29	0.79	0.78	0.79	0.79
	(0.47)	(0.47)	(0.47)	(0.47)	(0.49)	(0.49)	(0.49)	(0.49)
Post matric	0.45	0.33	0.45	0.32	2.47***	2.41***	2.45***	2.39***
	(0.43)	(0.43)	(0.43)	(0.43)	(0.48)	(0.48)	(0.47)	(0.47)
Observations	10179	10179	10179	10179	10763	10763	10763	10763
Adjusted R^2	0.09	0.09	0.09	0.09	0.11	0.11	0.11	0.11

* p <0.1, ** p <0.05, *** p <0.01. Standard errors in parentheses.
Note: Full set of covariates included but not displayed.

Table A.8.2: BMI OLS regressions stratified by urban residence, African and coloured men

	Urban				Rural			
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Mother matric	0.42	0.38	0.16	0.16	−0.03	−0.16	0.07	−0.27
	(0.42)	(0.41)	(0.41)	(0.46)	(0.73)	(0.73)	(0.81)	(0.94)
Household income	1.34***	1.21***	1.33***	1.21***	0.74***	0.84***	0.75***	0.84***
	(0.14)	(0.13)	(0.15)	(0.14)	(0.12)	(0.12)	(0.12)	(0.12)
Income sq.		0.25***		0.24***		0.13***		0.13***
		(0.06)		(0.06)		(0.03)		(0.03)
Mother matric x			0.30	−0.31			−0.10	−0.67
Household income			(0.37)	(0.33)			(0.51)	(0.67)
Mother matric x				0.30				0.38
income sq.				(0.22)				(0.36)
Matric	1.13***	1.05***	1.13***	1.06***	1.48***	1.40***	1.47***	1.39***
	(0.36)	(0.36)	(0.36)	(0.36)	(0.43)	(0.44)	(0.43)	(0.44)
Post matric	0.75**	0.54*	0.75**	0.55*	1.51***	1.34***	1.51***	1.35***
	(0.31)	(0.32)	(0.31)	(0.32)	(0.32)	(0.32)	(0.32)	(0.32)
Observations	7627	7627	7627	7627	6375	6375	6375	6375
Adjusted R^2	0.19	0.20	0.19	0.20	0.11	0.11	0.11	0.11

* p <0.1, ** p <0.05, *** p <0.01. Standard errors in parentheses.

Note: Full set of covariates included but not displayed.

Table A.8.3: BMI OLS regressions for combined urban and rural samples, African and coloured women

	(1)	(2)	(3)	(4)	(5)
Mother matric	−0.97	−1.03	−0.22	−0.09	2.13
	(0.95)	(0.95)	(1.09)	(1.21)	(1.89)
Household income	0.97***	0.99***	1.00***	1.03***	0.98***
	(0.13)	(0.13)	(0.13)	(0.13)	(0.13)
Income sq.		0.10***		0.12***	
		(0.04)		(0.04)	
Urban	0.74***	0.71***	0.74***	0.72***	0.83***
	(0.27)	(0.27)	(0.27)	(0.27)	(0.27)
Mother matric x Household income			−0.98*	−0.98*	
			(0.59)	(0.55)	
Mother matric x income sq.				−0.12	
				(0.32)	
Mother matric x Urban					−4.13**
					(1.84)
Matric	0.55	0.53	0.53	0.51	0.53
	(0.35)	(0.35)	(0.35)	(0.35)	(0.35)
Post matric	1.07***	0.97***	1.06***	0.94***	1.06***
	(0.34)	(0.34)	(0.34)	(0.34)	(0.34)
Observations	20942	20942	20942	20942	20942
Adjusted R^2	0.09	0.10	0.09	0.10	0.10

* p <0.1, ** p <0.05, *** p <0.01. Standard errors in parentheses.
Note: Full set of covariates included but not displayed.

Table A.8.4: BMI OLS regressions for combined urban and rural samples, African and coloured men

	(1)	(2)	(3)	(4)	(5)
Mother matric	0.27	0.20	−0.00	−0.07	0.04
	(0.38)	(0.37)	(0.37)	(0.42)	(0.75)
Household income	1.18***	1.15***	1.17***	1.15***	1.18***
	(0.10)	(0.10)	(0.11)	(0.10)	(0.10)
Income sq.		0.22***		0.21***	
		(0.04)		(0.04)	
Urban	0.41**	0.36*	0.41**	0.37*	0.40**
	(0.19)	(0.19)	(0.19)	(0.19)	(0.20)
Mother matric x Household income			0.31	−0.37	
			(0.32)	(0.30)	
Mother matric x income sq.				0.35*	
				(0.19)	
Mother matric x Urban					0.27
					(0.83)
Matric	1.26***	1.17***	1.26***	1.17***	1.26***
	(0.30)	(0.30)	(0.30)	(0.30)	(0.30)
Post matric	1.01***	0.78***	1.01***	0.79***	1.01***
	(0.25)	(0.26)	(0.25)	(0.26)	(0.25)
Observations	14002	14002	14002	14002	14002
Adjusted R^2	0.17	0.17	0.17	0.17	0.17

* p <0.1, ** p <0.05, *** p <0.01. Standard errors in parentheses.

Note: Full set of covariates included but not displayed.

A.9 Controlling for health behaviours

Table A.9.1: BMI OLS regressions stratified by urban residence showing effect of controlling for adult SES and health behaviours, women

	Urban					Rural				
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
Mother matric	−1.35*	−1.76**	−1.75**	−1.22	−0.90	2.15	0.96	0.93	2.84	2.79
	(0.77)	(0.78)	(0.76)	(0.88)	(0.90)	(1.70)	(1.63)	(1.64)	(2.13)	(2.14)
Household income		0.67***	0.65***	0.70***	0.70***		0.96***	0.96***	1.01***	1.00***
		(0.19)	(0.19)	(0.19)	(0.19)		(0.18)	(0.18)	(0.18)	(0.18)
Income sq.		−0.07	−0.07	−0.02	−0.02		0.06	0.06	0.09	0.08
		(0.05)	(0.05)	(0.05)	(0.05)		(0.07)	(0.07)	(0.07)	(0.07)
Mother matric x Household income				1.24	1.24				−1.33**	−1.32*
				(0.84)	(0.85)				(0.68)	(0.68)
Mother matric x income sq.				−0.71***	−0.80***				−1.40*	−1.38*
				(0.26)	(0.27)				(0.83)	(0.83)
Smoker			−2.68***		−2.80***			−2.58***		−2.56***
			(0.67)		(0.67)			(0.70)		(0.70)
Exercises weekly			−0.77**		−0.80**			−0.28		−0.27
			(0.35)		(0.34)			(0.31)		(0.31)
Matric		−0.44	−0.64	−0.49	−0.72		0.72	0.73	0.72	0.72
		(0.49)	(0.49)	(0.49)	(0.49)		(0.48)	(0.48)	(0.48)	(0.48)
Post matric		0.23	0.10	0.19	0.04		2.31***	2.34***	2.29***	2.32***
		(0.47)	(0.46)	(0.47)	(0.46)		(0.47)	(0.48)	(0.47)	(0.47)
Observations	10821	10821	10821	10821	10821	10892	10892	10892	10892	10892
Adjusted R^2	0.06	0.07	0.08	0.07	0.09	0.08	0.11	0.11	0.11	0.11

* p <0.1, ** p <0.05, *** p <0.01. Standard errors in parentheses.

Note: Full set of covariates included but not displayed.

Table A.9.2: BMI OLS regressions stratified by urban residence showing effect of controlling for adult SES and health behaviours, men

	Urban					Rural				
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
Mother matric	−0.31	−1.21**	−1.20**	0.56	0.62	0.78	−0.47	−0.36	−0.07	−0.04
	(0.52)	(0.48)	(0.50)	(0.47)	(0.49)	(0.71)	(0.70)	(0.60)	(0.92)	(0.86)
Household income		1.18***	1.07***	1.25***	1.13***		0.80***	0.75***	0.81***	0.76***
		(0.12)	(0.12)	(0.13)	(0.13)		(0.12)	(0.12)	(0.13)	(0.13)
Income sq.		0.15***	0.15***	0.23***	0.24***		0.11***	0.12***	0.12***	0.12***
		(0.05)	(0.05)	(0.05)	(0.05)		(0.03)	(0.03)	(0.03)	(0.03)
Mother matric x				−0.21	−0.07				−0.54	−0.53
Household income				(0.38)	(0.41)				(0.64)	(0.55)
Mother matric x				−0.47***	−0.55***				0.05	0.09
income sq.				(0.18)	(0.19)				(0.36)	(0.34)
Smoker			−2.04***		−2.09***			−1.94***		−1.93***
			(0.24)		(0.23)			(0.22)		(0.22)
Exercises weekly			−0.36*		−0.36**			−0.57***		−0.57***
			(0.19)		(0.18)			(0.19)		(0.19)
Matric		0.92***	0.73**	0.83**	0.62*		1.50***	1.21***	1.49***	1.20***
		(0.34)	(0.36)	(0.34)	(0.36)		(0.44)	(0.42)	(0.44)	(0.42)
Post matric		0.66**	0.44	0.56*	0.32		1.40***	1.09***	1.42***	1.10***
		(0.32)	(0.32)	(0.32)	(0.31)		(0.35)	(0.34)	(0.35)	(0.34)
Observations	8112	8112	8112	8112	8112	6488	6488	6488	6488	6488
Adjusted R^2	0.13	0.22	0.25	0.23	0.26	0.06	0.11	0.16	0.11	0.16

* p <0.1, ** p <0.05, *** p <0.01. Standard errors in parentheses.

Note: Full set of covariates included but not displayed.

APPENDIX B

DO GENERATIONAL SHIFTS DRIVE THE OBESITY TRANSITION?

B.1 Model-based trees

B.1.1 Model-based trees stratified by urban or rural residence

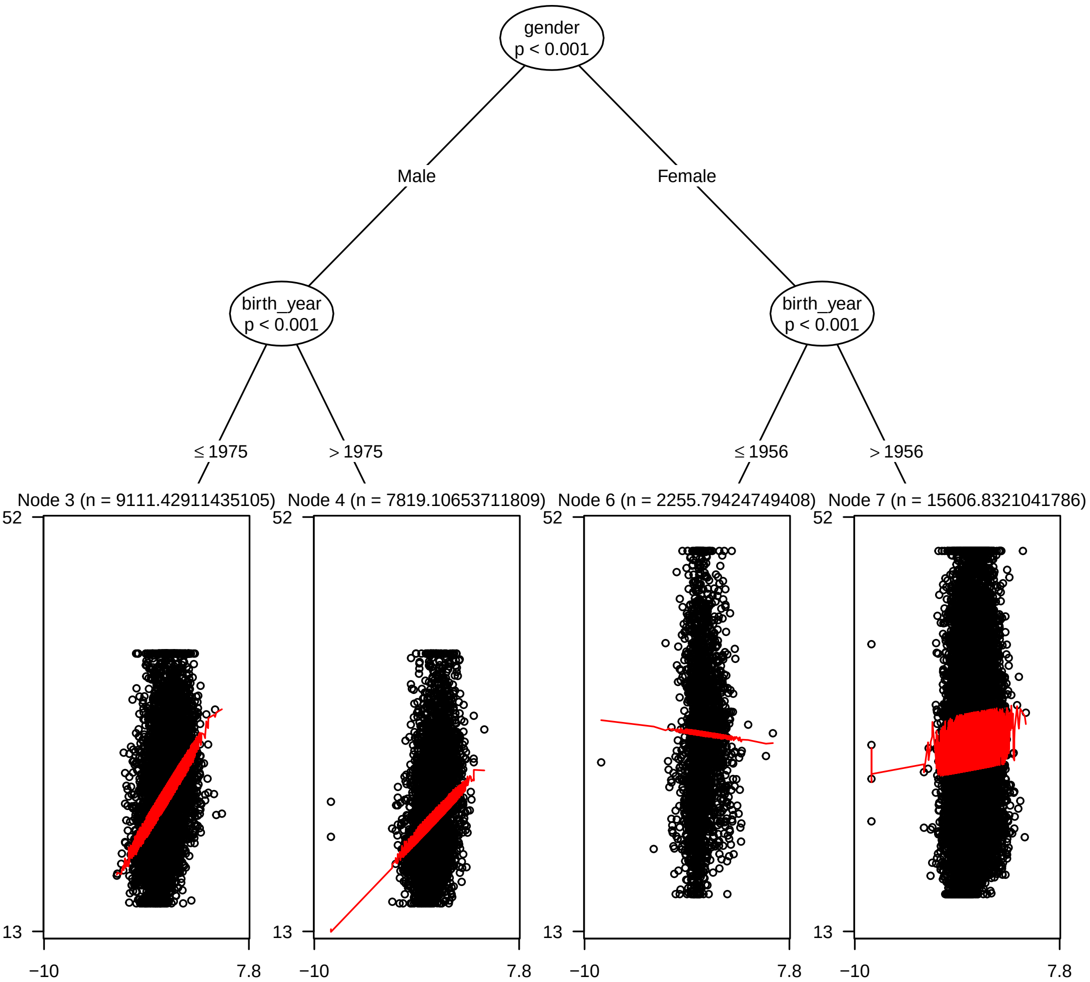


Figure B.1.1: Model-based tree to detect parameter instability in SES-BMI relationship for urban residents

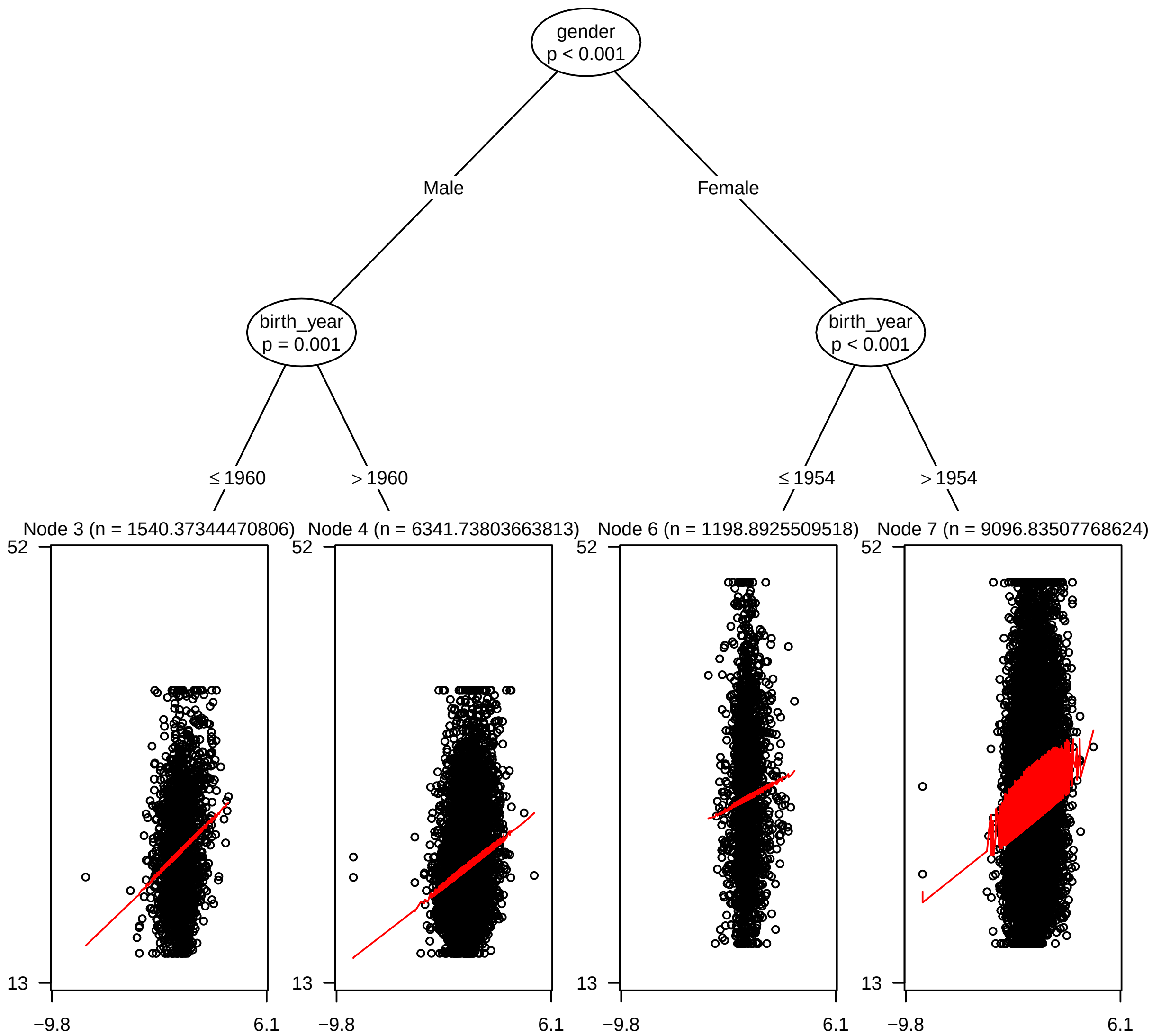


Figure B.1.2: Model-based tree to detect parameter instability in SES-BMI relationship for rural residents

B.1.2 Alternative specification without age control

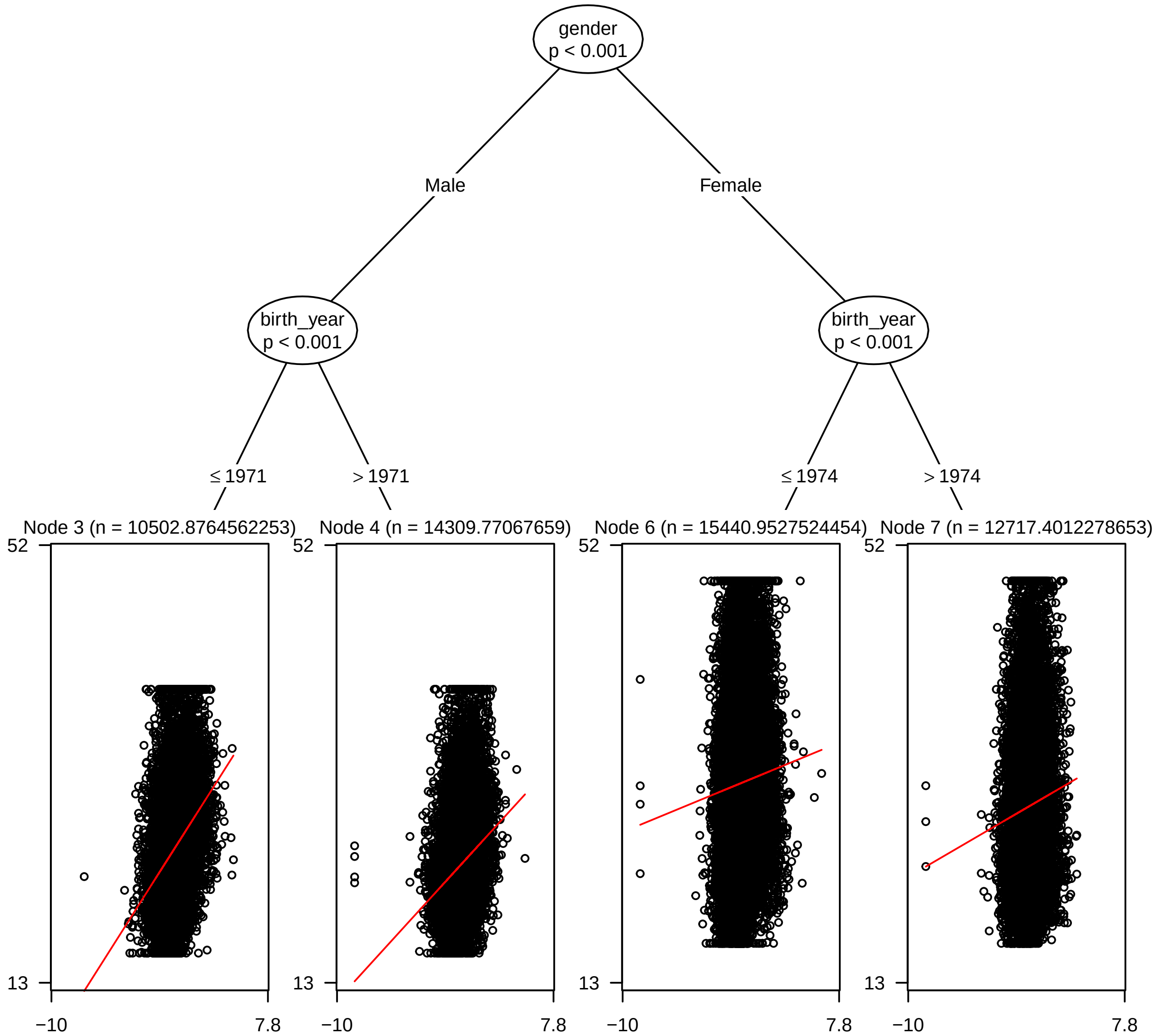


Figure B.1.3: Model-based tree to detect parameter instability in SES-BMI relationship for full sample without age control

Table B.1.1: SES gradients by alternative birth year split (from model-based recursive partitioning algorithm without age control)

	(1) Women	(2) Men
Born 1975 or later	−2.25	
	(1.57)	
Born 1975 or later x Income	0.13	
	(0.18)	
Born 1972 or later		3.69***
		(1.39)
Born 1972 or later x Income		−0.44***
		(0.16)
Income	0.45***	1.19***
	(0.16)	(0.14)
Age	0.06***	0.05***
	(0.02)	(0.01)
Urban	1.01***	0.22
	(0.24)	(0.17)
Constant	22.14***	11.60***
	(1.52)	(1.44)
Observations	31451	20958
Adjusted R^2	0.08	0.23
Controls	Yes	Yes

* p <0.1, ** p <0.05, *** p <0.01. Standard errors in parentheses.

Note: Standard errors clustered by individual. Controls are race group, education, employed, married/cohabiting, province, smoker, exercises weekly, and ever given birth.

B.2 Results for African subsample

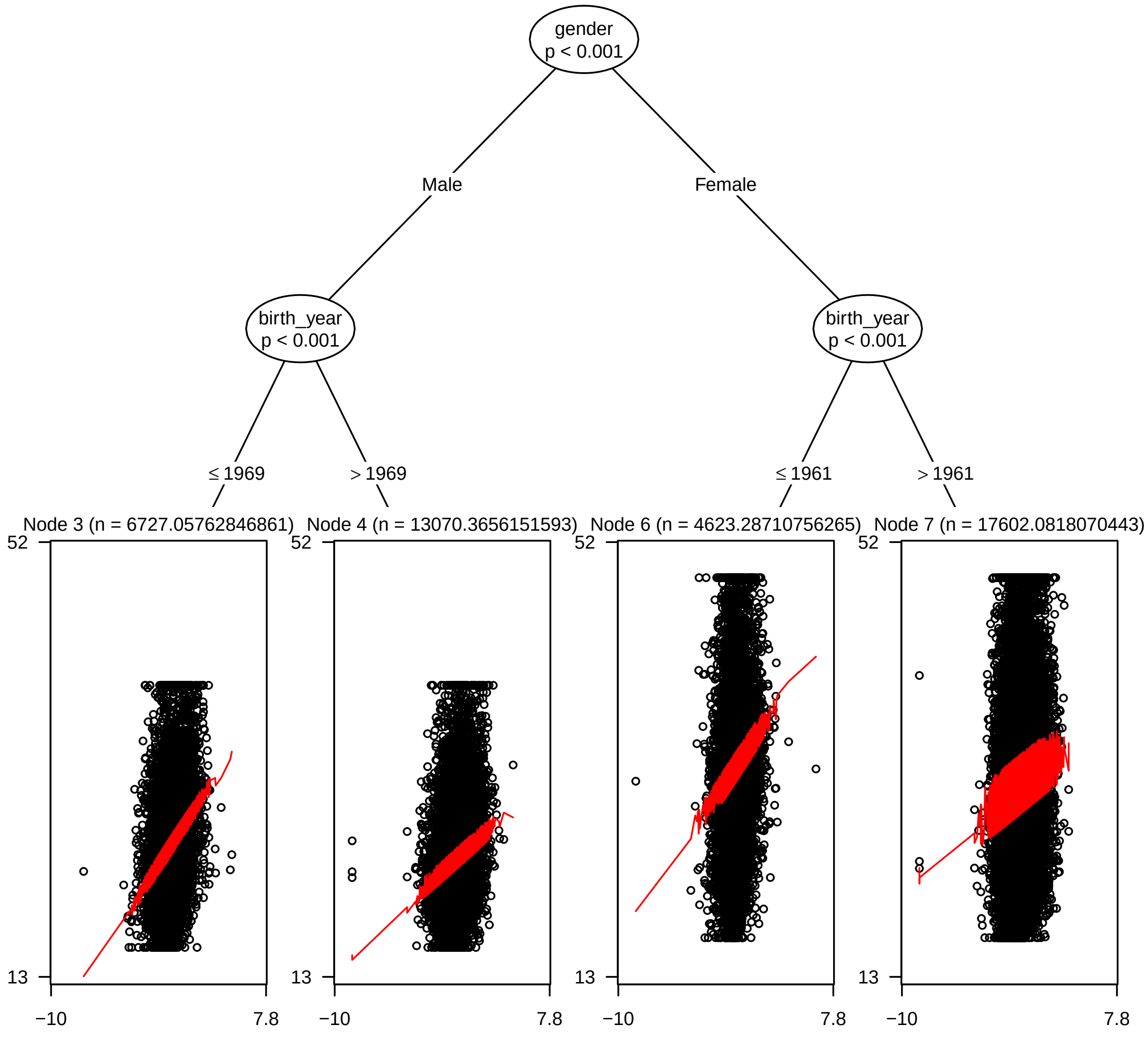


Figure B.2.1: Model-based tree to detect parameter instability in SES-BMI relationship for African subsample (with age control)

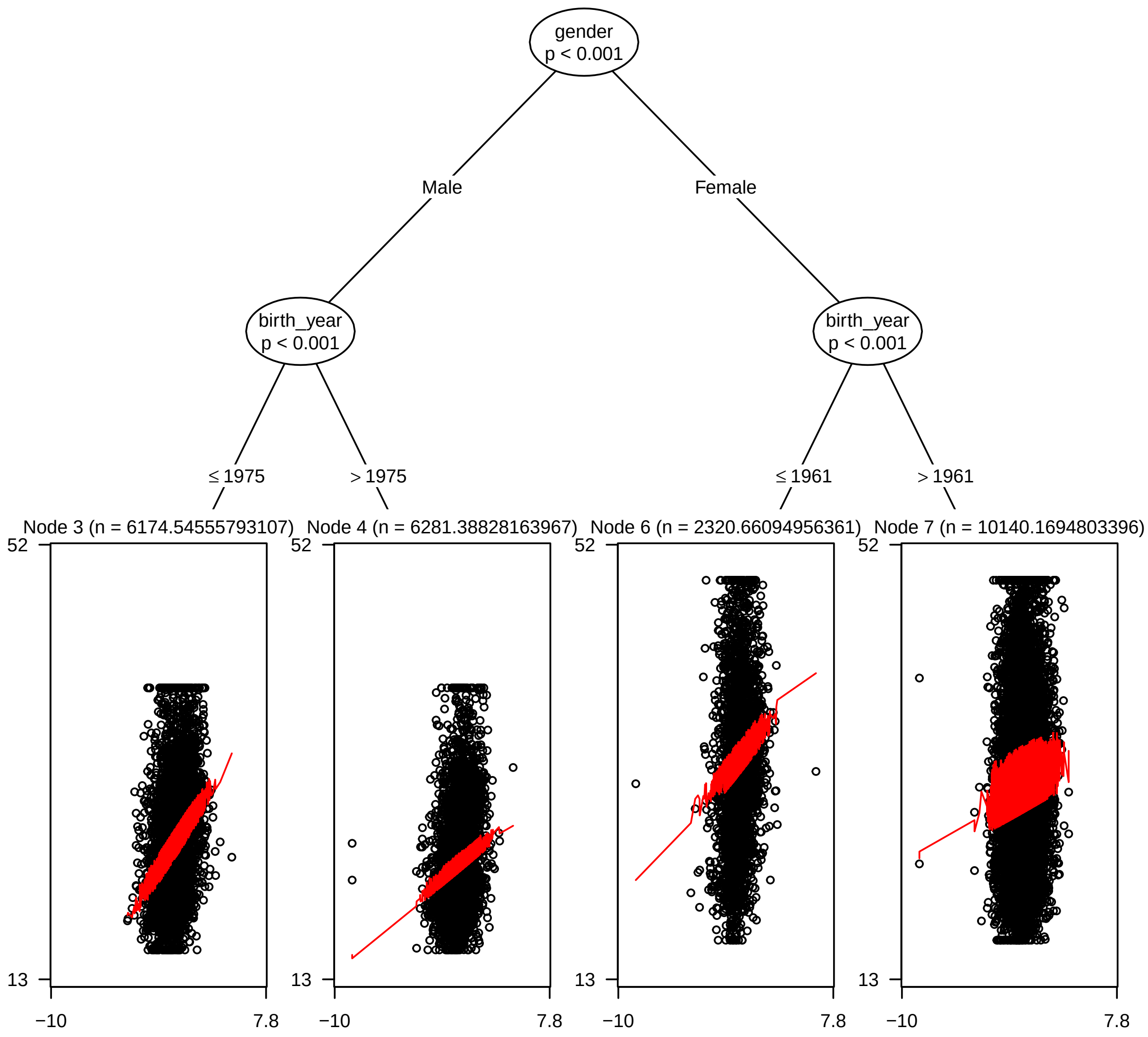


Figure B.2.2: Model-based tree to detect parameter instability in SES-BMI relationship for African urban residents (with age control)

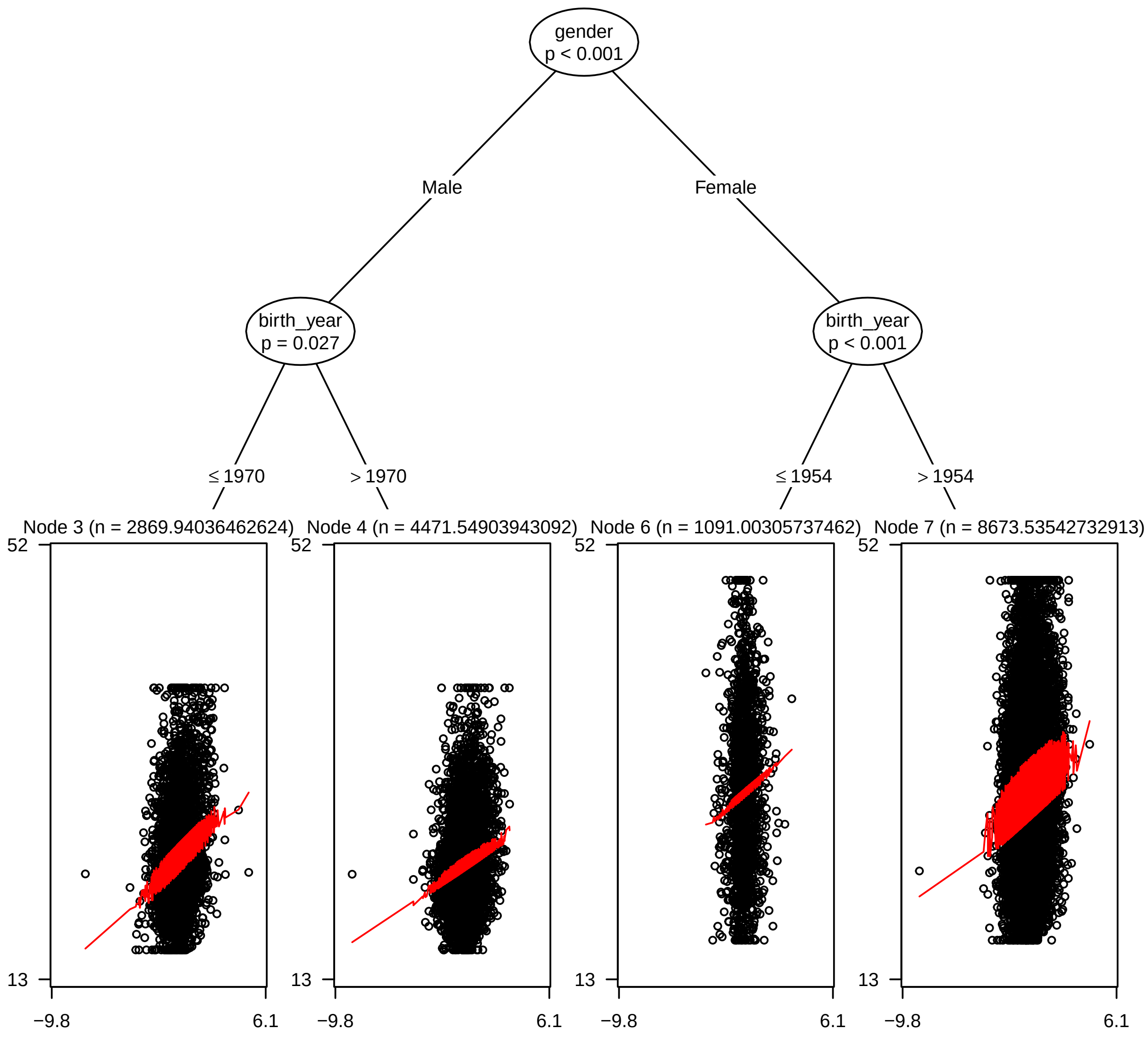


Figure B.2.3: Model-based tree to detect parameter instability in SES-BMI relationship for African rural residents (with age control)

Table B.2.1: SES gradients by age, birth year and wave and urban residence: African subsample

	Age		Cohort		Wave		Urban	
	(1) F	(2) M	(3) F	(4) M	(5) F	(6) M	(7) F	(8) M
Income	−0.68**	−0.50*	1.22***	1.29***	0.52***	0.56***	0.96***	0.71***
	(0.34)	(0.28)	(0.19)	(0.14)	(0.17)	(0.14)	(0.14)	(0.11)
Age	−0.16**	−0.23***	0.14***	0.06***	0.12***	0.07***	0.12***	0.07***
	(0.07)	(0.06)	(0.01)	(0.02)	(0.01)	(0.01)	(0.01)	(0.01)
Age x Household income	0.03***	0.03***						
	(0.01)	(0.01)						
Born 1962 or later			6.07***					
			(1.82)					
Born 1962 or later x Income			−0.66***					
			(0.21)					
Born 1970 or later				5.33***				
				(1.34)				
Born 1970 or later x Income				−0.64***				
				(0.16)				
Wave 2	0.64***	0.27*	0.60***	0.29*	1.11	0.32	0.64***	0.29*
	(0.16)	(0.16)	(0.16)	(0.16)	(1.69)	(1.50)	(0.16)	(0.16)
Wave 3	0.72***	0.38***	0.65***	0.40***	−1.26	−1.62	0.70***	0.42***
	(0.16)	(0.14)	(0.16)	(0.15)	(1.72)	(1.30)	(0.16)	(0.14)
Wave 4	0.80***	0.09	0.71***	0.13	−1.01	−5.67***	0.78***	0.12
	(0.16)	(0.15)	(0.17)	(0.16)	(1.69)	(1.68)	(0.16)	(0.15)
Wave 5	0.78***	−0.17	0.68***	−0.12	−2.16	−5.15***	0.76***	−0.15
	(0.17)	(0.16)	(0.19)	(0.17)	(1.73)	(1.59)	(0.17)	(0.16)
Wave 2 x Income					−0.05	−0.00		
					(0.21)	(0.18)		
Wave 3 x Income					0.24	0.24		
					(0.21)	(0.15)		
Wave 4 x Income					0.22	0.67***		
					(0.20)	(0.20)		
Wave 5 x Income					0.35*	0.57***		
					(0.21)	(0.19)		
Urban	1.14***	0.31*	1.16***	0.31*	1.17***	0.34*	4.95***	−1.83
	(0.24)	(0.17)	(0.24)	(0.17)	(0.25)	(0.17)	(1.61)	(1.23)
Urban x Income							−0.45**	0.25*
							(0.19)	(0.14)
Constant	28.53***	25.36***	11.61***	10.30***	18.25***	16.19***	14.71***	14.88***
	(2.94)	(2.37)	(1.80)	(1.33)	(1.54)	(1.23)	(1.33)	(1.01)
Observations	25669	16633	25669	16633	25669	16633	25669	16633
Adjusted R^2	0.10	0.19	0.10	0.19	0.10	0.18	0.10	0.18

* p <0.1, ** p <0.05, *** p <0.01. Standard errors in parentheses.

Note: Standard errors clustered by individual. Controls are education, employed, married/cohabiting, province, smoker, exercises weekly, and ever given birth.

Table B.2.2: SES gradients by age, birth year and wave, stratified by urban residence: women, African subsample

	Urban			Rural		
	(1) Age	(2) Cohort	(3) Wave	(4) Age	(5) Cohort	(6) Wave
Income	−0.62	1.04***	0.23	0.01	1.01***	0.99***
	(0.51)	(0.27)	(0.23)	(0.46)	(0.39)	(0.22)
Age	−0.11	0.16***	0.15***	−0.02	0.14***	0.14***
	(0.11)	(0.02)	(0.02)	(0.09)	(0.01)	(0.01)
Age x Household income	0.03***			0.02*		
	(0.01)			(0.01)		
Born 1962 or later	0.36	5.02*	0.39			
	(0.66)	(2.78)	(0.66)			
Born 1962 or later x Income		−0.54*				
		(0.31)				
Born 1955 or later				2.17***	3.79	2.22***
				(0.49)	(3.23)	(0.49)
Born 1955 or later x Income					−0.19	
					(0.41)	
Wave 2	0.70***	0.68***	−0.84	0.44**	0.43**	4.30**
	(0.25)	(0.25)	(2.59)	(0.20)	(0.20)	(2.07)
Wave 3	0.62**	0.61**	−3.11	0.63***	0.63***	0.60
	(0.25)	(0.25)	(2.48)	(0.19)	(0.20)	(2.12)
Wave 4	0.66**	0.66**	−4.65*	0.61***	0.60***	3.22
	(0.26)	(0.26)	(2.46)	(0.21)	(0.21)	(2.40)
Wave 5	0.81***	0.79***	−4.27*	0.27	0.26	−0.27
	(0.29)	(0.29)	(2.46)	(0.23)	(0.23)	(2.30)
Wave 2 x Income			0.18			−0.49*
			(0.30)			(0.26)
Wave 3 x Income			0.44			−0.00
			(0.29)			(0.27)
Wave 4 x Income			0.62**			−0.32
			(0.28)			(0.30)
Wave 5 x Income			0.59**			0.05
			(0.29)			(0.28)
Constant	28.21***	13.82***	20.65***	19.30***	11.10**	11.34***
	(4.73)	(2.63)	(2.31)	(5.54)	(4.99)	(4.27)
Observations	11252	11252	11252	14417	14417	14417
Adjusted R^2	0.09	0.09	0.09	0.10	0.10	0.10
Controls	Yes	Yes	Yes	Yes	Yes	Yes

* p <0.1, ** p <0.05, *** p <0.01. Standard errors in parentheses.

Note: Standard errors clustered by individual. Controls are education, employed, married/cohabiting, province, smoker, exercises weekly, and ever given birth.

Table B.2.3: SES gradients by age, birth year and wave, stratified by urban residence: men, African subsample

	Urban			Rural		
	(1) Age	(2) Cohort	(3) Wave	(4) Age	(5) Cohort	(6) Wave
Income	−0.54	1.24***	0.55***	−0.15	1.03***	0.52**
	(0.39)	(0.15)	(0.18)	(0.33)	(0.17)	(0.23)
Age	−0.26***	0.08***	0.07***	−0.11	0.07***	0.07***
	(0.08)	(0.02)	(0.02)	(0.07)	(0.02)	(0.02)
Age x Household income	0.04***			0.02***		
	(0.01)			(0.01)		
Born 1976 or later	−0.27	5.16***	−0.34			
	(0.40)	(1.84)	(0.41)			
Born 1976 or later x Income		−0.61***				
		(0.21)				
Born 1971 or later				0.62	5.08***	0.59
				(0.42)	(1.66)	(0.42)
Born 1971 or later x Income					−0.53***	
					(0.20)	
Wave 2	0.20	0.19	0.33	0.42	0.41	−0.38
	(0.21)	(0.22)	(1.91)	(0.25)	(0.25)	(2.87)
Wave 3	0.46**	0.46**	−2.35	0.30	0.28	0.05
	(0.20)	(0.20)	(1.74)	(0.23)	(0.23)	(2.20)
Wave 4	0.31	0.32	−6.82***	−0.20	−0.20	−3.19
	(0.22)	(0.22)	(2.42)	(0.23)	(0.23)	(2.19)
Wave 5	−0.03	−0.00	−5.81***	−0.33	−0.32	−4.06*
	(0.24)	(0.24)	(2.19)	(0.27)	(0.27)	(2.20)
Wave 2 x Income			−0.02			0.10
			(0.22)			(0.36)
Wave 3 x Income			0.33*			0.04
			(0.20)			(0.27)
Wave 4 x Income			0.81***			0.36
			(0.27)			(0.27)
Wave 5 x Income			0.66***			0.45
			(0.25)			(0.27)
Constant	26.07***	10.24***	16.54***	22.59***	12.62***	17.00***
	(3.47)	(1.48)	(1.70)	(3.28)	(2.21)	(2.53)
Observations	8289	8289	8289	8344	8344	8344
Adjusted R^2	0.20	0.20	0.20	0.16	0.16	0.16
Controls	Yes	Yes	Yes	Yes	Yes	Yes

* p <0.1, ** p <0.05, *** p <0.01. Standard errors in parentheses.

Note: Standard errors clustered by individual. Controls are education, employed, married/cohabiting, province, smoker, and exercises weekly.

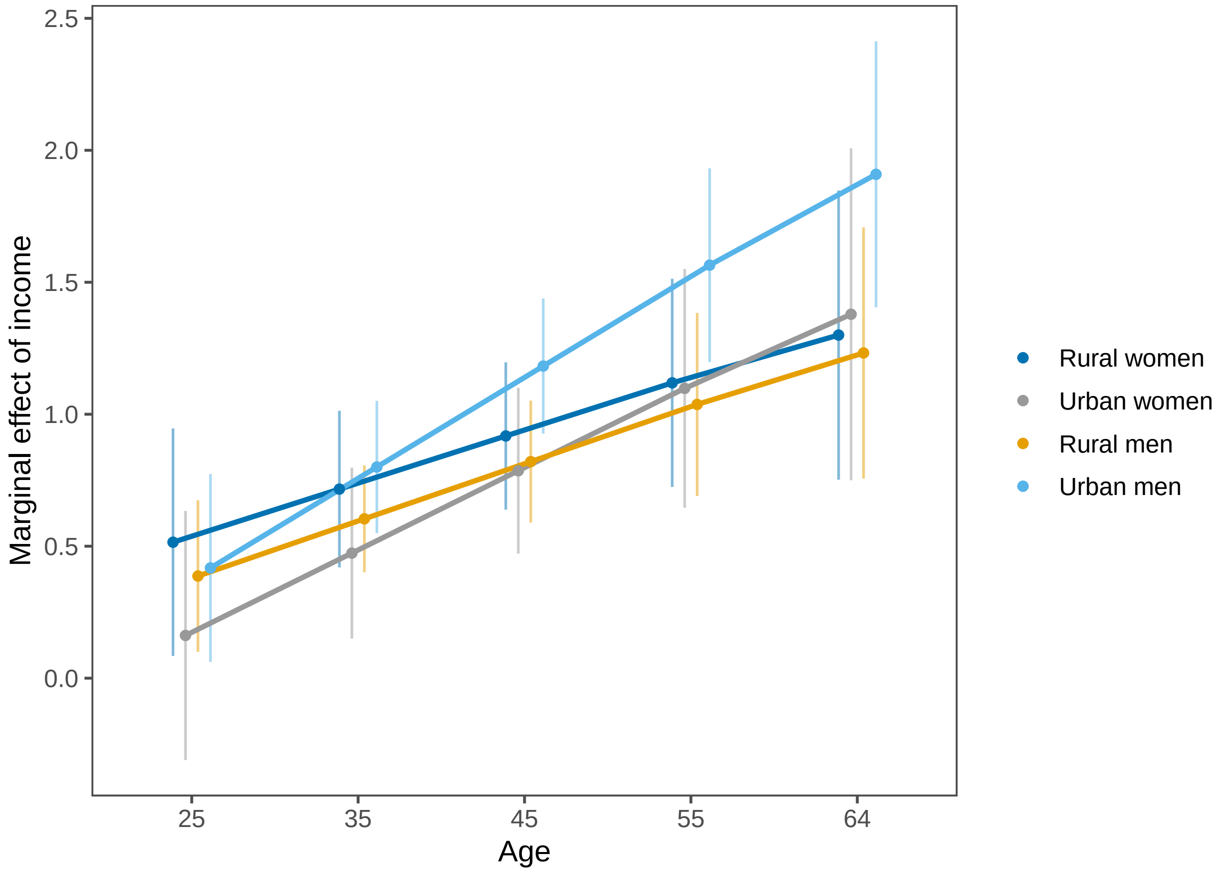


Figure B.2.4: Marginal effect of income by age

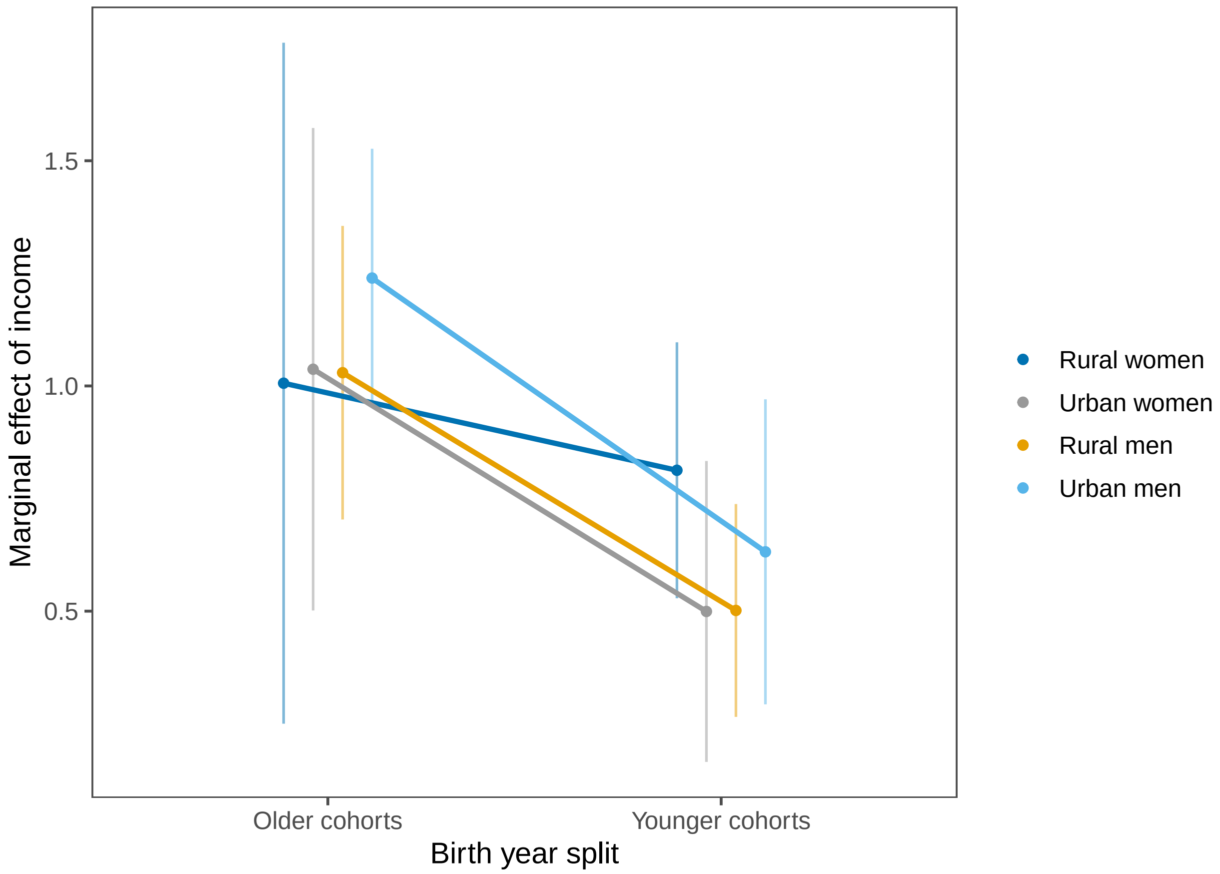


Figure B.2.5: Marginal effect of income by cohort based on birth year splits detected by model-based trees

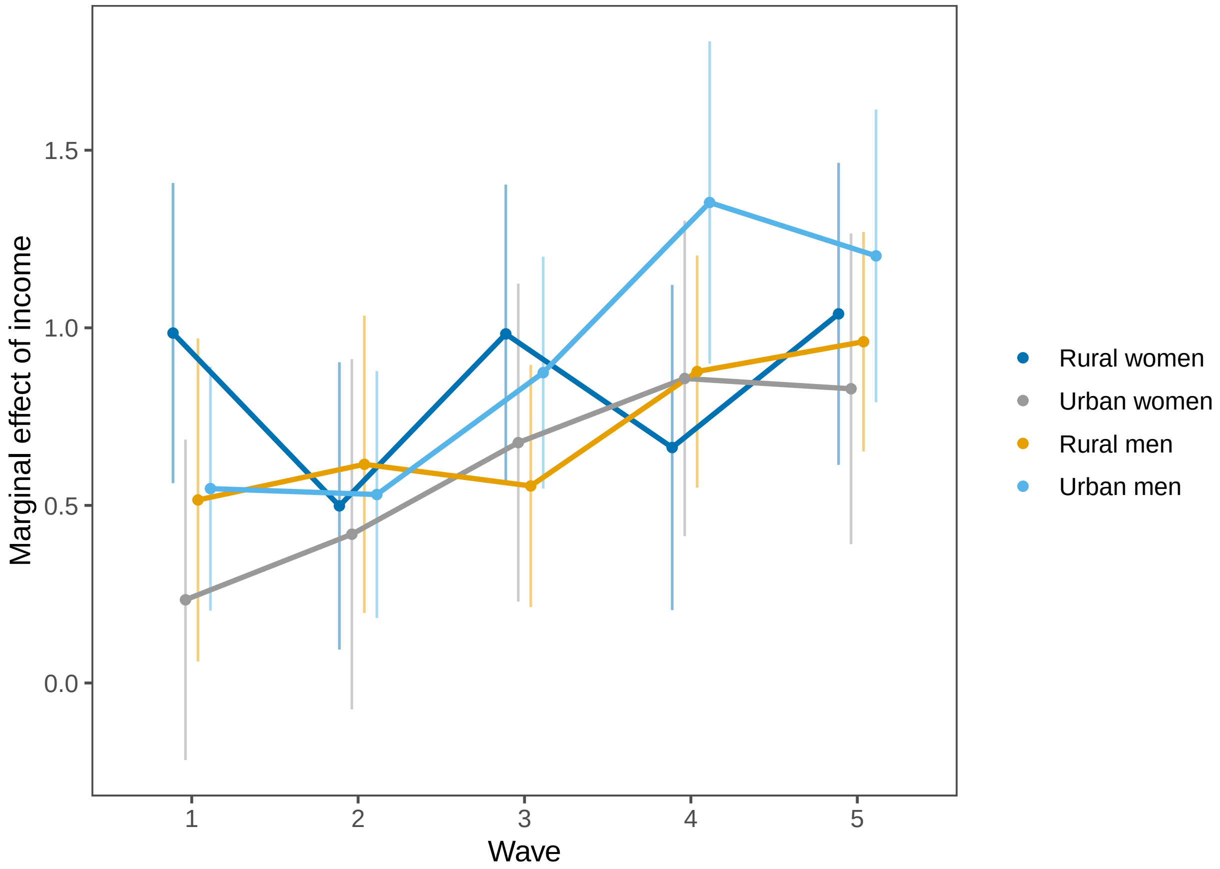


Figure B.2.6: Marginal effect of income by wave

B.3 Results for obesity

Table B.3.1: SES gradients in obesity by age, birth year, wave and urban residence

	Women				Men			
	(1) Age	(2) Cohort	(3) Wave	(4) Urban	(5) Age	(6) Cohort	(7) Wave	(8) Urban
Age	0.01	0.01***	0.01***	0.01***	−0.01**	0.00**	0.00**	0.00**
	(0.01)	(0.00)	(0.00)	(0.00)	(0.01)	(0.00)	(0.00)	(0.00)
Age x Household income	0.00				0.00***			
	(0.00)				(0.00)			
Born 1955 or later	0.09***	0.43**	0.09***	0.09***				
	(0.03)	(0.18)	(0.03)	(0.03)				
Born 1955 or later x Income		−0.04*						
		(0.02)						
Born 1968 or later					−0.03	0.45***	−0.03	−0.03
					(0.03)	(0.14)	(0.03)	(0.03)
Born 1968 or later x Income						−0.05***		
						(0.02)		
Wave 2	0.03**	0.03**	0.05	0.03**	0.02	0.02	−0.10	0.02
	(0.01)	(0.01)	(0.11)	(0.01)	(0.01)	(0.01)	(0.12)	(0.01)
Wave 3	0.05***	0.04***	−0.13	0.04***	0.01	0.01	−0.09	0.01
	(0.01)	(0.01)	(0.11)	(0.01)	(0.01)	(0.01)	(0.11)	(0.01)
Wave 4	0.06***	0.06***	−0.08	0.06***	0.01	0.01	−0.15	0.01
	(0.01)	(0.01)	(0.12)	(0.01)	(0.01)	(0.01)	(0.12)	(0.01)
Wave 5	0.05***	0.05***	−0.09	0.04***	−0.00	−0.00	−0.14	−0.00
	(0.01)	(0.01)	(0.11)	(0.01)	(0.01)	(0.01)	(0.10)	(0.01)
Wave 2 x Income			−0.00				0.01	
			(0.01)				(0.01)	
Wave 3 x Income			0.02				0.01	
			(0.01)				(0.01)	
Wave 4 x Income			0.02				0.02	
			(0.01)				(0.01)	
Wave 5 x Income			0.02				0.02	
			(0.01)				(0.01)	
Urban	0.06***	0.06***	0.06***	0.43***	0.02*	0.02	0.02*	−0.09
	(0.02)	(0.02)	(0.02)	(0.11)	(0.01)	(0.01)	(0.01)	(0.09)
Urban x Income				−0.04***				0.01
				(0.01)				(0.01)
Income	0.02	0.06***	0.02	0.06***	−0.03	0.08***	0.03***	0.03***
	(0.02)	(0.02)	(0.01)	(0.01)	(0.02)	(0.02)	(0.01)	(0.01)
Coloured	−0.02	−0.03	−0.02	−0.02	0.03	0.03	0.03	0.03
	(0.03)	(0.03)	(0.03)	(0.03)	(0.02)	(0.02)	(0.02)	(0.02)
Asian/Indian	−0.24***	−0.24***	−0.24***	−0.23***	−0.09**	−0.08**	−0.09**	−0.09**
	(0.06)	(0.06)	(0.06)	(0.06)	(0.04)	(0.04)	(0.04)	(0.04)
White	−0.10**	−0.10**	−0.10**	−0.08*	0.05	0.05	0.07*	0.06
	(0.05)	(0.05)	(0.05)	(0.05)	(0.04)	(0.04)	(0.04)	(0.04)
Constant	−0.25	−0.60***	−0.20*	−0.54***	0.37*	−0.64***	−0.21**	−0.24***
	(0.21)	(0.20)	(0.12)	(0.11)	(0.22)	(0.15)	(0.10)	(0.09)
Observations	31451	31451	31451	31451	20958	20958	20958	20958
Adjusted R^2	0.06	0.06	0.06	0.06	0.10	0.11	0.10	0.10
Controls	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes

* p <0.1, ** p <0.05, *** p <0.01. Standard errors in parentheses.

Note: Regressions are linear probability models of obesity. Standard errors clustered by individual. Controls are race group, education, employed, married/cohabiting, province, smoker, exercises weekly, and ever given birth.

B.4 Results controlling for quadratic age

Table B.4.1: SES gradients by age, birth year, wave and urban residence with quadratic age specification: women

	Age		Cohort		Wave		Urban	
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Age	0.44	0.48	0.47***	0.44***	0.47***	0.44***	0.48***	0.44***
	(0.55)	(0.52)	(0.08)	(0.08)	(0.08)	(0.08)	(0.08)	(0.08)
Age x Household income	0.00	−0.00						
	(0.06)	(0.06)						
Age sq.	−0.00	−0.00	−0.00***	−0.00***	−0.00***	−0.00***	−0.00***	−0.00***
	(0.01)	(0.01)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
Age sq. x Household income	−0.00	0.00						
	(0.00)	(0.00)						
Born 1954 or later	0.83	0.77	1.87	3.14	0.81	0.76	0.84	0.81
	(0.55)	(0.54)	(2.79)	(2.77)	(0.55)	(0.54)	(0.55)	(0.54)
Born 1954 or later x Income			−0.12	−0.27				
			(0.33)	(0.33)				
Wave 2	0.73***	0.66***	0.73***	0.65***	−0.00	0.22	0.71***	0.64***
	(0.17)	(0.17)	(0.17)	(0.17)	(1.40)	(1.35)	(0.16)	(0.17)
Wave 3	0.87***	0.86***	0.87***	0.85***	−2.54*	−2.41*	0.81***	0.81***
	(0.16)	(0.17)	(0.16)	(0.17)	(1.42)	(1.42)	(0.16)	(0.17)
Wave 4	1.07***	1.05***	1.07***	1.04***	−2.03	−1.79	1.00***	1.00***
	(0.17)	(0.18)	(0.17)	(0.18)	(1.51)	(1.48)	(0.17)	(0.18)
Wave 5	0.92***	0.81***	0.92***	0.80***	−2.97**	−2.37*	0.84***	0.75***
	(0.19)	(0.19)	(0.19)	(0.19)	(1.42)	(1.38)	(0.18)	(0.18)
Wave 2 x Income					0.09	0.05		
					(0.17)	(0.16)		
Wave 3 x Income					0.40**	0.38**		
					(0.17)	(0.17)		
Wave 4 x Income					0.36**	0.33*		
					(0.18)	(0.17)		
Wave 5 x Income					0.45***	0.37**		
					(0.17)	(0.16)		
Urban	0.81***	1.02***	0.81***	1.02***	0.81***	1.02***	7.28***	6.87***
	(0.21)	(0.24)	(0.21)	(0.24)	(0.21)	(0.24)	(1.56)	(1.59)
Urban x Income							−0.77***	−0.70***
							(0.18)	(0.19)
Income	0.30	0.58	0.45	0.79**	0.08	0.31**	0.91***	1.02***
	(1.28)	(1.24)	(0.31)	(0.34)	(0.13)	(0.15)	(0.14)	(0.15)
Constant	13.53	11.81	12.21***	9.96***	15.42***	14.08***	8.40***	8.13***
	(10.85)	(10.57)	(3.03)	(3.29)	(1.75)	(1.91)	(1.78)	(1.89)
Observations	31451	31451	31451	31451	31451	31451	31451	31451
Adjusted R^2	0.05	0.08	0.05	0.08	0.05	0.08	0.05	0.08
Controls	No	Yes	No	Yes	No	Yes	No	Yes

* p <0.1, ** p <0.05, *** p <0.01. Standard errors in parentheses.

Note: Standard errors clustered by individual. Controls are race group, education, employed, married/cohabiting, province, smoker, exercises weekly, and ever given birth.

Table B.4.2: SES gradients by age, birth year, wave and urban residence with quadratic age specification: men

	Age		Cohort		Wave		Urban	
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Age	−0.72	−0.53	0.14***	0.08	0.14**	0.08	0.13**	0.07
	(0.46)	(0.47)	(0.05)	(0.05)	(0.05)	(0.05)	(0.05)	(0.05)
Age x Household income	0.10*	0.07						
	(0.05)	(0.05)						
Age sq.	0.01	0.00	−0.00	−0.00	−0.00	−0.00	−0.00	−0.00
	(0.01)	(0.01)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
Age sq. x Household income	−0.00	−0.00						
	(0.00)	(0.00)						
Born 1970 or later	−0.23	−0.35	4.58***	4.05***	−0.26	−0.37	−0.22	−0.34
	(0.35)	(0.33)	(1.50)	(1.43)	(0.35)	(0.34)	(0.35)	(0.33)
Born 1970 or later x Income			−0.54***	−0.49***				
			(0.17)	(0.16)				
Wave 2	0.14	0.16	0.13	0.16	−0.45	−0.21	0.16	0.18
	(0.16)	(0.16)	(0.16)	(0.16)	(1.48)	(1.44)	(0.16)	(0.16)
Wave 3	0.24	0.37***	0.24	0.37**	−1.77	−0.95	0.29*	0.40***
	(0.15)	(0.14)	(0.15)	(0.14)	(1.41)	(1.31)	(0.15)	(0.15)
Wave 4	−0.11	0.07	−0.13	0.06	−4.58***	−3.83***	−0.08	0.09
	(0.16)	(0.16)	(0.16)	(0.16)	(1.62)	(1.48)	(0.16)	(0.16)
Wave 5	−0.39**	−0.20	−0.40**	−0.20	−4.62***	−3.70***	−0.37**	−0.19
	(0.17)	(0.17)	(0.17)	(0.16)	(1.40)	(1.32)	(0.17)	(0.17)
Wave 2 x Income					0.07	0.04		
					(0.17)	(0.17)		
Wave 3 x Income					0.23	0.15		
					(0.16)	(0.15)		
Wave 4 x Income					0.50***	0.44***		
					(0.18)	(0.17)		
Wave 5 x Income					0.47***	0.39***		
					(0.16)	(0.15)		
Urban	0.20	0.22	0.19	0.22	0.23	0.26	−4.49***	−2.35*
	(0.15)	(0.17)	(0.15)	(0.17)	(0.15)	(0.17)	(1.26)	(1.22)
Urban x Income							0.55***	0.30**
							(0.15)	(0.14)
Income	−1.02	−0.86	1.74***	1.23***	1.16***	0.72***	1.03***	0.72***
	(1.05)	(1.06)	(0.14)	(0.15)	(0.13)	(0.13)	(0.11)	(0.11)
Constant	29.17***	29.33***	4.65***	10.73***	9.96***	15.36***	11.12***	15.38***
	(9.15)	(9.31)	(1.69)	(1.79)	(1.59)	(1.59)	(1.48)	(1.49)
Observations	20958	20958	20958	20958	20958	20958	20958	20958
Adjusted R^2	0.15	0.23	0.15	0.23	0.15	0.23	0.15	0.23
Controls	No	Yes	No	Yes	No	Yes	No	Yes

* p <0.1, ** p <0.05, *** p <0.01. Standard errors in parentheses.

Note: Standard errors clustered by individual. Controls are race group, education, employed, married/cohabiting, province, smoker, and exercises weekly.

Table B.4.3: SES gradients by age, birth year and wave, stratified by urban residence with quadratic age specification: women

	Urban			Rural		
	(1) Age	(2) Cohort	(3) Wave	(4) Age	(5) Cohort	(6) Wave
Income	0.48	0.01	0.06	1.64	1.37***	0.99***
	(1.66)	(0.41)	(0.20)	(1.65)	(0.34)	(0.20)
Age	0.45	0.46***	0.47***	0.86	0.35***	0.34***
	(0.74)	(0.11)	(0.11)	(0.68)	(0.09)	(0.09)
Age x Household income	0.00			−0.06		
	(0.08)			(0.08)		
Age sq.	−0.00	−0.00***	−0.00***	−0.01	−0.00**	−0.00**
	(0.01)	(0.00)	(0.00)	(0.01)	(0.00)	(0.00)
Age sq. x Household income	−0.00			0.00		
	(0.00)			(0.00)		
Born 1957 or later	0.42	−3.64	0.39			
	(0.76)	(3.64)	(0.76)			
Born 1957 or later x Income		0.45				
		(0.40)				
Born 1955 or later				1.28**	5.80**	1.38**
				(0.58)	(2.85)	(0.59)
Born 1955 or later x Income					−0.54	
					(0.35)	
Wave 2	0.77***	0.78***	−0.70	0.45**	0.43**	3.94**
	(0.24)	(0.24)	(1.85)	(0.19)	(0.19)	(1.89)
Wave 3	0.89***	0.90***	−3.69**	0.69***	0.67***	0.16
	(0.24)	(0.24)	(1.87)	(0.19)	(0.19)	(1.93)
Wave 4	1.19***	1.20***	−3.73*	0.66***	0.64***	3.17
	(0.25)	(0.25)	(2.02)	(0.21)	(0.21)	(2.25)
Wave 5	0.95***	0.96***	−3.36*	0.36	0.34	−0.46
	(0.25)	(0.25)	(1.84)	(0.23)	(0.23)	(2.18)
Wave 2 x Income			0.17			−0.44*
			(0.21)			(0.24)
Wave 3 x Income			0.52**			0.06
			(0.22)			(0.24)
Wave 4 x Income			0.55**			−0.31
			(0.23)			(0.28)
Wave 5 x Income			0.49**			0.09
			(0.21)			(0.27)
Constant	13.58	17.87***	17.20***	4.17	6.53*	9.76***
	(14.78)	(4.26)	(2.53)	(13.49)	(3.51)	(2.54)
Observations	16222	16222	16222	15229	15229	15229
Adjusted R^2	0.08	0.08	0.08	0.10	0.10	0.10
Controls	Yes	Yes	Yes	Yes	Yes	Yes

* p <0.1, ** p <0.05, *** p <0.01. Standard errors in parentheses.

Note: Standard errors clustered by individual. Controls are race group, education, employed, married/cohabiting, province, smoker, exercises weekly, and ever given birth.

Table B.4.4: SES gradients by age, birth year and wave, stratified by urban residence with quadratic age specification: men

	Urban			Rural		
	(1) Age	(2) Cohort	(3) Wave	(4) Age	(5) Cohort	(6) Wave
Income	−0.84	1.24***	0.78***	−0.18	1.05***	0.55**
	(1.40)	(0.15)	(0.17)	(1.39)	(0.21)	(0.23)
Age	−0.60	0.07	0.06	−0.09	0.13*	0.12
	(0.65)	(0.09)	(0.09)	(0.57)	(0.07)	(0.07)
Age x Household income	0.07			0.03		
	(0.07)			(0.07)		
Age sq.	0.01	−0.00	−0.00	−0.00	−0.00*	−0.00
	(0.01)	(0.00)	(0.00)	(0.01)	(0.00)	(0.00)
Age sq. x Household income	−0.00			−0.00		
	(0.00)			(0.00)		
Born 1976 or later	−0.48	3.74**	−0.50			
	(0.39)	(1.76)	(0.39)			
Born 1976 or later x Income		−0.46**				
		(0.19)				
Born 1961 or later				−1.47***	2.25	−1.34***
				(0.46)	(1.92)	(0.46)
Born 1961 or later x Income					−0.44*	
					(0.23)	
Wave 2	0.09	0.08	−0.89	0.38	0.37	0.86
	(0.21)	(0.21)	(1.85)	(0.25)	(0.24)	(2.70)
Wave 3	0.41**	0.40**	−1.11	0.51**	0.50**	−0.18
	(0.19)	(0.19)	(1.66)	(0.22)	(0.22)	(2.23)
Wave 4	0.21	0.21	−4.08**	0.07	0.05	−2.16
	(0.21)	(0.21)	(2.00)	(0.22)	(0.22)	(2.23)
Wave 5	−0.11	−0.11	−3.79**	0.00	−0.01	−3.29
	(0.23)	(0.22)	(1.72)	(0.26)	(0.25)	(2.14)
Wave 2 x Income			0.11			−0.05
			(0.21)			(0.34)
Wave 3 x Income			0.17			0.09
			(0.18)			(0.27)
Wave 4 x Income			0.47**			0.27
			(0.22)			(0.27)
Wave 5 x Income			0.40**			0.39
			(0.19)			(0.26)
Constant	29.84**	10.99***	15.27***	24.08**	13.69***	17.95***
	(12.65)	(2.41)	(2.44)	(11.36)	(2.33)	(2.29)
Observations	11896	11896	11896	9062	9062	9062
Adjusted R^2	0.25	0.25	0.24	0.16	0.16	0.16
Controls	Yes	Yes	Yes	Yes	Yes	Yes

* p <0.1, ** p <0.05, *** p <0.01. Standard errors in parentheses.

Note: Standard errors clustered by individual. Controls are race group, education, employed, married/cohabiting, province, smoker, and exercises weekly.

B.5 Seemingly unrelated regressions

We explore whether there are significant differences in the SES gradients between men and women in urban and rural areas using seemingly unrelated regressions. One way to compare the coefficients on income would be to interact income with an indicator for gender and an indicator for urban residence. However, the specifications of the regressions for men and women are slightly different: for women we include an indicator for whether she has ever given birth. We therefore use seemingly unrelated regressions (using Stata's suest command) to test for differences between the coefficients. The regressions are estimated as a system of equations, allowing for correlation between the errors (Clogg et al., 1995; StataCorp, 2021).

The results are shown in Table B.5.1. The coefficient on income is smallest for urban women and largest for urban men. Among men, the coefficient on income is significantly smaller in rural areas, while among women, the coefficient on income is significantly smaller in urban areas. In urban areas, the coefficient on income is significantly larger for men than for women, while in rural areas the coefficient is smaller for men, but not significantly so.

Table B.5.1: Differences in income coefficients from seemingly unrelated regressions

	Coefficient	Standard error
Urban women	0.40**	(0.17)
Rural women	0.87***	(0.14)
Urban men	1.03***	(0.12)
Rural men	0.69***	(0.11)
	Difference in coefficient	p-value of difference
Urban vs rural, men	−0.35**	(0.033)
Urban vs rural, women	0.47**	(0.032)
Men vs women, urban	0.63***	(0.003)
Men vs women, rural	−0.18	(0.292)

APPENDIX C

THE ASSOCIATION BETWEEN ECONOMIC INSECURITY AND OBESITY MAY DEPEND ON ACCESS TO EXCESS CALORIES: EVIDENCE FROM SOUTH AFRICA

C.1 Economic insecurity measures

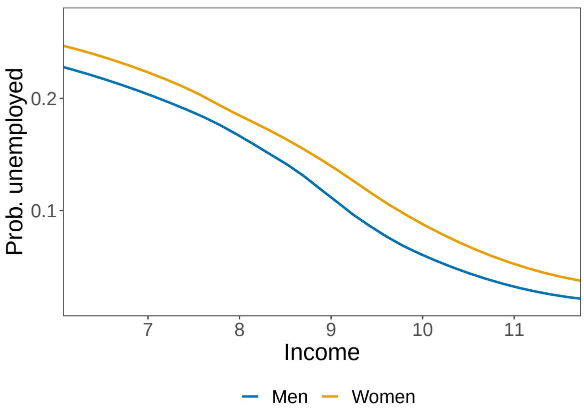
Table C.1.1: Probit regressions predicting probability of unemployment and of poverty in following wave

	(1) Unemployed		(2) Income drop		(3) Poor (food)		(4) Poor (upper-bound)	
Unemployed discouraged	0.27***	(0.05)	−0.12***	(0.05)	0.20***	(0.04)	0.01	(0.05)
Unemployed strict	0.26***	(0.03)	−0.13***	(0.03)	0.07***	(0.03)	0.04	(0.03)
Employed	0.02	(0.08)	0.01	(0.04)	0.12***	(0.04)	−0.03	(0.05)
Female	0.03	(0.02)	0.09***	(0.02)	0.03**	(0.02)	0.15***	(0.02)
Age	0.05***	(0.01)	0.00	(0.00)	−0.01**	(0.00)	0.00	(0.00)
Age sq.	−0.00***	(0.00)	−0.00	(0.00)	−0.00	(0.00)	−0.00***	(0.00)
Less than primary completed	−0.00	(0.05)	−0.03	(0.02)	−0.14***	(0.02)	−0.08***	(0.03)
Primary completed	0.09	(0.07)	−0.08***	(0.03)	−0.13***	(0.03)	−0.09**	(0.03)
Secondary not completed	0.19***	(0.05)	0.03	(0.02)	−0.46***	(0.02)	−0.43***	(0.03)
Secondary completed	0.21***	(0.06)	0.07**	(0.04)	−0.75***	(0.03)	−0.78***	(0.04)
Tertiary	0.00	(0.07)	−0.01	(0.04)	−1.26***	(0.04)	−1.22***	(0.04)
Employee	−0.33***	(0.09)	0.02	(0.05)	−0.00	(0.05)	0.08	(0.06)
Self-employed	−0.53***	(0.18)	0.06	(0.09)	−0.59***	(0.10)	−0.55***	(0.11)
Casual/help others	−0.08	(0.10)	−0.01	(0.05)	0.10*	(0.06)	0.34***	(0.07)
Union member	−0.22***	(0.08)	0.00	(0.04)	−0.14***	(0.04)	−0.20***	(0.03)
Permanent contract	−0.36***	(0.06)	−0.06*	(0.03)	−0.16***	(0.03)	−0.09***	(0.03)
Public share of employment	−0.19*	(0.11)	−0.08	(0.06)	−0.07	(0.06)	−0.33***	(0.06)
Formally employed	−0.00	(0.06)	−0.02	(0.04)	−0.19***	(0.04)	−0.13***	(0.04)
Coloured	−0.04	(0.05)	−0.08**	(0.04)	−0.41***	(0.04)	−0.25***	(0.03)
Asian/Indian	−0.21*	(0.12)	−0.02	(0.08)	−1.92***	(0.28)	−1.69***	(0.11)
White	−0.39***	(0.11)	−0.18***	(0.06)	−2.35***	(0.21)	−1.66***	(0.11)
No. employed in household	0.02	(0.01)	0.19***	(0.01)	−0.08***	(0.01)	−0.07***	(0.01)
No. children in household	0.00	(0.01)	−0.02***	(0.00)	0.20***	(0.00)	0.18***	(0.01)
Household receives grant	−0.07***	(0.03)	−0.00	(0.02)	−0.19***	(0.02)	−0.35***	(0.02)
Urban	−0.07***	(0.03)	−0.01	(0.02)	−0.19***	(0.02)	−0.27***	(0.02)
Farms	−0.16***	(0.05)	−0.07*	(0.04)	0.06	(0.04)	0.09**	(0.04)
Wave 2	0.14***	(0.03)	−0.19***	(0.02)	−0.08***	(0.02)	−0.08***	(0.03)
Wave 3	−0.05	(0.03)	−0.28***	(0.02)	−0.31***	(0.02)	−0.35***	(0.02)
Wave 4	−0.08***	(0.03)	−0.22***	(0.02)	−0.28***	(0.02)	−0.28***	(0.02)
Constant	−1.62***	(0.14)	−0.70***	(0.09)	0.48***	(0.08)	1.61***	(0.09)
Observations	56899		89938		89975		89975	
Pseudo R^2	0.11		0.02		0.29		0.35	

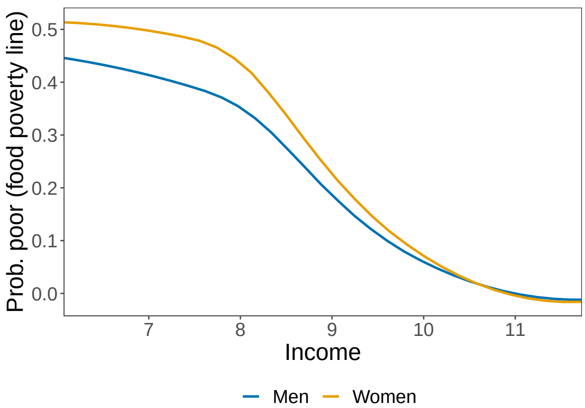
* p <0.1, ** p <0.05, *** p <0.01. Standard errors in parentheses.

Table C.1.2: Correlations between income and insecurity measures

	Income	Pr. unemployed	Pr. poor (F)	Pr. poor (UB)
Income	1.00			
Pr. unemployed	-0.49***	1.00		
Pr. poor (F)	-0.58***	0.43***	1.00	
Pr. poor (UB)	-0.68***	0.49***	0.91***	1.00



(a) Unemployment



(b) Poverty (food)

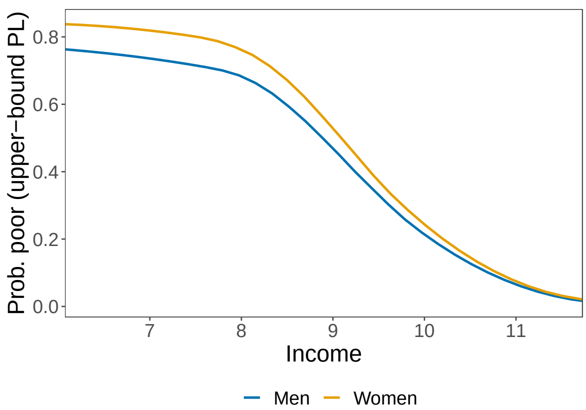


Figure C.1.1: Loess regressions of economic insecurity measures on income

C.2 Results for obesity

Table C.2.1: Probit regressions of obesity on insecurity measures with interactions: women

	(1)	(2)	(3)
Prob. unemployed	29.96***		
	(10.36)		
Prob. unemployed x Income	−7.02***		
	(2.47)		
Prob. unemployed x income sq.	0.41***		
	(0.15)		
Prob. poverty (upper-bound)		12.79***	
		(3.34)	
Prob. poverty (upper-bound) x Income		−3.01***	
		(0.75)	
Prob. poverty (upper-bound) x income sq.		0.18***	
		(0.04)	
Prob. poverty (food)			9.99**
			(4.25)
Prob. poverty (food) x Income			−2.44**
			(1.02)
Prob. poverty (food) x income sq.			0.15**
			(0.06)
Income	1.03**	1.80***	0.82**
	(0.44)	(0.57)	(0.39)
Income sq.	−0.05**	−0.10***	−0.04*
	(0.02)	(0.03)	(0.02)
Urban	0.14**	0.13**	0.12**
	(0.06)	(0.05)	(0.05)
Constant	−7.19***	−10.53***	−6.08***
	(1.98)	(2.61)	(1.75)
Observations	16307	16307	16307
Pseudo R^2	0.06	0.06	0.06

* p <0.1, ** p <0.05, *** p <0.01. Standard errors in parentheses.

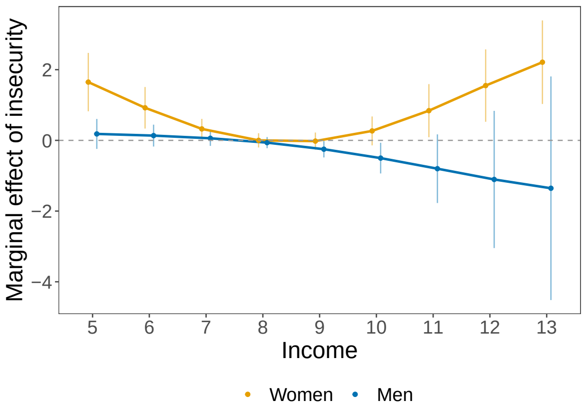
Note: Standard errors clustered by individual. Controls are age, race group, education, employed, married/cohabiting, province, smoker, exercises weekly, and ever given birth.

Table C.2.2: Probit regressions of obesity on insecurity measures with interactions: men

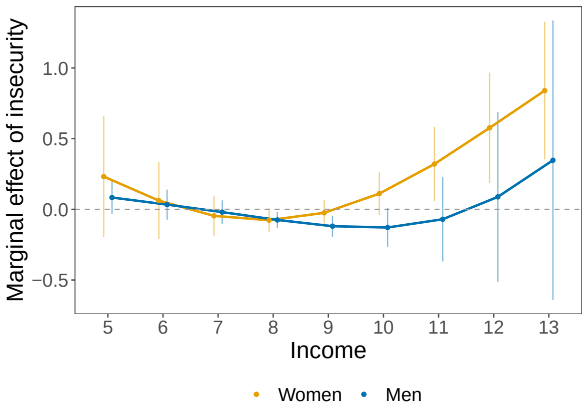
	(1)	(2)	(3)
Prob. unemployed	9.12		
	(21.29)		
Prob. unemployed x Income	−1.48		
	(4.86)		
Prob. unemployed x income sq.	0.04		
	(0.28)		
Prob. poverty (upper-bound)		3.05	
		(8.16)	
Prob. poverty (upper-bound) x Income		−0.81	
		(1.79)	
Prob. poverty (upper-bound) x income sq.		0.05	
		(0.10)	
Prob. poverty (food)			2.19
			(7.33)
Prob. poverty (food) x Income			−0.64
			(1.66)
Prob. poverty (food) x income sq.			0.04
			(0.09)
Income	0.20	0.01	−0.18
	(0.89)	(1.13)	(0.67)
Income sq.	0.01	0.01	0.02
	(0.05)	(0.06)	(0.04)
Urban	0.16*	0.13*	0.13
	(0.09)	(0.08)	(0.08)
Constant	−4.49	−3.32	−2.54
	(3.99)	(5.23)	(3.07)
Observations	10294	10294	10294
Pseudo R^2	0.14	0.14	0.14

* p <0.1, ** p <0.05, *** p <0.01. Standard errors in parentheses.

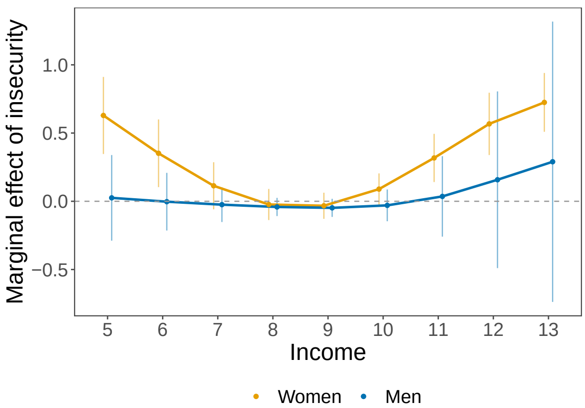
Note: Standard errors clustered by individual. Controls are age, race group, education, employed, married/cohabiting, province, smoker, and exercises weekly.



(a) Unemployment



Poverty (food)



(c) Poverty (upper-bound)

Figure C.2.1: Marginal effects of economic insecurity measures on obesity by income level

C.3 Results for blood pressure

Table C.3.1: Regressions of systolic blood pressure on insecurity measures

	Men			Women		
	(1)	(2)	(3)	(4)	(5)	(6)
Prob. unemployed	−26.86			−69.59***		
	(29.46)			(19.04)		
Prob. unemployed x Income	1.39			8.31***		
	(3.59)			(2.34)		
Prob. poverty (upper-bound)		−4.92			−36.85***	
		(9.35)			(10.27)	
Prob. poverty (upper-bound) x Income		−0.30			4.02***	
		(1.05)			(1.13)	
Prob. poverty (food)			10.25			−39.20***
			(11.85)			(9.74)
Prob. poverty (food) x Income			−2.08			4.50***
			(1.41)			(1.17)
Income	0.13	0.21	0.71	−1.99***	−3.10***	−1.97***
	(0.59)	(0.64)	(0.50)	(0.53)	(0.82)	(0.53)
Observations	10458	10458	10458	17327	17327	17327
Adjusted R^2	0.12	0.12	0.12	0.23	0.23	0.23

* p <0.1, ** p <0.05, *** p <0.01. Standard errors in parentheses.

Note: Full set of covariates (including an indicator for being on blood pressure medication) included but not displayed.

Table C.3.2: Regressions of diastolic blood pressure on insecurity measures

	Men			Women		
	(1)	(2)	(3)	(4)	(5)	(6)
Prob. unemployed	3.34			−26.11*		
	(17.89)			(14.68)		
Prob. unemployed x Income	−1.13			3.09*		
	(2.14)			(1.81)		
Prob. poverty (upper-bound)		2.41			−13.35**	
		(6.29)			(6.55)	
Prob. poverty (upper-bound) x Income		−0.69			1.37*	
		(0.71)			(0.73)	
Prob. poverty (food)			10.93			−11.82*
			(7.98)			(6.58)
Prob. poverty (food) x Income			−1.64*			1.18
			(0.95)			(0.78)
Income	0.51	0.54	0.69**	−0.87**	−1.26**	−0.80**
	(0.34)	(0.43)	(0.33)	(0.35)	(0.53)	(0.36)
Observations	10458	10458	10458	17327	17327	17327
Adjusted R^2	0.09	0.09	0.09	0.13	0.13	0.13

* p <0.1, ** p <0.05, *** p <0.01. Standard errors in parentheses.

Note: Full set of covariates (including an indicator for being on blood pressure medication) included but not displayed.

Table C.3.3: Regressions of high blood pressure on insecurity measures

	Men			Women		
	(1)	(2)	(3)	(4)	(5)	(6)
Prob. unemployed	−0.02			−1.12**		
	(0.79)			(0.50)		
Prob. unemployed x Income	−0.03			0.13**		
	(0.09)			(0.06)		
Prob. poverty (upper-bound)		0.10			−0.58**	
		(0.26)			(0.27)	
Prob. poverty (upper-bound) x Income		−0.03			0.07**	
		(0.03)			(0.03)	
Prob. poverty (food)			0.34			−0.64***
			(0.31)			(0.24)
Prob. poverty (food) x Income			−0.05			0.07**
			(0.04)			(0.03)
Income	0.01	0.02	0.02	−0.04***	−0.05**	−0.04***
	(0.02)	(0.02)	(0.01)	(0.01)	(0.02)	(0.01)
Observations	10458	10458	10458	17327	17327	17327
Adjusted R^2	0.07	0.07	0.07	0.12	0.12	0.12

* p <0.1, ** p <0.05, *** p <0.01. Standard errors in parentheses.

Note: Full set of covariates included but not displayed. High blood pressure defined as having a high blood pressure reading or being on blood pressure medication.

C.4 Association between economic insecurity and BMI without interactions

The results of regressions of BMI on the insecurity variables without interactions with income are shown in Tables C.4.1 and C.4.2. For women, none of the insecurity measures are significantly associated with BMI. For men, higher economic insecurity is associated with lower BMI. The negative association between insecurity and body fat for men contrasts with the pattern observed in the developed country literature, but is consistent with the positive association between body fat and income observed in South Africa, if income and economic *security* are seen as alternative measures of economic well-being. Income is negatively associated with body weight in developed countries and positively associated in developing countries; economic insecurity is generally positively associated with body weight in developed countries, but here we see a negative association.

Table C.4.1: Regressions of BMI on insecurity measures without income interaction: women

	(1)	(2)	(3)
Prob. unemployed	0.16		
	(1.42)		
Prob. poverty (upper-bound)		−0.44	
		(0.78)	
Prob. poverty (food)			−0.68
			(0.69)
Income	2.14*	2.13*	2.07*
	(1.25)	(1.29)	(1.15)
Income sq.	−0.09	−0.09	−0.09
	(0.08)	(0.08)	(0.07)
Urban	0.96***	0.90***	0.87***
	(0.29)	(0.30)	(0.30)
Constant	4.50	5.17	5.41
	(5.84)	(5.72)	(5.34)
Observations	16307	16307	16307
Adjusted R^2	0.09	0.09	0.09

* p <0.1, ** p <0.05, *** p <0.01. Standard errors in parentheses.

Note: Standard errors clustered by individual. Controls are age, race group, education, employed, married/cohabiting, province, smoker, exercises weekly, and ever given birth.

Table C.4.2: Regressions of BMI on insecurity measures without income interaction: men

	(1)	(2)	(3)
Prob. unemployed	−1.76		
	(1.38)		
Prob. poverty (upper-bound)		−1.11**	
		(0.45)	
Prob. poverty (food)			−0.84
			(0.54)
Income	−1.81*	−1.74**	−1.76***
	(0.97)	(0.75)	(0.66)
Income sq.	0.16***	0.16***	0.16***
	(0.06)	(0.05)	(0.04)
Urban	0.36	0.20	0.28
	(0.22)	(0.24)	(0.23)
Constant	24.41***	25.00***	24.37***
	(4.24)	(3.29)	(2.96)
Observations	10294	10294	10294
Adjusted R^2	0.24	0.25	0.24

* p <0.1, ** p <0.05, *** p <0.01. Standard errors in parentheses.

Note: Standard errors clustered by individual. Controls are age, race group, education, employed, married/cohabiting, province, smoker, and exercises weekly.

Table C.4.3: Variance inflation factors (VIFs) of insecurity measures from regressions of BMI on insecurity

	Women		Men	
	Coefficient	VIF	Coefficient	VIF
Prob. unemployment	0.82	2.17	-3.03***	1.99
Prob. poverty (food)	-0.65	2.59	-1.06**	2.30
Prob. poverty (upper-bound)	-0.73	3.72	-1.57***	3.10

C.5 Robustness checks

C.5.1 Negative events and job loss

Table C.5.1: Regressions of BMI on negative events and job loss, women

	(1)	(2)	(3)	(4)	(5)
Death of financial supporter	−10.20***				
	(3.92)				
Death of financial supporter × Income	1.31***				
	(0.50)				
Death of household member		−6.11***			
		(2.05)			
Death of household member × Income		0.76***			
		(0.25)			
Negative agricultural event			−5.09*		
			(2.71)		
Negative agricultural event × Income			0.58*		
			(0.32)		
Became unemployed (broad)				−7.14**	
				(3.44)	
Became unemployed (broad) × Income				0.90**	
				(0.43)	
Became unemployed (strict)					−8.41**
					(3.57)
Became unemployed (strict) × Income					1.04**
					(0.44)
Income	0.32**	0.48***	0.51***	0.54***	0.54***
	(0.13)	(0.13)	(0.12)	(0.15)	(0.15)
Constant	12.63***	12.31***	12.10***	12.08***	12.11***
	(2.10)	(1.73)	(1.73)	(2.04)	(2.04)
Observations	15997	31517	31456	20627	20627
Adjusted R^2	0.09	0.08	0.08	0.09	0.09

* p <0.1, ** p <0.05, *** p <0.01. Standard errors in parentheses.

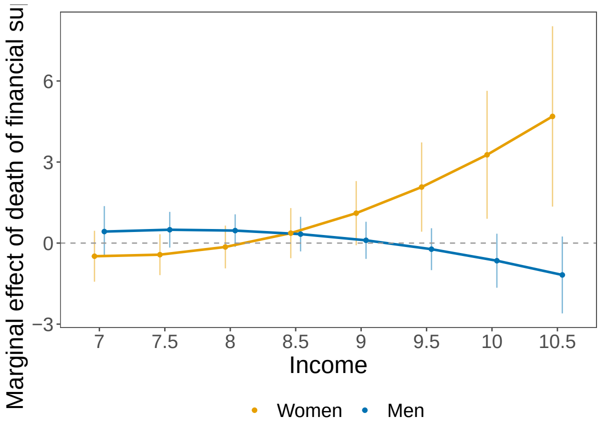
Note: Full set of covariates included but not displayed. Standard errors clustered by individual. A financial supporter is a non-resident friend or family member who provided financial assistance. A negative agricultural event is defined as a major crop failure or widespread disease or death of livestock. 'Became unemployed' is defined as being employed in the previous wave and unemployed in the current wave.

Table C.5.2: Regressions of BMI on negative events and job loss, men

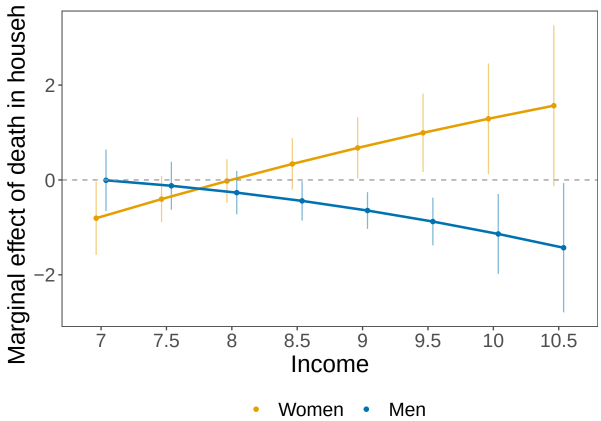
	(1)	(2)	(3)	(4)	(5)
Death of financial supporter	3.78*				
	(2.21)				
Death of financial supporter × Income	−0.43*				
	(0.26)				
Death of household member		3.19			
		(1.95)			
Death of household member × Income		−0.43*			
		(0.23)			
Negative agricultural event			−2.29		
			(2.90)		
Negative agricultural event × Income			0.30		
			(0.36)		
Became unemployed (broad)				4.25*	
				(2.49)	
Became unemployed (broad) × Income				−0.49	
				(0.30)	
Became unemployed (strict)					4.29*
					(2.53)
Became unemployed (strict) × Income					−0.52*
					(0.31)
Income	0.83***	1.02***	0.99***	1.20***	1.19***
	(0.10)	(0.09)	(0.09)	(0.12)	(0.12)
Constant	10.56***	12.33***	12.46***	11.43***	11.50***
	(1.76)	(1.40)	(1.41)	(1.76)	(1.76)
Observations	10239	20991	20948	12994	12994
Adjusted R^2	0.22	0.22	0.22	0.24	0.24

* p <0.1, ** p <0.05, *** p <0.01. Standard errors in parentheses.

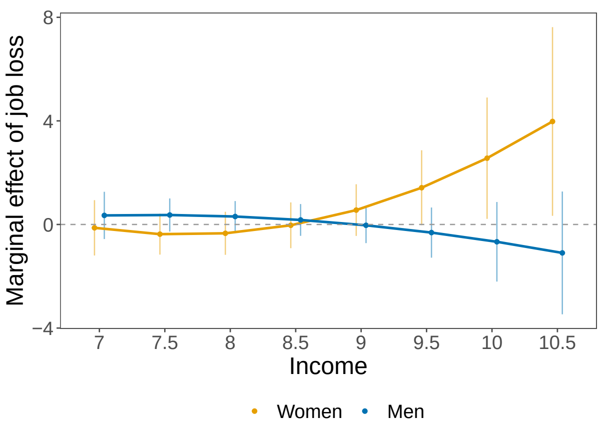
Note: Full set of covariates included but not displayed. Standard errors clustered by individual. A financial supporter is a non-resident friend or family member who provided financial assistance. A negative agricultural event is defined as a major crop failure or widespread disease or death of livestock. 'Became unemployed' is defined as being employed in the previous wave and unemployed in the current wave.



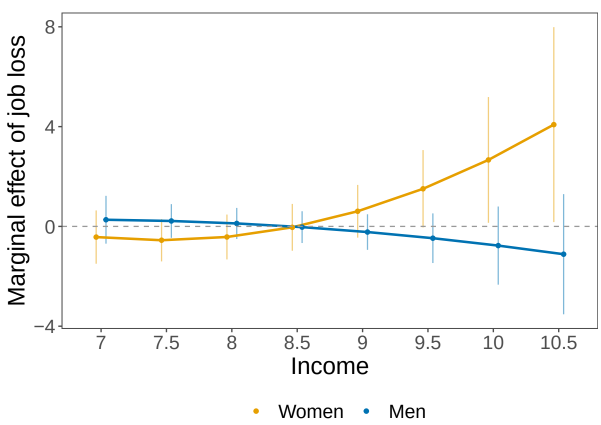
(a) Death of financial supporter



(b) Death in household



(c) Became unemployed (broad)



(d) Became unemployed (narrow)

Figure C.5.1: Marginal effects of negative events and job loss on BMI
Source: Own calculations, NIDS.

C.5.2 Narrow definition of unemployment and prob. unemployed for those currently employed

Table C.5.3: Alternative specifications of probability of future unemployment

	Men			Women		
	(1)	(2)	(3)	(4)	(5)	(6)
Prob. unemployed	93.67			186.67***		
(narrow)	(65.22)			(47.43)		
Prob. unemployed	−16.46			−43.70***		
(narrow) × Income	(15.36)			(11.30)		
Prob. unemployed	0.60			2.53***		
(narrow) x inc. sq.	(0.90)			(0.67)		
Prob. becoming		260.07**			264.24	
unempl. (broad)		(121.11)			(166.42)	
Prob. becoming		−56.89**			−59.40	
unempl. (broad) × Income		(27.45)			(38.51)	
Prob. becoming		2.99*			3.32	
unempl. (broad) x inc. sq.		(1.55)			(2.21)	
Prob. becoming			258.91**			418.86**
unempl. (narrow)			(129.27)			(182.60)
Prob. becoming			−55.20*			−95.01**
unempl. (narrow) × Income			(29.34)			(42.34)
Prob. becoming			2.80*			5.30**
unempl. (narrow) x inc. sq.			(1.66)			(2.46)
Income	1.69	3.16	3.14	5.82***	6.89*	7.15**
	(2.13)	(3.01)	(2.93)	(1.98)	(3.75)	(3.41)
Income sq.	−0.00	−0.08	−0.08	−0.29**	−0.33	−0.35*
	(0.12)	(0.16)	(0.16)	(0.12)	(0.21)	(0.19)
Observations	10294	6158	6158	16307	6270	6270
Adjusted R^2	0.25	0.26	0.26	0.09	0.09	0.09

* p <0.1, ** p <0.05, *** p <0.01. Standard errors in parentheses.

Note: Full set of covariates included but not displayed. Standard errors clustered by individual. Prob. unemployed (narrow) is the predicted probability of being unemployed (narrow definition) in the next wave, regardless of current employment status. Prob. becoming unemployed (broad) is the predicted probability of becoming unemployed (broad definition) for those currently employed. Prob. becoming unemployed (narrow) is the predicted probability of becoming unemployed (narrow definition) for those currently employed.

C.5.3 Probability of unemployment for those under 60

Table C.5.4: Regressions of BMI on insecurity measures with interactions, under-60s

	Men			Women		
	(1)	(2)	(3)	(4)	(5)	(6)
Prob. unemployed	40.71			183.27***		
	(49.07)			(52.63)		
Prob. unemployed x Income	−5.60			−42.24***		
	(11.57)			(12.49)		
Prob. unemployed x income sq.	0.06			2.41***		
	(0.68)			(0.74)		
Prob. poverty (upper-bound)		16.18			62.42***	
		(17.54)			(21.32)	
Prob. poverty (upper-bound) x Income		−3.57			−14.80***	
		(4.12)			(4.72)	
Prob. poverty (upper-bound) x income sq.		0.18			0.87***	
		(0.24)			(0.26)	
Prob. poverty (food)			6.56			49.72**
			(18.55)			(24.29)
Prob. poverty (food) x Income			−1.04			−12.30**
			(4.42)			(5.76)
Prob. poverty (food) x income sq.			0.02			0.75**
			(0.26)			(0.34)
Income	0.69	0.28	−1.15	7.38***	9.77***	4.86**
	(2.16)	(2.14)	(1.72)	(2.26)	(3.47)	(2.10)
Income sq.	0.05	0.05	0.13	−0.38***	−0.52***	−0.25**
	(0.12)	(0.12)	(0.10)	(0.13)	(0.19)	(0.12)
Urban	0.30	0.20	0.24	0.90***	0.86***	0.79**
	(0.22)	(0.25)	(0.24)	(0.29)	(0.33)	(0.31)
Constant	13.28	16.50*	22.68***	−17.64*	−27.70*	−5.40
	(9.69)	(9.81)	(7.81)	(10.21)	(15.96)	(9.64)
Observations	9556	9556	9556	14891	14891	14891
Adjusted R^2	0.25	0.24	0.24	0.10	0.10	0.10

* p <0.1, ** p <0.05, *** p <0.01. Standard errors in parentheses.

Note: Full set of covariates included but not displayed. Standard errors clustered by individual.

C.6 Interactions between economic insecurity and urban residence

Tables C.6.1 and C.6.2 show the results of regressions interacting the economic insecurity measures with an indicator for residing in an urban area, and Figure C.6.1 plots the marginal effects of each measure by urban or rural residence. There is a significant positive interaction between urban residence and the poverty measure for women, indicating a stronger association between insecurity and BMI in urban areas. However, Figure C.6.1 shows that the marginal effects of the insecurity measures are not significantly different from zero in urban areas. There is a significant negative interaction between urban residence and the unemployment measure for men, indicating a weaker association in urban areas. All other interactions are insignificant.

Table C.6.1: Regressions of BMI on insecurity measures with urban interaction: women

	(1)	(2)	(3)
Prob. unemployed	−0.35		
	(1.49)		
Urban x Prob. unemployed	0.98		
	(1.91)		
Prob. poverty (upper-bound)		−1.72*	
		(0.90)	
Urban x Prob. poverty (upper-bound)		2.06**	
		(1.02)	
Prob. poverty (food)			−1.25*
			(0.71)
Urban x Prob. poverty (food)			1.67
			(1.23)
Income	2.10*	1.90	1.98*
	(1.18)	(1.27)	(1.14)
Income sq.	−0.09	−0.08	−0.08
	(0.07)	(0.08)	(0.07)
Urban	0.81*	−0.48	0.31
	(0.44)	(0.77)	(0.50)
Constant	4.66	7.08	5.94
	(5.43)	(5.86)	(5.29)
Observations	16307	16307	16307
Adjusted R^2	0.09	0.09	0.09

* p <0.1, ** p <0.05, *** p <0.01. Standard errors in parentheses.

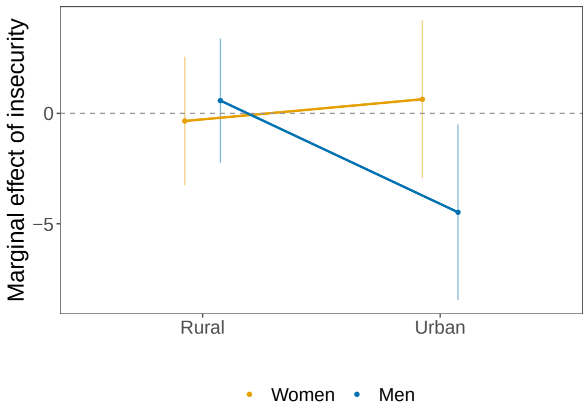
Note: Standard errors clustered by individual. Controls are age, race group, education, employed, married/cohabiting, province, smoker, exercises weekly, and ever given birth.

Table C.6.2: Regressions of BMI on insecurity measures with urban interaction: men

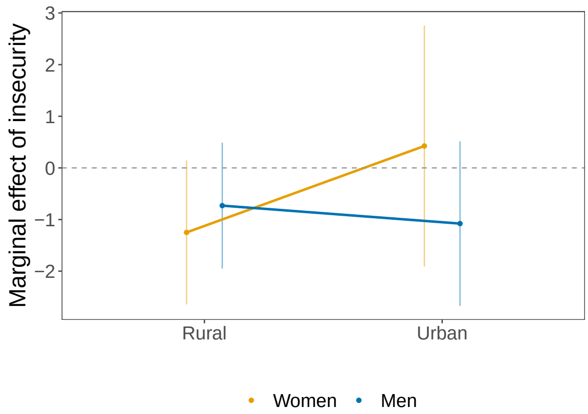
	(1)	(2)	(3)
Prob. unemployed	0.58		
	(1.43)		
Urban x Prob. unemployed	−5.05**		
	(2.01)		
Prob. poverty (upper-bound)		−1.03	
		(0.72)	
Urban x Prob. poverty (upper-bound)		−0.13	
		(0.78)	
Prob. poverty (food)			−0.73
			(0.62)
Urban x Prob. poverty (food)			−0.35
			(0.92)
Income	−1.72*	−1.73*	−1.75***
	(1.00)	(0.95)	(0.66)
Income sq.	0.16***	0.16***	0.16***
	(0.06)	(0.06)	(0.04)
Urban	0.99***	0.27	0.35
	(0.36)	(0.50)	(0.32)
Constant	24.05***	24.92***	24.32***
	(4.41)	(3.98)	(2.96)
Observations	10294	10294	10294
Adjusted R^2	0.25	0.25	0.24

* p <0.1, ** p <0.05, *** p <0.01. Standard errors in parentheses.

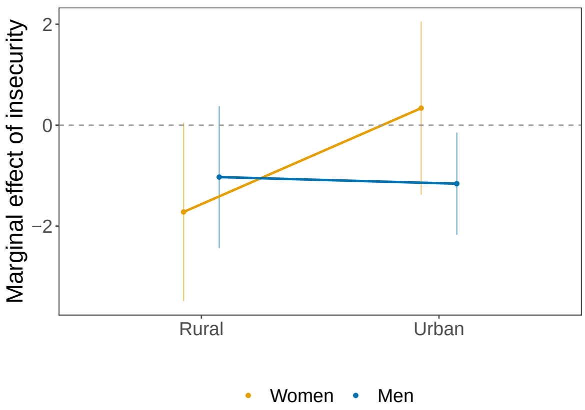
Note: Standard errors clustered by individual. Controls are age, race group, education, employed, married/cohabiting, province, smoker, and exercises weekly.



(a) Unemployment



(b) Poverty (food)



(c) Poverty (upper-bound)

Figure C.6.1: Marginal effects of economic insecurity measures on BMI by urban residence