

A GROWING RISK: CLINICAL, EPIDEMIOLOGIC, AND SUBJECTIVE AMBIGUITY IN THE  
RELATIONSHIP BETWEEN WEIGHT AND HEALTH

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## **ABSTRACT**

Iliya Gutin: A Growing Risk: Clinical, Epidemiologic, and Subjective Ambiguity in the Relationship between Weight and Health  
(Under the direction of Robert A. Hummer)

This dissertation examines the complex and often uncertain relationship between body weight and health in a highly weight-conscious society like the United States, using a mixed methods approach to study three key domains in which this ambiguity is evident. The first chapter draws on interviews with clinicians to examine the tension between medical definitions of healthy weight used by practitioners, the metrics of success they seek to promote among patients, and the broader messaging about weight and health in the culture at-large. Notably, practitioners often avoid “diagnosing” childhood obesity and poor health in favor of emphasizing a more optimistic “prognosis” emphasizing children’s and families’ success in developing healthy beliefs and behaviors that engender long-term success. The second chapter questions the assumption of homogeneously poor health among adults with obesity by examining the clustering of body size and other measures of health in a large nationally-representative data set. Medical research often frames “healthy” and “unhealthy” obesity as a function of random biological differences in the population; conversely, my work shows that these phenotypes are socially-patterned on the basis of individuals’ socioeconomic status, helping to explain group differences in mortality. Finally, the third chapter examines the consequences of individuals’ perceptions of their weight over the life course. Social and cultural stereotypes about individuals on the basis of their weight suggest that negative perceptions of one’s weight can be psychosocially damaging, leading to many of the harmful outcomes that we associate with body weight. This study demonstrates that objective and subjective weight status influence each other over time, such that both impact health in adulthood. Critically, these analyses underscore the consequences of weight-related stigma as source of poor health that is attributable to social norms about

what constitutes a “healthy” and “normal” body. In sum, this dissertation advances a more comprehensive approach to the study of and messaging about body weight and health, inclusive of a broader and more nuanced set of physiological and psychosocial explanations.

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## LIST OF ABBREVIATIONS

Add Health	National Longitudinal Study of Adolescent to Adult Health
AIC	Akaike information criterion
ALT-FE	autoregressive latent trajectory-fixed effects
AME	average marginal effects
AR	autoregressive
BIC	Bayesian information criterion
BMI	body mass index
CDC	Centers for Disease Control
CFA	confirmatory factor analysis
CFI	comparative fit index
CRP	C-reactive protein
CVD	cardiovascular disease
DBP	diastolic blood pressure
EBM	“evidence-based movement”
FCT	fundamental cause theory
FIML	Casewise Maximum Likelihood
HAES	Health At Every Size movement
KHB	Karlson, Holm, and Breen
LCA	Latent Class Analysis
LPA	Latent Profile Analyses
MHO	metabolically-healthy obesity
NASEM	National Academies of Sciences, Engineering, and Medicine
NCHS	National Center for Health Statistics
NH	non-Hispanic
NHANES	National Health and Nutrition Examination Survey

NHLBI	National Heart, Lung, and Blood Institute
NLSY97	National Longitudinal Survey of Youth 1997
OBJ	Objective Weight
OR	odds ratio
RICLPM	random intercept cross-lagged panel models
RMSEA	root mean square error of approximation
SBP	systolic blood pressure
SD	standard deviation
SEM	structural equation modeling
SES	socioeconomic status
SUBJ	Subjective Weight
TLI	Tucker-Lewis index
WI-V	waves I through V
WLSMV	weighted least square mean and variance adjusted

## CHAPTER 1: INTRODUCTION

Sociologists representing a broad range of sub-disciplinary areas and interests have come to recognize obesity, and weight-related health more broadly, as a growing risk in U.S. society; however, the nature of this risk remains highly contested. Many argue that the *biophysiological* toll of obesity as state of impaired health is considerable, and likely to increase in coming decades as a greater proportion of adults spend a larger share of their lives exceeding recommended thresholds for a “healthy” weight (National Academies of Science, Engineering, and Medicine 2021). There is evidence to suggest that obesity’s impact on population health is already implicated in stagnating and/or declining life expectancy in the United States (Masters et al. 2018; Olshansky et al. 2005; Preston et al. 2014; Preston et al. 2018). Yet others take the position that the *psychological* and *social* toll of obesity, as a source of inequality and stigma, has inflicted the most harm on millions of adults whose individual experiences of their bodies, weight, and health are increasingly circumscribed by clinical and epidemiologic standards for disease and unhealthiness (Greenhalgh 2015; Saguy 2012; Shugart 2016). Thus, rather than adhering to these unambiguous standards for normal or ideal bodies, there is a push to recognize individuals’ health as a multidimensional and holistic construct, with an emphasis on understanding heterogeneity in body weight as a *contributing* rather than *deterministic* component.

Indeed, researchers primarily care about body weight and size inasmuch as it purports to convey information about other aspects of health, like the types of behaviors a person engages in or their level of physiological impairment, to the extent that having a scale or measurement like the body mass index (BMI:  $\text{weight}[\text{kg}]/\text{height}[\text{m}]^2$ ) facilitates comparisons and rankings (Bowker and Star 1999; Fourcade 2016; Jutel 2006). These behavioral and disease frameworks are inherently attractive as they convey information about what a person is doing or how they are, at present, allowing researchers to make more



definitive pronouncements about individuals' healthiness based on their BMI. However, there is increasing recognition of an alternative perspective that bridges clinical and epidemiologic research on the limitations of BMI as a health surrogate with a sociological and psychological understanding of body size as an axis of inequality. This 'weight neutral' framework does not downplay the importance of studying body size and health; rather, it downplays the need to directly and unambiguously equate body size with health in research, the practice of medicine, and the conceptualization of overweight and obesity. Body size is acknowledged as a neutral form of human variation (Saguy 2012), whereby BMI reflects both biophysiological and psychosocial mechanisms of risk.

This framing is integral to maintaining a sociological perspective on the role of body size and weight in individuals' lives and society as a whole. Body size has been problematized and stigmatized as an abnormal form of human variation just as other forms of human variation have been considered 'undesirable.' In a highly weight-conscious society where individuals' social worth is tied to their appearance (Gutin 2021; Jutel and Buetow 2007; Shugart 2016), body size represents another form of stratification that influences health. Stigma is a fundamental mechanism underlying health disparities (Hatzenbuehler et al. 2013), and body weight is historically one of the first forms of stigma examined by sociologists (Cahnman 1968; Maddox et al. 1968). Yet, more than a half-century later, it continues to be a "socially acceptable form of bias" due in no small part to the presumption that individuals with medically "unacceptable" bodies are a social, economic, and health burden (Puhl and Heuer 2010: 1019). To the extent that body weight and size provide some indication of individuals' physical appearance, they have significance as markers of social abnormality and inequality which are independently associated with health by way of individuals' social interactions and experiences. Consequently, there is considerable heterogeneity in the mechanisms and explanations underlying how and why body weight and health are associated with one another.

Critically, this uncertainty in our understanding of the relationship between weight and health is more than just a function of disciplinary differences in methodology and theoretical grounding, or even individual choices in the definition or measurement of disease and health. More fundamentally, it is a

reflection of the difficulties in assessing the risk that obesity poses in a medically and socially dynamic world, wherein the relationship between one's weight and health is simultaneously constructed at a clinical, epidemiological, *and* subjective level. Indeed, the co-construction of health and illness across multiple social domains is a foundational principle within medical sociology; decades of research have examined the social practices underlying the creation of medical knowledge and practice (Conrad and Barker 2010; Foucault 1963; Timmermans and Berg 2003), the definition and diagnosis of disease and illness (Brown 1995; Jutel 2009; Jutel 2014; Rosenberg 2002), and the significance of individuals' experiences of their health and wellbeing (Brown 1995; Bury 1991; Bury 2013; Hydén 1997; Kleinman 1988; Parsons 1975; Rosenberg 2002). Yet this comprehensive approach to documenting multiple stakeholders and perspectives in the study of health and illness writ-large is rarely applied to the study of *specific* diseases and health conditions (Timmermans and Haas 2008). This is an unfortunate limitation of much extant medical sociology, as it “grant[s] health professionals, many health researchers and, increasingly, epidemiologists the clinical facts, leaving [social scientists] no choice either to accept clinical parameters at face value, tirelessly denounce the ‘construction’ of factual knowledges, or, more often, to ignore such factors” in conducting research and making claims about its implications (Timmermans and Haas 2008: 662).

However, the broad repertoire of analytic techniques, data sources, and theoretical frameworks employed by sociologists can and *should* be leveraged towards greater specificity in our research on health – especially in the study of body weight. Sociologists' contributions to the understanding of body weight and size – and the broader concept of obesity that they help define – as “clinical facts” is contingent upon recognizing what is unique, rather than exclusively generalizable, about their relationship with health. Yet, capturing the uniqueness of how body weight is associated with health requires knowledge of the many contexts in which body weight is defined as a health risk, such as the perspective of clinicians and health practitioners studying obesity and working with patients, the results of epidemiological analyses of population health data, and individuals' subjective experiences and perceptions of their weight. Consequently, the overarching goal of this dissertation is to examine these

different disciplinary *inputs* in the framing of body size as a health risk and, in doing so, help resolve some of the ambiguity and uncertainty in the study of obesity and weight-related health.

In brief, the first project examines obesity within a medical/clinical research setting, focusing on the challenges of conceptualization and communication in childhood obesity as a diagnosis in clinical encounters. Clinicians are tasked with conveying the *potential* for future harm to patients and families, rather than pointing to clear signs that something is *already* wrong. This is especially difficult given the emphasis on certainty and unambiguity in medical care; doctors are looked to as arbiters of healthiness, but healthiness takes on many meanings among children and families. However, recent sociological research has noted the importance of *prognostication* in diagnosis and how thinking more critically about patients' future, rather than present, circumstances necessitates a kind of *social diagnosis* informed by multiple stakeholders and knowledge of the many social, rather than medical, aspects of their day-to-day lives. This emerging diagnostic model is especially key for clinicians' success in creating a partnership with patients and families, understanding their psychosocial milieu, and identifying an individualized model of success rather than continuing to promote universal criteria for defining healthiness. To better understand the challenges and nuances of body weight and health in early life, this project draws on semi-structured interviews with health professionals to examine their strategies for communicating risk and defining success in the diagnosis and treatment of childhood obesity.

The second project examines obesity as an epidemiological measure, documenting the co-occurrence of obesity (on the basis of body weight and size) with key indicators of cardiometabolic health risk within the U.S. adult population over the past three decades. Many critiques of obesity as a “disease” take issue with the implicit assumption of homogeneously poor health among adults exceeding clinical thresholds for obesity, as both the definition and measurement of obesity is not necessarily a reflection of individuals' underlying physiological state. Yet, biophysiological variation underlying obesity as an “unhealthy” condition or a disease can be empirically analyzed by examining its co-occurrence with other cardiometabolic health indicators within the population, and in their association with mortality risk. Indeed, many studies have identified a “Metabolically Healthy Obesity” phenotype, which represents a

substantial proportion of adults with obesity, and the population as a whole. However, past research has primarily described these obesity phenotypes as biologically or genetically patterned, ignoring key social factors – like educational attainment – that shape individuals’ risk profiles. This study uses data from the National Health and Nutrition Examination Survey (1988-2014) linked with follow-up mortality data to identify which set of body sizes and cardiometabolic health profiles best characterize the U.S. adult population over the past decades, how they are associated with premature mortality risk, and how the social patterning of these profiles explains overall educational gradients in mortality risk.

Finally, the third project examines body weight as a subjective, individual-level construct, analyzing the relationship between individuals’ “objective” body weight and their perceptions of their weight, and how the two interact with one another in their associations with both physical and mental health outcomes. Individuals’ subjective experiences and assessments of their health are commonly-used variables in social and health research, with demonstrated utility over and above objective or clinical measures. Yet, subjective experiences or (mis)perceptions of weight are primarily framed as an obstacle to improving population health, as they may lead individuals to inaccurately assess their weight status and engage in unhealthy behaviors. This perspective ignores a large body of research on the deeply stigmatizing aspects of *being* overweight as a unique driving force underlying poor mental and physical health among many children, adolescents and adults. The extent to which adults negatively perceive themselves and their bodies in relation to their weight (i.e., subjective weight) – in a society where so many are devalued and derogated on the basis of their physical appearance – can be psychologically damaging and stressful, leading not only to poor mental health, but also to many of the harmful physical risk factors typically associated with objective weight. Using five waves of the National Longitudinal Study of Adult to Adolescent Health (1994-2019), this study uses structural equation modeling to analyze the cumulative effect of both objective and subjective weight on health, while allowing for cross-lagged and direct associations between the two. By examining a mix of both physical and mental health outcomes, this study is the first to compare the relative impact of objective and subjective weight on health throughout the early portion of life course.

Though the three projects answer different research questions, focus on different bodies of literature, and use different sources of data and methodological approaches, they are united by a shared objective of improving the conceptualization, definition, and measurement of body size and obesity in sociological, demographic, epidemiologic, and medical research, as well as in informing research priorities and interventions for improving population health. Taken together, this dissertation seeks to emphasize the value of adequately documenting the different ways that individuals' weight interacts with their health as a function of it being both a physical trait and social identity. Moreover, both perspectives on weight inform the actions taken by institutions and individuals to improve the health and quality of life among the many individuals for whom body weight is a defining aspect of their day-to-day health and social experiences.

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## **CHAPTER 2: NOT ‘PUTTING A NAME TO IT’: INTEGRATING UNCERTAINTY INTO THE DIAGNOSIS AND TREATMENT OF CHILDHOOD OBESITY**

### **Introduction**

Childhood obesity is consistently singled out as a key public health challenge facing the United States. Recent data show that approximately one-in-five youth ages 2-19 have a body mass index (BMI:  $\text{height[m]}/\text{weight[kg]}^2$ ) that is considered “obese” (Fryar et al. 2020). Often described as a “crisis” or a “threat,” the sense of urgency is understandable given the emergence of obesity as a major contributor to morbidity and mortality (RWJF 2021; WHTFCO 2010), especially as U.S. adults spend an increasing proportion of their lives in an ‘unhealthy’ body weight (Lee 2010; NASEM 2021). Indeed, while childhood obesity is associated with worse health in early life (CDC 2021), the greater concern is over its future implications because youth with obesity are more likely to become adults with obesity (CDC 2021; Gordon-Larsen et al. 2010; Ward et al. 2017). Given the social and economic costs stemming from poor health and limitations linked to obesity throughout the life course, the Obama administration explicitly made “solving the problem of childhood obesity within a generation” a key public health priority (WHTFCO 2010). Unfortunately, this ambitious goal has not been achieved and recent evidence suggests that any leveling off or decrease in childhood obesity observed over the past decade was illusory (Skinner et al. 2018).

The discrepancy between our ever-increasing knowledge of the etiology and consequences of childhood obesity, and our continued inability to address it in any meaningful way, is a source of frustration for pediatricians and many other health professionals (Carroll 2020). Diagnosing, discussing, and treating weight-related health in a clinical setting represents a *distinct* set of challenges compared to population-level initiatives. In theory, there is considerable certainty on how to address obesity – as suggested by the results of numerous studies and interventions that lead to weight loss. Yet in practice



there is considerable uncertainty in understanding how this knowledge can be translated into actual improvements for individuals (Carroll 2020). Due to this uncertainty, the health professionals involved in the development and deployment of clinical protocols have been castigated for using obesity as a medical diagnosis if “the field of medicine has no safe, reliable means to enable [children and teens] to lose the weight and keep it off, and so become ‘well’ and ‘normal’” (Greenhalgh 2015: 281-282).

Though controversial, diagnoses like “childhood obesity” are a critical organizational tool in medicine, imposing certainty by categorizing individuals as healthy or unhealthy (Jutel 2009). Yet weight and health are hard to definitively characterize in children. Direct evidence of poor health is rarely observed, and in some cases unobservable, beyond the tautological observation that the BMI itself is “unhealthy” (Gutin 2018; Sharma and Campbell-Scherer 2017). Youth with obesity are at higher risk for developing cardiometabolic comorbidities like hypertension, dyslipidemia, and hyperglycemia (Skinner et al. 2015), but the lower absolute prevalence of these conditions in early life suggests that most pediatric patients with obesity do not exhibit clear signs of poor health. In the highly time-constrained context of a clinical encounter – where a typical visit is less than 20 minutes (Halfon et al. 2011) – practitioners may not be able to identify an observable issue *caused* by weight. Moreover, childhood is an incredibly dynamic period in life; weight naturally fluctuates as children grow and the same BMI has different interpretations at different ages (CDC 2020). Visual assessments of a child’s weight may not comport with clinical definitions of “healthiness,” introducing an added layer of complexity and uncertainty for both practitioners and patients (CDC 2014).

Finally, diagnosis is further complicated by increased recognition that obesity has social meanings and consequences that transcend the confines of a clinical encounter. In theory, obesity is an objective clinical diagnosis (Jutel 2019); in practice, it is far from neutral with respect to the social stigmas surrounding the term (Murray 2009; Saguy 2012; Shugart 2016). Medicine is a powerful and authoritative social institution, and many contend that the medicalization of childhood obesity in contemporary society perpetuates these harmful social beliefs about what constitutes a healthy and good body (Dew 2012; Fox 2012). Obesity is a paradigmatic example in the medicalization literature,

representative of how some aspect of one's biology or appearance that is superficially 'abnormal' becomes harmful when subjected to the framework of a biomedical model for health (Jutel and Dew 2014). There is a societal tendency to "medicalize that which we find morally unacceptable," such that children with obesity become "patients who must be cured" and parents are implicated for failing to protect their children (Moffat 2010: 5). Thus, just as the diagnostic label of obesity carries meaning in the social world outside the clinic, these broader social norms and beliefs about weight and health reciprocally influence medical knowledge and patient-provider interactions (Conrad and Barker 2010).

### *Current Study*

In describing the inherent diagnostic uncertainty of childhood obesity, I contend that health professionals dealing with issues of weight and health in early life are in an unenviable position. That is, they are trying to address a legitimate health concern, often before it is clearly observed as a health issue, while also being careful to avoid causing new problems in labeling children as "unhealthy." Striking a balance between these goals speaks to sociological research on uncertainty and diagnosis in medicine, and how these two concepts intersect. The complex biological and social etiology and sequelae of childhood obesity make it a challenging health issue to neatly define and categorize, and thus address in clinical settings. I do not challenge the fact that a high BMI can be and often is unhealthy in early life, but instead focus on the fact that practitioners' certainty about this relationship at the individual level of a patient is limited. Specifically, this chapter examines how uncertainty influences the diagnostic process, focusing on how children's BMIs are interpreted and used during clinical encounters.

Through in-depth interviews with health professionals working with pediatric patients, I examine how uncertainty affects communication about risk, the structuration of treatment, and the determination of what constitutes a successful outcome. Research on pediatric medicine is instrumental in exploring how uncertainty manifests in different clinical settings, and in broadening our understanding of how diagnoses differ based on the health issue under examination (Timmermans and Haas 2008). This study contributes to a growing body of research building a case for integrating children and youth into sociological research on health and medicine, recognizing the need to augment extant theory and

knowledge in light of their unique health experiences and concerns (Brady et al. 2015; Bray et al. 2014; Mayall 1998). Specifically, I focus on uncertainty in the *process* of diagnosis, by examining the act of diagnosis or how practitioners “do diagnosis” on a day-to-day basis (Armstrong and Hilton 2014), which remains an understudied topic with respect to pediatric care (Lutz 2019; Timmermans and Stivers 2018).

The chapter begins with an overview of extant literature on the role of uncertainty in diagnosis and its relevance for obesity, emphasizing uncertainty in diagnostic tools and criteria, as well as noting how diagnoses have come to encompass individuals’ future health. In using an abductive analytic approach to interpret and analyze the interview data (Timmermans and Tavory 2012), this study demonstrates how uncertainty can be a defining feature of the diagnostic process rather than treated as something to be ignored, avoided, or suppressed (Jutel 2009). I focus on BMI growth charts as a diagnostic tool and technique that circumvents the need to explicitly label a child as unhealthy, and thus mitigates the need for immediate solutions. Practitioners are less declarative about a diagnosis of obesity and shift the clinical narrative towards *prognosis* and what the future holds for a given patient and family – which is inherently uncertain. Moreover, the focus on diagnosis and treatment as a *long-term* process recognizes that individuals’ weight trajectories are often unpredictable – reflecting numerous influences outside the realm of medicine. In turn, this acknowledgement of non-clinical uncertainty broadens practitioners’ definitions and standards of success across patients and families. Ultimately, I use this insight on the beneficial aspects of uncertainty in diagnosis to challenge how we evaluate the success of childhood obesity interventions at the population level.

## **Theoretical Background**

### *Clinical Uncertainty*

Uncertainty is a central theme in medical sociology and research on the social construction of health and illness, presenting itself in various ways among practitioners and patients (Mackintosh and Armstrong 2020). Indeed, uncertainty was the defining feature of clinical care for much of human history, as health practitioners and healers practiced medicine with a primitive toolkit for examining the inner workings of the body and no systematic knowledgebase to guide their work (Foucault 1963; Jutel 2019;

Schubert 2011). In turn, the push to formalize medicine as a discipline – and thus establish a consistent set of standards, guidelines, and protocols to ensure uniform care (Timmermans and Berg 2003) – represents a decades-long effort to minimize uncertainty during clinical encounters and provide practitioners and patients with accurate and actionable information. A broad exploration of clinical uncertainty is beyond the scope of this study; however, the broader effort to mitigate uncertainty in medicine has had a profound influence on shaping clinical knowledge and practice as it related to BMI as a diagnostic instrument and obesity as a diagnosable condition.

The “evidence-based movement” (EBM) in medicine is most representative of this battle against uncertainty (Fox 2000; Lambert 2006; Timmermans and Angell 2001). Health professionals follow established protocols and guidelines based on “best” practices and “gold standards” of measurement identified in research (Timmermans and Berg 1997; Timmermans and Berg 2003; Timmermans and Kolker 2004; Upshur 2005). To the extent that uncertainty introduces subjectivity into clinical practice, EBM thus helps maintain “objectivity” (Cambrosio et al. 2006; Camrosio et al. 2009; Goldenberg 2006; Weisz et al. 2007). Clinical cutoffs or guidelines “map the area over which health care providers maintain professional sovereignty” (Timmermans and Berg 2003: 93), translating subjective, lay assessments of health into objective health standards, measures, and benchmarks. Obesity clearly reflects this push for standardization, wherein qualitative and holistic assessments of individuals’ bodies and health were replaced by quantitative measures like the BMI (Jutel 2006; Jutel 2009; Jutel 2011), which now serves as the basis for obesity as a diagnosis.

This is not to suggest that evidence-based decision-making or standardization represent negative attributes of contemporary medicine. It is uncontroversial to suggest medical practice should be based on the best available evidence, while standardization helps organize health care and facilitate communication across the many parties involved (Timmermans and Berg 2003). Rather, the concern is defining what does and does not count as “best evidence,” as maximizing ‘universality’ through the use of quantifiable and objective standards is contingent on minimizing the alleged uncertainty introduced by subjective experience and expertise (Berg 1997; Dew 2012; Goldenberg 2006). BMI is by many accounts a flawed

measure (Burkhauser and Cawley 2008; Gutin 2018), but it continues to be the “gold standard” for body size (Hu 2008) – and the defining attribute of obesity – because it represents the kind of objective measure of health prioritized by contemporary medicine.

### *Uncertainty in Diagnostic Tools*

The evidence that informs medical knowledge and practice requires diagnostic tools and instruments that allow for the collection and evaluation of ‘objective’ evidence about individuals’ health (Armstrong and Hilton 2014; Brown 1995). Namely, they are perceived as the objective and neutral counterpart to the subjective and biased assessments that come from practitioners (Schubert 2011). These tools and instruments have advanced medical care and saved countless lives, but medical sociologists have been careful to note that their perceived objectivity and infallibility – and thus the certainty with which they are used – is not necessarily guaranteed (Armstrong and Hilton 2014; Schubert 2011; Timmermans and Oh 2010). These tools create additional uncertainty when they are perceived to provide incomplete information about the issue at hand, as demonstrated in the case of invasive urodynamic tests (Armstrong and Hilton 2014), CT scans (Saunders 2008), or even more cutting-edge methods concerning genetic testing (Timmermans 2015; Timmermans et al. 2017). As these studies suggest, the subjective clinician is not absent from the diagnostic process, imposing their judgement and knowledge – and reacting to patient feedback – in determining how and when to interpret and use these diagnostic tools.

Uncertainty is inherent to many of the diagnostic tools now regularly used to measure health. With the rise of medical screening and the enumeration of ‘risk,’ the consistent measurement and tracking of various health metrics is an integral aspect of the clinical encounter. The influence of the ‘risk factor’ framework stemming from epidemiologic perspectives on health means that practitioners measure and monitor health even when signs of poor health are absent (Armstrong 1995; Armstrong 2012). This is not to suggest that medical screening is unimportant, or not integral to the practice of preventive medicine. Rather, the issue is that screening creates more uncertainty by expanding our societal notions of risk to categorize many seemingly benign behaviors and attributes as risky and unhealthy (Armstrong and Eborall 2012). While screening and diagnosis represent distinct concepts (Armstrong and Eborall 2012),

we use many of the same metrics to both gauge risk and diagnose health, giving rise to “proto-diseases” like *pre*-hypertensive, *pre*-diabetic, and overweight, rather than obese (Jutel 2006; Rosenberg 2007). The use of medical screening is well-intentioned in seeking to reduce uncertainty about individuals’ propensity for poor health in the future; however, these screening tools often introduce more uncertainty among those deemed “at risk,” leading them to conflate their *potential for* poor health with a perception of their *already having* poor health (Cupit et al. 2020; Gillespie 2015; Jauho 2019). Thus, many screening tools and measures situate individuals in an unsatisfying and uncertain state of diagnostic liminality, which creates problems for practitioners trying to communicate meaningful information to their patients and articulate a clear course of action (Armstrong 2019; Cupit et al. 2020; Gaspar 2020; Saukko et al. 2012).

This hybrid screening and diagnostic framework describes BMI, which “screens” for future risk but is also used to “diagnose” obesity. While we may not think of BMI as an instrument in a traditional sense like a stethoscope or pulse oximeter (Schubert 2011), it is a measure derived from *measured* height and weight, using a stadiometer and a scale. BMI is automatically calculated and incorporated into many patients’ charts, just as one would find with other diagnostic measures like blood pressure, pulse, or any lab work. The resulting values provide practitioners with some sense of patient risk, but there is little certainty with respect to BMI as a measure of individuals’ current health. BMI provides limited insight on body composition, as the biophysiological attribute of concern (Snijder et al. 2006). More importantly, there is a lack of consensus as to the diagnosability of obesity as a condition; the definition of obesity is being conflated with its measurement based on BMI (Gutin 2018; Nicholls 2013), rather than using a definition of obesity as a state of impaired health caused by excess adiposity (Sharma and Campbell-Scherer 2017). Thus, when used as a diagnostic tool, there is considerable uncertainty as to what BMI measures or what broader construct this measure maps onto.

#### *“Putting a Name to It”*

Despite the uncertainty in BMI as a diagnostic tool, many medical organizations have adopted the practice of formally labeling obesity as a disease in “putting a name to it” and legitimizing it as a

diagnosable health condition (Bray et al. 2017; Jutel 2014; Kyle et al. 2016). This decision relocates obesity further from its causes, such as diet and physical activity, and closer towards the kinds of comorbidities and outcomes that fall under the purview of medical care (Chang and Christakis 2012). More importantly, disease diagnoses are “non-negotiable” and “make no space for error or uncertainty” in defining healthiness (Jutel 2019: 66), and thus serve as a gateway for intervention and reimbursement.

However, this disease label has encountered pushback among those contending that this certainty is misplaced and diagnosing obesity as a disease is hampered by an inability to definitively determine “whether abnormal or excessive body fat is actually impairing [a] person's health” (Sharma and Campbell-Scherer 2017: 660). Indeed, scrutiny of the decision-making underlying this decision suggests that, ironically, the diagnosis does not come from a place of certainty in being able to “scientifically prove either that obesity is a disease or that it is not a disease,” but is instead a response to how this uncertainty in classification causes problems for practitioners and patients that *needs* to be resolved. Consequently, medical organizations adopted a “utilitarian” position (Allison et al. 2008: 1162) – asking whether obesity *should* be called a disease, rather than whether it *is* a disease – in recognizing that health conditions “come to be considered diseases as the result of a social process when it is assessed to be beneficial to the greater good that they be so judged” (p. 1161). The diagnosis is thus a pragmatic concession – or a “bureaucratic and an emotional necessity” (Rosenberg 2002: 256) – sanctioning obesity treatment as a billable transaction.

Diagnoses are designed to impose certainty for practitioners, and the medical system at large, but this certainty is not without consequence for the patients to whom diagnoses are assigned. Diagnoses redefine individuals’ health, with the “diagnostic moment” marking a critical transition from healthy to unhealthy (Heritage and Macarthur 2019; Jutel 2014). The label of “obese” can produce “an instantaneous – and traumatic change in [individuals’] sense of self” and perceptions of their overall health (Greenhalgh 2015: 113; Jutel 2014). Though the disease label is used to legitimize obesity as a health issue and remove personal blame, this diagnosis cannot be disentangled from the social consequences of labeling individuals as diseased and unhealthy in a society where body size is construed as the product of poor

choices (Greenhalgh 2015; Saguy 2012). Thus, while diagnoses legitimize obesity in medicine, the need to impose certainty about good versus bad health has consequences for individuals whose bodies do not conform to ideals and norms about who is considered “healthy” and “good” in our society. These unintended consequences are anticipated in extant theories in the sociology of diagnosis (Jutel 2009; Jutel 2019). However, we lack more comprehensive knowledge of how the meanings attached to diagnostic labels may impact the diagnostic process itself, influencing how practitioners discuss diagnoses and treatment (Heritage and Macarthur 2019).

### *Diagnosis as Prognosis*

Recognition of diagnoses as laden with both clinical and social uncertainty has led scholars to reconsider how diagnosis – as a process – often transcends the confines of a given clinical encounter. Though diagnoses can provide certainty to practitioners and patients in search of a clear name or label for a health condition (Jutel 2019), the diagnostic process is often less clear-cut than the guidelines and standards promoted under EBM might suggest. Much of contemporary health and medicine is defined by conditions that unfold over the course of many years, and thus diagnoses are often made with limited insight on a patient’s health trajectory. Moreover, this trajectory is not solely a function of individuals’ latent health; it is also affected by many non-medical factors. Recognition of this multi-layered uncertainty informs the social diagnosis framework (Brown et al. 2011), which emphasizes the concurrent influence of practitioners, patients, and the many individual- and social-level actors and forces shaping diagnoses and health. As Brown and colleagues (2011) explain, this comprehensive theory of social diagnosis makes “time” as a critical dimension of treatment in bringing these various actors and forces together: diagnoses are often less contingent on the past and present experience of a condition as much as they “explicitly consider the *potentiality* of future conditions” and the extent to which this potentiality is shaped by social causes and liable to have social consequences (p.941). Thus, the inherent individual-level biomedical uncertainty about patients’ diagnosis and the trajectory of their condition intersects with macro-level uncertainty about their social circumstances (Brown et al. 2011).



Temporal uncertainty is critical to more recent work on the sociology of prognosis, which examines how health professionals allow for uncertainty in practice and deviate from established protocols when faced with unclear prospects for their patients. While the prognosis framework has generally been applied to end-of-life circumstances or terminal cases (Christakis 1997), Timmermans and Stivers' (2018) analysis examines prognosis in the context of chronic illness, focusing on the trajectory of epilepsy throughout childhood. Critically, their work documents the utility of prognoses as a means by which practitioners avoid declarative – and potentially incorrect – conclusions about the severity of a condition, and instead emphasize plausible trajectories. Practitioners were unlikely to communicate explicit diagnoses to patients and families and would instead “tip their hand” about their expectations (Timmermans and Stivers 2018). In turn, this openness about uncertainty helped facilitate a view of patients as representing individual cases rather than trying to situate their prognoses in a fixed framework of guidelines and protocols; likewise, it helped maintain a collaborative, long-term relationship with patients, as is crucial to long-term treatment (Timmermans and Stivers 2018).

Indeed, the importance of the individual – as more than a clinical entity – is central to the prognostic model of clinical practice. Echoing Brown et al.'s (2011) emphasis on accounting for non-biophysiological factors, Croft et al. (2015) contend that the emphasis on diagnosis in contemporary medicine enforces an unnecessarily limited view of patients. Diagnoses reinforce a binary view of health and disease, which implicitly categorizes individuals into homogenous groupings based on their having or not having a given condition, rather than considering how their condition may interact with various non-clinical aspects of their life (Croft et al. 2015). By contrast, prognosis “offers an alternative starting point with wider incorporation of factors relevant to patient outcomes than diagnosis alone”; namely, the framework encourages the collection and integration of information on the totality of a patient's biological, psychological, and social environment, which may convey more certainty about future health than data from imprecise diagnostic tools and cutoffs (Croft et al. 2015). In this way, prognoses prioritize clinician's own judgement and “shared exploration and understanding” with patients in evaluating all of

the information on hand and identifying what outcomes are truly “wanted or needed” for all parties involved (Croft et al. 2015).

## **Methods**

This study draws on data from a purposeful sample of health professionals seeing children and adolescents, as well as their families, in clinical settings. Recruitment was conducted at a large university hospital system via email listservs for various departments and working groups, as well as in-person recruitment at a research group specifically focused on childhood obesity. The email and presentation provide a general overview of the project – i.e., approximately 60-minute interviews focused on the diagnosis and treatment of childhood obesity – and offer an incentive for participation. Following this initial recruitment, participants were asked to circulate details about the study among their practices, departments, and additional organizational listservs (Biernacki and Waldorf 1981). This study was exempted by the institutional IRB following a review of the recruitment methods and interview guide (#19-2361).

In total, 28 participants contributed to the study, representing a diverse group of practitioners in terms of years of experience and areas of expertise in diagnosing and treating weight and health in young populations. Half of participants were general pediatricians (N=14), with five to 30 years of clinical experience. Their perspectives were complimented by clinicians and residents in family medicine (N=3), behavioral specialists (e.g., adolescent psychology and dieticians) regularly seeing children and adolescents with overweight and obesity (N=5), and medical students with clinical experience (N=6). Except for three of the medical students and two of the behavioral specialists, all respondents were actively engaged in clinical practice during the time of interview, though the proportion of time in clinical hours versus other activities varied between 30 and 100%. In general, respondents described working with fairly diverse patient populations or, as one pediatrician explained, “one day I see a kiddo whose dad is the head of cardiology, and the next day I see someone from a very low-income, under-resourced area.” Some respondents also noted that they were bilingual, and probably saw more Hispanic patients and families than other clinicians that they work with.

Interviews were conducted between January 2020 and March 2021; due to the overlap with the COVID-19 pandemic, 24 of 28 interviews were conducted using videoconferencing software, which were recorded with the participant's consent. These semi-structured interviews encouraged an open, but focused, conversation about how practitioners approach weight and obesity as a health issue in early life, how they communicate on this subject with patient and families, and how treatment is provided and monitored (see Appendix). The interview questions were piloted as part of a shorter set of interviews with clinicians one year prior to data collection to help better specify questions and identify appropriate terminology; any clinicians participating in the earlier stage were not re-interviewed. Interviews ranged from 45 minutes to 1.5 hours, depending on participants' availability. The recorded interviews were transcribed and matched to any memos written while they were being conducted.

The overall analytic approach is informed by the abductive framework outlined by Timmermans and Tavory (2012), using a version of flexible coding suggested by Deterding and Waters (2018) for researchers using QDA software to conduct abductive analysis of interview data. Extant medical sociological theory helped to motivate interview questions on uncertainty in practitioners' approach to diagnosis and treatment; the abductive analysis framework allows for this extant knowledge to serve as the basis for identifying key themes in the data, upon which coding iterates and elaborates. However, this abductive approach encourages researchers to privilege "observational surprises or puzzles" rather than ignore them in favor of predetermined theories (Timmermans and Tavory 2012: 169). In the case of this study, reviewing transcripts during both data collection and analysis yielded interesting and recurring insights on "trajectories" and "growth" as both a diagnostic tool and mentality in the care of childhood obesity – speaking to, but also deviating from, extant theory on diagnosis and uncertainty. Likewise, I used an iterative coding scheme based on multiple reviews of transcripts. Initial coding identified broad themes consistent with extant theories of diagnosis and uncertainty; these codes were then re-evaluated in light of findings specific to childhood obesity, with an eye towards illustrative quotes (Deterding and Waters 2018). All interview data were coded and analyzed using Dedoose Version 8.0.35 (2018).

## **Findings**

### *Diagnosing “Growth” Rather Than Weight*

During interviews, practitioners were asked how and why weight is brought up during a clinical encounter and prompted to describe typical interactions with patients and families. Most respondents explained that taking height and weight is a now standard part of the “flow” of clinical visits, such that seeing and reacting to BMI is a very natural. Dietician Patricia casually notes how “BMI just shows up” in patient charts, conveniently categorized as “either red or not red” in relation to obesity, at which point her concern is making sure to enter the correct diagnostic code and initiating the conversation. Indeed, several respondents spoke positively of how automatically BMI is incorporated during the visit. General pediatrician Nancy noted that using BMI is a more recent innovation given the advent of electronic records, emphasizing that she used to not use BMI “because I’m not going to sit there and a 15-minute visit and calculate someone’s BMI, because I got too many things to do.” She noted that BMI, and BMI growth charts, were a “great tool” as compared to past protocol of asking “what did the kid look like” and “going through what their diet and habits were.” Likewise, general pediatrician William appreciated how getting BMI into electronic health records “just puts it in front of us” to start the conversation with families. Along these lines, adolescent medicine specialist Elliott described how he “might sort of edge into it a little bit more peripherally” in starting a visit by “just go[ing] through the numbers” – and bringing up weight alongside height or blood pressure – before talking about patient experiences.

Despite these conveniences, practitioners expressed apprehension about how these numbers shape the clinical encounter. Medical student Sandra noted that seeing and talking about BMI felt “almost like a reflex”; while having the conversation is important, she felt “forced to think about weight” and worried how it made it “easy to write off all of the problems” a patient has with obesity. Similarly, general pediatrician Erika explained that “get[ting] everyone to a healthy BMI” is often the only framework she has during clinical encounters, and BMI is “the only tool” available to make decisions. Both Sandra and Erika explain how the ubiquity of BMI in clinical settings does not always square with the fact that patients present other health concerns that need to be prioritized, or they feel healthy and being presented

with a diagnosis may not be productive. Sandra described a patient with back pain who has been repeatedly told to lose weight as the solution; yet imaging found no signs of something being physically wrong that were attributable to weight. Sandra maintained that a healthier weight would benefit the patient, but it was important to recognize that this “was not the solution [the patient] was looking for” or the “solution that would work for her at the point she was at”: rather than focusing on weight, Sandra retrospectively felt that it would have been better to acknowledge the patients’ specific concern. Erika also noted how the parents she interacts with probably feel like “all [doctors] want to talk about is my kid’s weight,” ignoring families’ other concerns. Erika wondered if she and other pediatricians are part of the problem in bringing up weight at every visit, which makes patients feel like “if their weight is not within the healthy zone of a BMI, someone’s going to tell them about it, and someone’s going to shame them about it.”

The latter point presented a conundrum for many respondents: the evidence practitioners are presented with instantaneously informs them of whether a patient is healthy or not based on BMI, but they have good reason to withhold a diagnosis from patients and families. When probed on how they use the diagnosis of “obesity” during clinical interactions, all but two of the respondents reported ever using that word – or the label of “obese” – in talking to patients and families. The two respondents who did use the term worked more with patients and families seeking treatment for severe long-term obesity, wherein bariatric surgery or medicine might be the next option. Otherwise, some of the longer-practicing practitioners mentioned it being a term they used early on but no longer employed.

Specifically, respondents did not see the utility of using obesity as a diagnosis given the inability to clearly state something is wrong with patients. Medical student Kimberly did not disagree that it was wrong to describe obesity as a disease but acknowledged that using the term was not very helpful because she could not definitively say “what that means for my patient right now” in the sense that “clinically, I can’t tell anyone here’s why it’s bad.” Family medicine resident Jonathan also did not think the diagnosis was helpful when patients had other concerns given that “weight is not something that will kill you

instantly” and thus not “an imminent threat”; thus, he was reliant on the patient to know when to discuss weight.

However, the primary motivation for avoiding a diagnosis and not mentioning obesity was recognition that the medicalized language surrounding weight can be harmful and counterproductive, with “obese” being a harmful label in a clinical setting. According to general pediatrician Frederick:

[M]ost people who are obese know they're obese. And they get so many negative messages about their obesity. And they know, or have heard over and over again, that there's an epidemic of obesity that is out of control and problem. [They] probably heard from other clinicians before about their obesity and may have attempted to achieve some improvements in their BMI and it had failed. And now here comes another condition. All right, tell me what I already know.

Frederick explained that the diagnosis and label was an obstacle to maintaining a positive atmosphere in clinical settings. This sentiment was echoed by other practitioners noting how important it is to keep patients and families “engaged” when it comes to weight. William observed that families interpret obesity in unpredictable ways; in some cases it might be helpful, but he is concerned that the term “ostracizes people or turns them off” and “maybe erodes trust” when it runs counter to patients’ and families’ experiences. Pediatrician Nancy summarized this conundrum in recognizing the need to talk about weight but worrying how to do this “without causing another issue?” Nancy tried to balance the harmful connotations of obesity by reinforcing all the positive aspects of a child’s appearance because she thought “most of them look in the mirror, and they don’t see that happy stuff at all.” Elliott has a similar mentality when it comes to “articulat[ing] the positive truth” of a patient’s health. As a doctor, it is his responsibility to tell adolescents the truth, but to “do it in a way that they understand” – which involves telling them the “full truth, both the good and the less good” when it comes to all the things going right.

Thus, practitioners tried to be cognizant of BMI as a number that provided objectivity and evidentiary basis to care, but that needed to be contextualized among patients’ other attributes. This was observed in their framing of obesity as a diagnostic “code” – in a bureaucratic sense – rather than something that provides greater understanding for the patient and family. General pediatrician Cassandra conveyed the opinion of many respondents in explaining “it’s most significant from a billing standpoint”

given the emphasis on “coding for as many diagnoses that exist as possible.” She drew a comparison to riding a motorcycle as a situation where “your lack of helmet wearing isn’t a disease” in the sense that disease better communicates the risk compared to simply having a discussion with patients. General practitioner Joseph avoided using the term obesity in clinical encounters, but on the rare occasion where he was asked by families he was careful to explain that it is “not an adjective,” but just a diagnostic formality.

More broadly, this distinction between practicality and clinical utility in diagnosis was seen in respondents’ perceptions of how much information BMI provides about patients. Practitioners acknowledged BMI as a limited measure, highlighting different sources of uncertainty in the measure and how it maps onto health. Some practitioners, such as William, recognized BMI as, at best, a screening tool, knowing that lab work was unlikely to show clear evidence of insulin resistance or more serious concerns. In working with young patients, William viewed his role as “preventative,” noting how they are “still, hopefully in the front end of [health] and there may be things smoldering” like a high BMI. The measure is not sufficient to describe underlying health and thus William acknowledged that “I don’t have a diagnosis. You’re not treating anything, you’re, you’re just heightening your antenna for prevention.” Kimberly shared these concerns, wanting a more “meaningful” definition of obesity that is “not just a number” because number definitions are “what we like” in medicine. The emphasis on identifying a diagnostic threshold that corresponds with this number represents yet another source of uncertainty for William, as well as Cassandra, both of whom felt that “nothing magical happens when you go from the 84<sup>th</sup> to the 85<sup>th</sup> or 94<sup>th</sup> to the 95<sup>th</sup> percentile [for BMI]” (William) or that a “BMI of 32 is that much better or worse than a BMI of 30” (Cassandra). This combination of measurement and diagnostic uncertainty motivated Erika’s highly-critical position on BMI, leading her to question whether BMI “means anything at all?” Namely, she described an evolution in thought on BMI, transitioning from an unquestioning position influenced by her medical training to a skeptical perspective on BMI as a “made up thing” based on both personal professional experience of its discordance with individuals’ perceptions of themselves as healthy. Yet, Erika acknowledged the impetus to “measure something” and that “there has to be a line

somewhere” when it comes to BMI and obesity; thus, while she continued to use the measure, it is “now more as a signal rather than a final label.”

Indeed, many respondents expressed this sentiment – feeling that they had no choice but to continue using BMI given a lack of alternate measures and due to its centrality in bureaucracy. However, rather than fixate on its limitations, practitioners were strategic in reorienting their use of BMI as a measure of *growth and development* as opposed to a diagnostic measure of health. Practitioners explained how BMI is situated on a growth curve relative to both the individual child and other children of their age, which is then shown to parents and families as an indicator of a child’s overall trajectory. In turn, the emphasis on future outcomes allowed practitioners to convey the appropriate level of concern without causing harm and disengagement. Erika explicitly used this strategy to circumvent the problems with obesity as a label and diagnosis:

I do talk about growth at every well child visit that I have. I will talk about BMI. And the way I talk about it is more that BMI is a general indicator of your weight and your height together. So I'm not focused on your weight as a number because I get a lot of things about like, how much should we weigh? But that's not where we're going... Like, let's talk about BMI more in the context of like proportionality of your weight. But that's kind of how I'll approach it is just and then I'll say not I will never say like, we want your kid to lose weight... And to attain a healthier weight. I'll often tell them, depending on how old they are, how much growth potential they have, you know, if you could just keep your weight the same and grow taller, we're gonna even this out!

Other practitioners noted how this growth perspective on BMI helped shift the nature of the clinical encounter from disease and diagnosis – and a focus on the child and their health at present – to a gentler language of concern and worry about deviation from these trends. For instance, Nancy described the diagnostic utility of growth curves:

I always pointed out the growth charts and I always talked about how we got a pattern here that can lead to trouble. And I always point out from the very beginning: this does not show me what you look like. This doesn't tell me anything about you. This is just a pattern.

Likewise, general pediatrician Olivia noted that growth and development are the defining aspect of pediatric medicine and provided a natural opportunity to discuss any concerns. Olivia looked at the growth curves to describe healthy growth in relation to both inches and pounds, telling patients and



families that discordant trends for these two measures might be a cause for worry with respect to having a healthy weight.

From a diagnostic perspective, situating BMI in this longitudinal, growth context also shifts the focus to *potentiality* and *prognosis* (Brown et al. 2011; Croft et al. 2015; Timmermans and Stivers 2018), encouraging practitioners to set aside the obese/non-obese diagnostic binary in favor of a more holistic perspective on future health. Joseph described how a future-oriented mentality influenced his diagnostic approach, given the uncertainty in what BMI means at a given point in time:

Even if I have a kid who's at the 50<sup>th</sup> percentile for their BMI, but their intake is largely soda and unhealthy foods, I tend to spend a lot of time talking about future cardiovascular health and stuff. As opposed to, you know, only worrying about what their BMI looks like, or who's at the 84<sup>th</sup> percentile for BMI. But if they were at the 30<sup>th</sup> a year ago, and the 50<sup>th</sup> six months ago, I know that even though they're not even in an overweight category... if the appointment is three days later, they would be. So, it's not so much I have a different framework for obesity, specifically, in my head, but in terms of patients who I am more worried about and spend more time talking about nutrition and activity with, it's people who habits are leaning towards unhealthy or people who's who are moving in the wrong direction.

Later in the interview, Joseph described how his own children followed a non-standard trajectory of sharply increasing BMIs before suddenly falling into a healthier range. This recognition of looking beyond point-in-time estimates was echoed by pediatrician Tina, who is not “100% convinced that all of us belong on the same growth curve,” which makes it difficult to understand what a certain BMI means at a given point, or how to characterize a brief period of increased or decreased weight due to the complex interplay of genetics, the environment, and a host of other factors that practitioners cannot account for.

Practitioners were not ignoring the health implications of an obese BMI; rather, they emphasized patterns and trends to signal concern about the child or adolescent patient as a hypothetical future *adult* patient – extending the BMI and health trajectory beyond early life. Practitioners’ prioritized prognosis and raised concern without diagnosing and labeling the child at present. Talking about other health conditions or diseases *associated with* obesity in adulthood provided practitioners with yet another strategy to acknowledge the lack of certainty in what obesity means as a diagnosis, and what BMI signals as a diagnostic tool, while continuing to fulfill their responsibility as care providers. For instance, Nancy

and Kathryn both used growth curves to initiate a conversation about maintaining a healthy lifestyle that helps children avoid “going down the road” (Kathryn) or “pathway” (Nancy) of higher risks for heart disease and hypertension. Joseph openly told parents that he “can’t look into a patient’s future,” but the fact that a child seems pretty healthy at the moment doesn’t mean he can “predict... what their heart is gonna be like 50 or 60 years from now.” He acknowledged that this could go either way, and that even a child with a high BMI can be fine, so his goal “isn’t to make your weight X, Y, or Z in 2021 or 2022” but “to have you having healthy habits grow up... that are going to keep you healthier and alive longer.” As Elliott explained, clinicians are on a long-term trajectory with patients, and this relationship requires acting in a way that maintain this relationship. In talking about weight, Elliott adopted a neutral position in explaining that his goal is to work together with patients to make sure they are “not held back by any health issue” in the future, rather than providing a diagnosis and “tell[ing] them you have a disease.”

*Delaying diagnosis to maintain the patient-provider relationship*

The emphasis on maintaining a working relationship with patients and families was a recurring theme throughout the interviews. Simply assessing a patient’s BMI was insufficient; practitioners also needed to ‘diagnose’ a child’s and family’s level of emotional and cognitive readiness to provide the best possible care. Rather than being prescriptive in their advice and course of treatment, respondents expressed a desire to “meet patients where they are” (Joseph) on this long-term trajectory of weight and health. In turn, clinical encounters focused on understanding and responding to ‘where’ the patients and families are rather than reacting to where the BMI is and what actions need to be taken. Nancy described this aspect of her clinical duties as “detective work” where a visit might start with patients and families telling you “everything is perfect, and then you start asking some questions and things start coming out... and suddenly you’re in the game.” Nancy knows there are no guarantees or certainties in how these conversations go, mentioning how a lot of the challenge is to “just try and fine tune it as you’re talking” in figuring out what children’s and parent’s concerns are and how to address them. Patricia explicitly said that it is “super boring to reflect upon the fact that that’s happening” in simply seeing that a child has obesity; the challenge is “decid[ing] whether or not [mentioning it] is appropriate in that moment.” That

decision is often based on incomplete information; Elliott explained that a patient might be a “10 out of 10 on importance” in understanding the importance of losing weight, but a “2 out of 10 on confidence” and thus the priority becomes giving them “a framework of hope” and providing “some sort of affirmation of their value and worth and potential for change.”

In turn, practitioners were very open about how the diagnosis and treatment of childhood obesity is shaped by negotiation and compromise with patients and families to preserve the relationship and maintain “buy in” (Nancy). Patients are not always where practitioners “need them to be” in terms of “even thinking about changing” (Erin), and practitioners know that they are not going to make any progress until patients reach a baseline level of “finally wanting to set goals for themselves” (Joseph). This meant that a lot of the clinical protocols were accommodating of uncertainty in patients’ and families’ understandings of and beliefs about weight, obesity, and health. Many respondents described forgoing a clinical and diagnostic language to facilitate a working – rather than prescriptive – relationship. Family medicine clinician Robert, explained how emphasis on this long-term relationship shapes her diagnostic approach:

I’m much, much more dedicated to this conversation because I’m now their primary care doc. I’m going to take care of them for a much longer period of time. And so I personally am more invested in making sure to have a thorough conversation and really explore: What does this mean? What does this weight mean to you? What does it mean for your body?... How can we start having a conversation that’s valuable to you about changing some of the numbers that are valuable to me? And how can we do that together?... And then use that response as a way to move forward.

Nancy also questioned how the things she said were interpreted, asking “What’s gonna mean the most?” in finding strategies that motivated patients since “I can tell people all day what I think they should do, but it may not be what they should do” based on where they are. This search for the interpretative meaning of diagnoses was difficult for practitioners because it runs against their training to “fix things and make them better” (Olivia); however, “changing the approach of the visit to be more collaborative and patient-centered” rather than just diagnosing children is important for “using what [patients] want” to structure care. Elliott also recognized how much of his training involved a “one size fits all approach” but experience has shown that when it comes to obesity, “it’s not going to always be a clinically oriented

conversation, like, we're going to do this, this, and this to granularly modify a particular number to our liking.” Rather, the trick was finding a “backdoor” into weight issues that works for individual patients.

Indeed, the interviews revealed uncertainty and apprehension in how explicit practitioners were when discussing weight and obesity; just because the clinical evidence suggested that weight merits attention does not mean that having a diagnostically oriented conversation was the best course of action. Later in the interview, Elliott explained that there is an “ideal situation [where] a young person is wanting in that moment” to have the conversation about weight, but that this is almost never the case. Elliott acknowledged that it is not a pleasant conversation to have or a diagnosis to hear, and the issue is not that he thinks he is saying “something that’s overtly wrong, or negative, but it’s just the timing” of the conversation being inappropriate. He maintained that “forcing the conversation in a particular direction because, clinically, I have to, or I’m supposed to in that moment” was unlikely to have a good payoff. Though it may run counter to clinical protocol, Nancy knew that it was important to “figure out where that family is in what they want to hear” because they might just “tune out if you bring up weight.” She instead changed the subject, knowing that “if I bring it up totally as something else, as a healthy, whatever, for whatever it is, they're more likely to hear” than bringing up weight.

In balancing this need to address weight as a health concern, while not imposing it on patients and families, respondents were comfortable extending the diagnosis of obesity over an extended period. Namely, in meeting patients where they are, practitioners set aside or delay the conversation about obesity, with the hope that both practitioner and patient interests are better aligned in the future. For instance, William explained that for some families the primary objective was simply “planting a seed” and then maybe “a year later, six months later, I am going to bring it up again, and they're gonna say, Well, you know, what, maybe we can talk about this a little bit more.” Joseph had a similar mentality about working with families to figure “what’s gonna work best for them” and to “know how far I can push them on things”: even if some families are incredibly responsive, for others “it’s almost like a one- or two-year orientation process.” Elliott described this as a trade-off between short- and long-term gains

in the larger narrative of patients' lives and health, wherein a diagnosis of obesity might cause more harm than good:

I think we all sort of acknowledge at some level weight is... an expression of a variety of things. And so I think it's helpful to try to sort of put it on the radar at some level [and] raise it as part of the conversation. But then to like, park it in a particular spot, and come back to it when it seems appropriate, right?... I see it as a long-term thing. And the only hope that I can help a young person with a long-term thing is if we engage in a long-term fashion.... And if I rush to judgment, and I sort of push my agenda upon them to do it like today, when they're not ready for that conversation, you know, I sort of see that as a loss, big picture, right?

More broadly, nearly all respondents recognized that individuals – and especially children – were not ideal, rational actors who perfectly respond to health messaging and recommendations, and that there were many other factors at play influencing how a diagnosis related to weight and health is likely to be received and responded to.

#### *Defining Success Without a Diagnosis*

Given these uncertainties in patients' diagnostic trajectories, coupled with uncertainty in their cognitive and emotional preparedness to receive or act on concerns about weight and health, practitioners provided a broad definition of 'success' when it comes to characterizing patients' and families' progress. While weight loss may be the most obvious measure of success for a condition defined by weight, William noted the disconnect between the binary nature of a diagnosis and the nuanced reality of how and why a patient acquired this condition:

It's unfortunate that we've gotten to this point of needing a number for... defining that cohort with no other way of defining kids that don't have other manifestations. And trying to hold that with the fact that there are 1000 different ways and 1000 different influences on that number. Yeah, it's just a weird thing. We have a disease that's defined by excess adiposity, which can be measured with two numbers.

The emphasis on these numbers does not reflect the fact, as pediatrician Samantha explained, "in real life none of us have the same endpoint when we're talking about our values for our own wellness, [which] are all over the map." In that sense, practitioners like Elliott contended that patients can have legitimate definitions of success, and sometimes the doctor's job is to assess the feasibility of their goals and "negotiate around it" to "help them to flesh it out a little to... make it a bit more accessible." One can

observe the influence of thinking about patients on a trajectory in guiding these definitions of success, with pediatricians like Joseph explaining that the key question he asks himself when defining success for individual families is “where are you on your curve?,” knowing that the answer influences their probability of success.

In broadening their definitions of success to include patients’ and family’s input, many respondents were responsive to the notion that the field of medicine may, indeed, not have a “safe or reliable” way of keeping off weight (Greenhalgh 2015). Their challenge was instead to justify outcomes that adhered as closely as possible to safety and reliability – and thus sustainability – for the individual patients and families they work with. Tina recalled how in medical school she was taught “to be very prescriptive in care, meaning, we find a problem, we define that to the patient, and then we tell them what they do about it” but that this approach does not work with obesity; rather, she shifted towards “allowing [patients] to team with you, but really form their own plan, because they know themselves better than we do and what they are capable of and what they may or may not do.” Medical student Adele explained how this mentality helps her be more proactive when not seeing weight loss, leading her to ask “what’s been going on?” and probe deeper to realize that telling a patient to exercise after work might not be feasible as compared to just “taking the stairs at work, or taking a 10-minute walk break during lunch” as well asking them to generate ideas and then help tailor them. Elliott commented that many patients have well-intended goals, but that they are “actually just not reasonable” or maybe “just kind of unfounded” even if they would result in a patient no longer having obesity.

Indeed, despite obesity being defined by BMI, this was rarely the key metric of success described by respondents. Many practitioners are happy to see patients’ BMIs decrease, but their preferred definition of a successful outcome involved a more holistic assessment of the patient. Nancy explained how “all of a sudden, [a patient’s] self-esteem is better, they have a healthier relationship with food and how they think about it, they have confidence to do whatever it is at a school..., they have more energy [were] all wins” even though “the weight didn’t change.” Likewise, pediatrician Joshua, who primarily works with patients with very severe obesity, used a broad measure of success and “not necessarily

focusing on a particular BMI,” telling patients that she wanted to make sure “you feel content in your body.” Approaching success from this perspective reinforced practitioners’ concerns about the utility of BMI as a diagnostic tool, especially if it contradicted patients’ experiences. Tina described how her definition of success has changed towards prioritizing “how the family feels like they’re doing” and “do they feel good about changes” even in those instances where “their weight is the same... or it's going up a little bit.” She was uncertain about how to proceed, explaining that:

I don't want to show them the growth curves... I focus more on the history and follow up than I do in the actual numbers because it just feels like patients make a big change and sometimes those numbers don't confirm it. And that's just wrong. Like, why do I even bother?

The actions patients and families take to improve their health, and what the scale says or where their BMI falls on a growth chart, often follow different trajectories and time scales, especially in early life. Cognizant of this discordance, practitioners described goal setting and measures of success that may not comport with clinical standards of how we define healthiness and the types of behavioral changes that engender weight loss. In addition to the holistic emphasis described above, many practitioners focused on “really small things like... rather than drinking three bottles of juice a day, they’re going to drink one bottle” even if the practitioner did not want them drinking any juice (Kimberly). Similarly, G.P defined success as something as simple as “go[ing] for a walk around the block once a week”; she looks at their lab results and BMI but notes that “I really am mostly thrilled when they actually do the things that we discussed last time” or to even just “try” to do these things. Practitioners identified activities that are particularly important to children, like “joining a dance team” (Samantha) or “starting to play soccer” (Frederick), as worthwhile indicators when “things haven’t improved on the graph” (Frederick). Kimberly noted that some of these measures of success can “feel weird” and recounted an example of an endocrinologist who works with adults “cringing” when hearing about how the goals of obesity treatment are so low. However, Kimberly and other practitioners emphasized that if these are meaningful changes for patients, then they should be meaningful to providers as well.

Despite efforts to shift the nature of the clinical encounter and diagnosis away from obesity, many patients and families continued to define success based on weight, which provides a more direct and tangible measure than changes in behaviors and beliefs. Olivia talked about how she might try to focus on these small successes like *“whether we were able to try a new vegetable every week,”* but it is often the case that “parents are like, I don’t care” and the first thing they ask in the visit is “What was their weight? Did they lose weight? Yeah, how much weight did they gain?” Patricia also noted this tension between trying to “celebrate the small behavioral changes” and encountering resistance among patients who “want to see weight loss” or having a mentality that “all their progress and changes are medical.” Samantha attributed this tension to confusion about BMI and the fact that “we’re a very weight-focused culture.” Even when she explains the concept of a BMI curve, how it varies across children and over time, and stresses the importance of long-term lifestyle changes, the response is a blunt: “Okay, I get it, and how much weight should she lose?... How much should he lose? What’s an optimal weight for my child, like, the weight, pounds? Like that’s sort of their focus.”

Practitioners try to de-emphasize the importance of weight as a measure of success, but both patient’s and medicine’s desire for unambiguous and objective indicators of progress continues to be an obstacle. Respondents were sympathetic to patients’ and families’ confusion about what it means to be healthy based on weight, and how that intersects with social norms and expectations. Adele commented on how she’s seen “a provider be like, let’s try and climb the stairs more at work and a patient’s like, well, that’s not gonna get me to my 20-pound [weight loss] that I want to do.” She explained that this mentality meant that a lot of patients already felt like they failed when walking into the doctor’s office as the first thing they do is step on a scale and see the number is not where they want. Elliott was aware of this “super binary, black and white... success versus failure mindset” in patients that is “driven by the number”; thus, he tried to “diversify their view of what success could look like.” However, he knew that there was mixed messaging on BMI in the doctor’s office: “We spend so much time trying to like acknowledge the number and use it for what it’s worth and recognize that it’s a legit data point, but then sort of really quickly backtracking and saying, like, it really doesn’t show the whole story.” Once again



emphasizing the importance of having a long-term view of weight and health, he tried to explain that patients should “not take too much stock in the number” because they “don’t truly have control over what the scale would say to you on a particular day” as opposed to looking at a trend over “a month or six months or a year” and how that is related to what you are doing. Samantha, who splits her time between research and practice, provided additional insight when comment on how much of the emphasis on BMI as an unequivocal measure of success comes from being “shoehorned into picking an outcome number, and that often is BMI, or relative BMI, or percent change or whatever”. These metrics are pervasive in the clinical and social context, but they do not always make sense for patients and families. Erika summarized this disconnect in the kinds of measures of success dictated by clinical standards and diagnoses as compared to the kinds of goals that work for individuals:

I mean, this is, you know, qualitative versus quantitative, right? Where the only the only outcome that matters is did the BMI come down?... When actually, what if that kid feels stronger? What if that kid feels healthier? What if the goal of doing exercise wasn't to punish your body for eating?... Or toward losing weight? And if you don't lose weight, then you failed? What if it was like, my back doesn't hurt as much?... Or, you know, I can run and play with my friends, and I don't get tired?... What if those were our outcomes? And so I feel like those are the ones that actually matter.

In turn, patience can be practitioners’ most valuable skill, in acknowledging and responding to the uncertainty in what works and why – and how long it might take – when it comes to seeing the weight loss that patients and families desire. Periods of plateauing or increases in weight may not be a signal of something being wrong, even if this is something that patients and families tend to fixate on. Frederick is very open with patients and families about the difficulty of seeing quick and clear improvements in weight, and gave an example of how he redirects questions like “what is my weight today?” towards his personal interest in “and emphasizes tangible accomplishments like looking for long-term changes in lifestyle [that] we hope will lead to healthier outcomes in the future” walking more. Olivia noted that having this patience, and maintaining positivity, is difficult to maintain when it’s hard to point to clear signs of improvement. Nevertheless, she described the importance of “focus[ing] on the loop” in terms of creating a positive feedback cycle, which can be broken by fixating on weight:

You know, we often have this conversation at that point where it's like, I'm doing all the right things, and I'm making all these changes – how come my weights not improving? It's a tough one. It's hard to square. But I do try to be transparent with them that the changes are good, and that there are some parts of their health that they can't see that are improving. Usually though there is always something you can point to is better. It's not the number on the scale, like, they can run more laps or that, you know, they're thrilled that now they're eating vegetables.

Samantha described how this patience and long-term mentality on the diagnosis and treatment of childhood obesity has a payoff. She recounted experiences where something has changed for the better, even if she was not certain about what exactly happened. She, and other practitioners, hoped it was a function of accumulated messages and behaviors over time, but also recognized that it could be due to factors shaping patients' lives, weight, and health outside of the medicine entirely:

I've had some families that have stuck with us for a really long time, like eight years, 10 years. Like a really long time. To the point where their kids left home. It really took working on this for a long time and then something just like changed, you know, something just stuck... The behaviors they had to remind, remind, and remind and model and make the environment safe for healthy eating and blah, blah, blah, that they, they always had to push on it... All of a sudden, they turned 17 and the kids just decided to do it on their own. So there does seem to be an element of just sort of sticking with it, even through failure, you know, through hard times through not seeing body weight change, but just continuing to pay attention to it over time.

#### *Accounting for Social Sources of Uncertainty*

Therein lies a final source of uncertainty for many practitioners working with patients and families: clinicians recognize that they play a minor part in the broader context of patients' lives, and the many non-clinical reasons that help explain BMI trajectories. All respondents acknowledged the importance of non-clinical, social and structural factors as a key missing piece of information in their diagnostic assessments and resulting recommendations. As Erin explained, the physician's role is to offer treatment and advice based on a diagnosis, but the major unknown in these encounters is whether “everything in [a patient's] life being in a position for them to act on it.” Tina had the view that “this world just sets a lot of people up to be obese, just by living.” This made it hard for her to view obesity and her role in addressing it through a purely medical lens, explaining that “in medicine, we categorize everything based on a billing code, [which] are all clumped under something like the word disease when it's really much more complicated than that.” Elliott most directly addressed the substantial *social*

uncertainty in treating patients with obesity, reflecting on the challenges of understanding enough about patients to provide meaningful help:

I think for me, the biggest thing is this is a clinical issue. And we sort of think about it medically... but it's kind of amplified and worsened and locked in by a lot of things that are just not medical. They're sort of out of out of the purview, and even out of line of sight of a clinician. And I think just the idea that I, as a clinician, can manage somebody's weight... in brief visits every several months, without a full view of their social context, the, you know, inner workings of their emotional life, their financial wellbeing... There's a lot that goes into that. And so, I do feel like we're often flying blind, which is why, you know, we tend to cling so hard to like the number and specific things that we can like, just point to. But I tend to believe that for the vast majority of folks that we engage clinically, around this, that we just know the tip of their particular story, right? We don't know the full thing. And so when they come back, you know - IF they come back - and they've succeeded, or, you know, quote, unquote, or they haven't made progress, we're still blind, right? We really don't know the reality of what's going on.

Elliott tied this uncertainty back to his original concerns that dealing with weight is often an issue of patients not having “confidence”; however, he now explained that it’s not necessarily an issue of confidence “in themselves, but in their circumstances.” These social aspects are hard to integrate into a medical, diagnostic lens, given that when he is defining the “problem statement” at the end of the visit, he realizes that “Oh, they're like, super poor... and there's no quick response to that.”

Many practitioners were quick to emphasize these social and structural factors when explaining the difficulty of responding to childhood obesity using traditional diagnostic protocols. Patricia explained that the standard protocol for diagnosis is to “talk to people about food intake and exercise... and then from there [it’s] like ‘Best of luck!’” in terms of knowing how the patients will act on this information. She knows there is much more that can be done for patients, in understanding issues of food insecurity and finding affordable and safe options for exercise, but “the problem is, this type of stuff to do the job... doesn’t occur in the 20-minute visit.” In an ideal world, practitioners could “prescribe food and activity” (Sandra), but this is not a right granted to health professionals. Adele commented on how “as with anything in medicine, I think like higher you raise your level of training, or rise in the ranks, you realize your own limitations, and I think it gets more and more frustrating that... there’s not that much in and individual moment you can do about it.” Indeed, many practitioners invoked income inequality, racism, neighborhoods, and the massive multibillion-dollar food industry as seemingly insurmountable obstacles

to both their and patient's success. However, Kathryn noted that even if clinicians are not able to directly intervene on these social determinants, "our roles as doctors have changed over the past several years" such that "we're sticking our nose in, we're investigating more aspects of our family's life, rather than just looking at their blood pressure or some number on a piece of paper." In turn, she found that "families are pretty receptive to it" and have "kind of accepted that this is the doctor's role now" which involves "looking at the family more as a whole rather than just focusing in on the health problems."

This broadening of clinicians' responsibility and scope of practice to encompass non-clinical sources of uncertainty was reflected in respondents' approaches to communicating with patients and families in a way that was sensitive to social influences. Practitioners realize they cannot use the same diagnostic protocol and apply the same standards of success to all individuals, cognizant of how their goals – and ability to attain them – is variable. Joseph explained how "no two families have the same situation," with completely different barriers that he needs to be aware of. On the one hand, "for some families, they truly live in a place where they're not gonna be able to get healthy groceries... or to have a place where they can let their kids play outside without fear of, you know, gunshots and stuff." On the other hand, even for a family that is "socioeconomically kind of in the dreamland," the parents might both be "super busy career people" and not have time to be active with kids "because there is too much going on from work pressures and whatnot." For Joseph, "the trick is to get on the same page" in figuring out what the specific barriers are. Kathryn echoed these concerns in explaining that "that you've definitely got to kind of assess the social situation a little bit more," and then "kind of tailor your suggestions to that." She also reflected on how diagnosis and treatment vary based on socioeconomic position, saying that "if I've got a more affluent family, who eat out all the time, because they can and it's not a resource problem, then we might make some suggestions from like, from the restaurants that they go, like let's look at the menus, let's look at the healthiest things they can pick; whereas I'm not going to do that for somebody who can't afford it."

Practitioners explained that this tailored diagnosis and treatment is important in talking and thinking about weight in a way that recognizes "the factors that affect [them] are not often in the control

of the person that needs to make the change” (Erin). Cassandra was explicit that “I don’t think [practitioners] should advise someone to do something unless they are really having some idea of the social context for this advice” and how it will be received. A lot of what people are doing is “a matter of habit and convenience and just like what they’re used to, or what they’re able to afford, or the food options available” (Sandra). Thus, the concern is creating a situation where following a diagnostic and treatment protocol centered exclusively on ‘fixing’ patients’ weight and health is counterproductive and results in a breakdown of the patient-provider relationship. To this end, Kathryn commiserated:

I mean, how do I fix somebody's diet. Like, they can't afford to buy healthy food... I can't fix that. Specifically, I can give suggestions. I can give some low-cost healthy recipes and things like that. But I can totally see a family's struggle, and it must feel pretty bad for them – me telling them these are the things you need to do for your kids’ health. And they just can't do it for whatever reasons. I mean, that sucks. Everybody wants to have their kids be healthier... Knowing what you need to do, but just not being able to do it – it’s gonna feel pretty bad for the family.

Ultimately, the difficulty with childhood obesity is the fact that the diagnostic, prescriptive model of health often used in medicine does not lead to successful outcomes. Frederick reflected on his career-long evolution in thought on childhood obesity in clinical settings, noting that he used to be far “more apt to be prescriptive of certain things the patient needed to do, because I’m the doctor and I’m supposed to tell you what you need to do.” Having seen this strategy fail on numerous occasions, he adheres to a more passive model of care, where the doctor’s authoritative, diagnostic role is less apparent. In maintaining positivity and preserving the patient-provider relationship, he is comfortable acknowledging uncertainty with patients, in seeing himself as “more of a partner, more of an advocate, who is here to try to be encouraging and to not promise more than I can about what’s possible.”

## **Discussion and Conclusion**

Building on extant research on how uncertainty is managed in clinical settings (Berg 1997; Macintosh and Armstrong 2020) – and the utility of diagnoses as a source of certainty (Jutel 2014) – this study analyzed interview data from pediatricians and other health professionals working with young patients to understand the unique challenges of diagnosing and treating obesity in early life. As the results show, clinicians interacting with pediatric patients and their families work within the constraints of a

healthcare system that demands formal diagnoses and codes to help “put a name to” specific issues. Yet there is uncertainty about the clinical utility of these diagnoses and labels, as well as concern about their potential to undermine the provider-patient relationship.

Thus, returning to the dilemma posed at the onset of this paper, how do practitioners make sense of imperfect and imprecise diagnostic tools, evidence, and criteria in addressing issues of weight, health, and overall wellbeing in early life? Though I do not claim to fully resolve this complex question, I use the findings to show how uncertainty is integrated *into* diagnosis and treatment and used to challenge medical standards and protocols. Clinicians acknowledge and capitalize on the uncertainty in the diagnosis of obesity to facilitate a better relationship with their patients and families. Moreover, uncertainty is an *integral* part of the diagnostic process, as it circumvents the needs for formal medical labels or language and offers pathways to treatment that reflect the needs and abilities of individual patients and families rather than adhering to “one-size-fits-all” solutions. By situating their young patients – and their BMIs – on a trajectory, clinicians counteract the biomedicalized framing of obesity as a sign that something is wrong and needs to be fixed in favor of a more socially-attuned view of weight and health as malleable processes that unfold over a lifetime.

Specifically, the interviews demonstrate a consistent emphasis on framing childhood obesity in terms of prognosis rather than diagnosis. Emulating the prognostic framework (Croft et al. 2015; Timmermans and Stivers 2018), practitioners use children’s BMI trajectories as a diagnostic tool rather than focusing solely on their status at the time of the clinical encounter. Clinicians’ uncertainty about BMI as a measure of health is used to justify this approach. Patients’ BMIs are suggestive rather than declarative about their health at present and where it may be headed, which makes for an ineffective and unsatisfying “diagnosis,” to the extent that the label is used to provide clarity about a patient’s health (Jutel 2019). Thus, unlike the dominant conceptualization of diagnosis and disease in medical sociology, the clinicians in this sample actively choose to *not* “put a name to” obesity as a diagnosis to impose certainty and delineate good from bad health. What a diagnosis means in this context is difficult to

discern; clinicians still use *a* diagnosis of obesity for bureaucratic purposes, but this diagnosis is not verbalized because there may not be anything to formally diagnose beyond noting the child's BMI.

Extant theory on the sociology of diagnosis emphasizes its role as an explicit, disruptive label, demonstrating how the “diagnostic moment” serves as a point of cleavage in an individual's social and health history and identity (Heritage and Macarthur 2019; Jutel 2014; Jutel 2019). However, this theory does not articulate a clear case for how we categorize diagnoses that are not shared with patients and thus do not provide this clear pre- and post-diagnosis moment of clarity intended to facilitate treatment and a path to wellness. The legitimization of obesity by way of diagnosis is the primary motivation for labeling it as a disease (Allison et al. 2008); yet the clinicians avoided the label, cognizant of how the meanings that obesity has outside of a clinical setting are not conducive to fruitful clinical interactions and care. Past medical sociological research on health and illness in early life emphasizes the concept of biographical disruption (Bury 1982), based on how children and young adults react and respond to being diagnosed with different conditions (Bray et al. 2014; Monaghan and Gabe 2015; Polidano et al. 2020). However, these theories are less applicable to a more fluid and less binary condition like childhood obesity, which is not treated as a formal diagnosis in clinical settings.

Rather than treat this as an aberrant finding, I argue that the diagnostic process underlying childhood obesity provides novel insight on how a diagnosis can be continuously and smoothly integrated into an individual's health narrative and trajectory. In avoiding the diagnostic moment, practitioners are no longer acting exclusively in response to the diagnostic label of obesity and the kinds of clinical guidelines and protocols designed to treat it. They feel free to talk *around* weight and obesity by focusing on the gradual, organic adoption of certain behaviors and lifestyles that promote better long-term health. The practitioners in this study consistently express a desire to engage in preventative, rather than prescriptive, care focused on future outcomes; in turn, they avoid direct emphasis on short-term weight loss as the only treatment befitting a diagnosis of obesity given that, medically-speaking, this is the only way to ‘undo’ a diagnosis based on BMI.

Beyond the implications for medical sociological theories of diagnosis, integrating uncertainty into the diagnostic process proves consequential for the kinds of relationships and interactions providers have with patients and families. Trajectories are not only a diagnostic tool in the context of childhood obesity; they also represent a diagnostic mentality that influences the nature of treatment and how practitioners and patients define success. Social diagnostic theory emphasizes the importance of diagnosis as a negotiation among the different stakeholders shaping the diagnostic process (Brown et al. 2011) – a sentiment which is echoed in the conceptual framework underlying prognosis as a more comprehensive model of care (Croft et al. 2015). This negotiation – or “meeting patients where they are,” as mentioned by many respondents – proves central to childhood obesity medicine, where clinicians are dependent on patients’ and families’ “buy in” to see some behavioral or lifestyle change. Treating childhood obesity is not a linear process, and clinicians acknowledge normal patterns of ebb and flow in their interactions with patients regarding their weight and weight-related behaviors. There is no guarantee, or certainty, that weight will be the focal topic of a given visit; patients may present with other health issues needing more immediate response or patients and families are not in an emotional or cognitive state to discuss weight.

Indeed, these findings suggest that the role of emotion in the diagnostic process merits greater attention. Provider’s and patient’s “affect” – with respect to their emotional response during clinical encounters – is often framed as a source of unwanted bias (Kozlowski et al. 2017; NASEM 2015). Moreover, the underlying premise of a biomedical definition of diagnosis emphasizes rationality and certainty, seeking to minimize subjective sources of influence on clinical decision making (Marcum 2013). Yet, patient’s emotional states readily influence childhood obesity diagnosis and discourse. Clinicians are not only evaluating patient’s weight and health, but also their emotional and cognitive capacity to instigate behavioral change, as an independent and valuable source of diagnostic information. As noted in the interviews, getting patients and families to a state where they can *begin* further discussion of weight and lifestyle changes is often more important than specific goals related to diet and physical activity.



Relatedly, the acknowledgement of uncertainty in a patient's trajectory of weight and health encourages providers and patients to arrive at mutual definitions of success that are not solely dictated by diagnostic criteria, such as dropping below a specific BMI. Many of the practitioners were comfortable with measuring success in more qualitative terms, based on how patients and families are feeling or what they are doing, even if the changes appear insignificant. While practitioners note the difficulty of exclusively relying on qualitative metrics in a field that values objective and numerical evidence, maintaining a long-term view of patient's weight and health makes this uncertainty more tolerable. Namely, the lack of obvious signs of progress does not mean progress is not occurring. Unlike many health conditions diagnosed and treated in clinical settings, the timeframe for childhood obesity is fraught with uncertainty – if not stochasticity – in terms of when and why a child or adolescent may internalize certain weight-related beliefs and behaviors and attain a healthier BMI. This more *passive* approach to diagnosis and treatment speaks positively to a sociology of “doing nothing” (Scott 2018), which represents a plausible clinical approach amid ongoing concerns about overdiagnosis and overtreatment in the medical field (Armstrong 2021; Croft et al. 2015). This should not be interpreted as practitioners choosing to *do nothing* for their patients and families; rather, there is recognition that patience and vigilance is an appropriate course of action based on the limited knowledge on hand.

Finally, these results provide empirical support for the theory that greater acceptance of diagnostic and clinical uncertainty leads practitioners to better engage with the wide range of social and non-medical factors shaping clinical encounters and patients' health (Brown et al. 2011; Croft et al. 2015). Though medicine, as a field, has elevated concern for social determinants of health over the past few decades, integrating these social factors into the practice of care remains a challenge (Metzl and Hansen 2014). The interviews demonstrate high levels of structural competency on the part of practitioners in recognizing that patients' and families' trajectories are shaped by their social environment. Indeed, there is considerable frustration – if not futility – among practitioners in reflecting on their individual ability to provide meaningful care that counteracts these structural forces. Recognition of these social sources of uncertainty in diagnosis speaks to the broader issue of treating patients and

families as unique cases, rather than situating them within established clinical and diagnostic standards and guidelines. Practitioners acknowledge structural explanations for how and why certain patients and families do better than others. In turn, their communication with and expectations for these patients and families is contingent upon how ready and able the patients and families are to deal with weight and health at a given point in time.

### *Limitations*

Prior to concluding with a discussion of how integrating uncertainty in diagnosis and treatment can inform future research on childhood obesity, I note the limitations of this analysis. First and foremost, the interview data come from a purposeful sample of childhood obesity practitioners whose views on weight and health may not be representative of the full spectrum of approaches among health professionals working with young populations. The relationship between weight and health is a pervasive issue in medicine, which many practitioners encounter on a regular basis: the challenge in this study was identifying respondents for whom childhood obesity is a key area of interest, but not the only health issue they encounter among patients. Consequently, most respondents were general pediatricians and medical students and residents interested in pediatrics. A larger sample could facilitate more in-depth comparisons of how childhood obesity is approached across different clinical subfields and specialty areas, such as endocrinology, gastroenterology, and rheumatology, where the role of obesity may vary based on both practitioners' and patients' concerns. Though I did not observe much variation based on providers' background in this sample, it is worth noting that family medicine clinicians – who also deal with adult patients – tended to be more comfortable with “blunt” assessments of individuals' weight and health, and more focused on weight loss. Likewise, medical students on surgical rotations noticed less apprehension about using BMI as a diagnostic tool among surgeons. Further exploring these sub-disciplinary differences could help uncover variation in diagnostic reasoning.

Ideally, the practitioners' perspectives in this study would be complemented by patients' and family's views to better illustrate the interpretation of diagnoses by all parties involved. An in-depth examination of the “social diagnosis” framework requires input from the multiple, competing

stakeholders shaping diagnosis as a process (Brown et al. 2011). I discuss how practitioners *believe* they arrive at mutual understandings of weight and health with their patients and families; yet, this may differ from patients' and families' interpretations of these same encounters and conversations, as noted in past literature (Lutz 2019). Cognizant of these limitations, this study limits itself to examining other aspects of social diagnosis, such as the emphasis on *potentiality* and patients' future health and the structural sources of uncertainty that influence this diagnostic trajectory. The focus on practitioners – and openness to unexpected findings encouraged by abductive analysis (Timmermans and Tavory 2012) – allowed for a careful examination of growth curves/charts as both a diagnostic tool and diagnostic philosophy. I use these findings to suggest that uncertainty is openly acknowledged in clinical settings, as evidenced by a lack of clear diagnoses. However, directly observing clinical encounters could reveal important discrepancies in how practitioners recall their diagnostic strategy as compared to what they actually say and do in describing patients' weight and health.

Finally, the focus of the interviews on issues of diagnosis and uncertainty did not allow for a more comprehensive examination of how these topics intersect with patients' and families' identities and backgrounds with respect to gender, race and ethnicity, and socioeconomic status, among others. While respondents were well-aware of structural factors influencing patients' trajectories and how that influenced the success of one patient as compared to another, this is an important topic that would require more focused questions geared towards understanding issues of social class and power dynamics in the context of clinical encounters. Likewise, many respondents understood the gendered nature of body weight and body image, and the need to tread more carefully when discussing these sensitive topics with girls versus boys, as well as how terms like “overweight” or “unhealthy” weight have very different cultural connotations. Unfortunately, the fairly limited time for interviews led to some inconsistency in how often these topics came up across respondents, and thus I did not feel it was appropriate to extrapolate from these conversations. It is interesting to note that a number of clinicians did not appear to be comfortable discussing about some of these topics – at least to the extent that they were explicit about changing their approach to diagnosis and treatment based on patients' and families' backgrounds.

However, this limitation uncovers an important – and heretofore unaddressed – question in medical sociology about the influence of structural factors and social identities on clinicians’ management of uncertainty during diagnosis. Namely, how do clinicians’ perceptions of uncertainty about what body weight means for a child’s overall health and wellbeing – as well as their beliefs about how stigmatizing a conversation about body weight and health might be – change as a function of a patient’s background or identity? One can imagine that this question is further complicated by clinicians’ own background and identity. Critically, it is important to investigate whether and how these perceptions and beliefs affect patient outcomes and potentially shape population-level trends and disparities.

### *Conclusion*

Uncertainty in the diagnosis and treatment of childhood obesity stems from the fact that health is a complex and multidimensional construct, and that medicine is rarely able to address a single dimension like body size without implicating many other aspects of individuals’ health and wellbeing. In keeping with Timmermans and Haas’s (2008) call for a sociology of disease – wherein distinct diseases, or conditions, serve as the units of sociological inquiry and analysis – this study examines the diagnosis of childhood obesity to better understand how practitioners evaluate body size as both a medical and social construct in the lives of their patients and families. The results document numerous challenges and sources of uncertainty in providing care. Clinicians are often tracking multiple metrics and aspects of physical, mental, and social health, which are not equally applicable to all individuals and operate on different time scales and trajectories; moreover, these trajectories are rarely linear or predictable. Patients’ BMIs are a central measure of progress – and the defining attribute of obesity as a diagnosis – but they can be a poor indicator of how patients and families are feeling and what they are doing to try and succeed.

Despite the concern that the researchers and practitioners studying and diagnosing childhood obesity are perpetuating simplistic and harmful narratives about children and adolescents as ‘unhealthy’ and ‘diseased’ (Greenhalgh 2015; Moffat 2010; Saguy 2012; Shugart 2016), this study suggests that many clinicians do not view obesity through this binary lens, and instead practice a form of medicine that

comports with a more holistic understanding of health as more than the absence of disease or infirmity (VanderWeele 2017; VanderWeele et al. 2019). Practitioners working with youth encounter a variety of health issues and ailments; in many cases, they see entirely healthy patients with no concerns – as one would hope for children and adolescents. In this context, having too narrow a view or focus on weight is a liability, potentially leading them to overlook important information or cause harm by fixating on an attribute that is closely interlinked with many other domains of health and wellbeing.

Maintaining this holistic view presents a challenge at the population level, where the focus is on broad interventions and solutions that have an impact at the aggregate level but may have no direct bearing on discrete individuals (Rose 1985). This tension is at the crux of the debate over obesity as a diagnosis and disease, wherein population-level narratives about weight and health often take precedence over the individual experiences and needs of people categorized by this medical label (Shugart 2016). Clinicians are actively “doing diagnosis” in this space between population and individual level health, making decisions about how much certainty to accord to aggregate-level medical knowledge when faced with conflicting and uncertain information about a given patient (Timmermans and Angell 2001).

More importantly, population-level decisions about how we define and measure health – and what constitutes or counts as progress towards *becoming* healthier – are not always compatible with individual-level diagnoses and treatment. This is evident in the uncertainty and difficulty of reaching conclusive assessments about young patients based on a single data point. BMI provides a snapshot of a broader trajectory of health; measuring this trajectory is more informative, but it only reflects a single trajectory describing patients’ health, and only one way of tracking progress. Clinicians thus try to bring in additional information to form a more complete picture, which may complicate the otherwise ‘simple’ narrative provided by BMI alone. This study reveals myriad emotional, cognitive, and behavioral aspects of the diagnostic and treatment process that function independently of patients’ weight, but which are often more consequential for patients’ health. The discordance between these other outcomes and BMI suggests that population-level knowledge about childhood obesity as a condition *inclusive* of physical,

mental, and social health is incomplete; thus, the diagnosis of obesity does not provide the clinical certainty desired by practitioners or patients.

This disconnect underscores the importance of recognizing the reciprocal relationship between population- and individual-level health research and theory. The dominance of the evidence-based movement in medicine, and the influence of epidemiologic notions of screening and risk on clinical practice, demonstrate how population-based findings influence how practitioners view their patients, and the kinds of decisions and actions they take as a result (Armstrong 1995; Armstrong 2012; Armstrong and Eborall 2012). However, this downstream flow of scientific evidence and knowledge that creates and categorizes health can be reversed (Hacking 2007): the individual-level insights gained during clinical encounters – like the many non-diagnostic metrics used to assess patients and gauge progress in the treatment of childhood obesity – are an equally valuable source of evidence and knowledge that can diffuse outward and shape the decisions guiding definitions and measurement at the population level.

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## **CHAPTER 3: POPULATION HEALTH AT EVERY SIZE?: BODY SIZE, CARDIOMETABOLIC RISK, AND EDUCATIONAL DISPARITIES IN MORTALITY AMONG U.S. ADULTS, 1988-2015**

### **Introduction**

The growing proportion of adults classified as “obese” (in reference to a body mass index [BMI] exceeding BMI, per clinical definitions, rather than a qualitative descriptor) is a legitimate concern for researchers and policymakers seeking to improve population health in the United States. Reviews of large-scale epidemiologic studies consistently find that obesity is associated with elevated risk for many heart conditions, diabetes, cancer, and kidney disease (National Heart, Lung, and Blood Institute [NHLBI] 2013), as well as various other cardiometabolic conditions that account for the leading causes of mortality (Ahmad and Anderson 2021). In turn, the fact that over four-in-ten U.S. adults are now considered obese (Hales et al. 2020) – up from 30% only 20 years ago, and 14% in the early 1960s (Fryar et al. 2012) – has led to increasingly dire predictions about worsening health and declining life expectancy in the decades to come (National Academies of Sciences, Engineering, and Medicine [NASEM] 2021; Preston et al. 2018; Stokes and Preston 2016).

At the same time, some scholars contend that this pessimism is misplaced, if not harmful, in perpetuating the belief that individuals’ body size is an accurate reflection of individuals’ underlying health and wellbeing (Saguy 2012; Shugart 2016). Decisions about how we conceptualize, define, measure, and then label health are consequential; many scholars note that obesity represents a crude categorization of individuals’ health status (Jutel 2009; Jutel 2011), whose issues are magnified by BMI being an imprecise measure of obesity as a state of excess adiposity (Burkhauser and Cawley 2008; Müller et al. 2016). Recent evidence suggests that many adults classified as obese are ‘misclassified’ as unhealthy, as they do not have a cardiometabolic health profile consistent with elevated chronic disease

risk; in fact, they are cardiometabolically healthier than many of their lower weight counterparts (Tomiyaama et al. 2016; Wildman et al. 2008). Thus, with some estimates suggesting that over half of U.S. adults will be considered obese by the end of this decade (Finkelstein et al. 2012), more careful scrutiny of heterogeneity in a health condition that may soon categorize the *majority* of the U.S. population seems appropriate.

More broadly, debates about the relationship between body size and health reflect the challenges of operationalizing health at the population level. Individuals' health is rarely defined by a singular ailment, and researchers have called for a more *systems-wide* approach to conceptualization and measurement that better accounts for the complexity and nuances of health, and the presence of *multiple* morbidities (Ahn et al. 2006; Barnett et al. 2012; Boyd and Kent 2014; Guthrie et al. 2012; Seeman et al. 2004). Indeed, current discourse on obesity assumes a relatively uniform distribution of risk at the population level, ignoring differing levels of severity (Sharma and Kushner 2009), and obscuring the fact that excess weight tends to be assumed as harmful even when co-occurring with more severe morbidities (Sharma and Campbell-Scherer 2017).

National Heart, Lung, and Blood Institute guidelines for clinical practice already recommend that other risk factors are taken into consideration when evaluating obesity (Jensen et al. 2014). Fortunately, large-scale biomarker collection in nationally-representative surveys has facilitated the study of individuals as health *systems* rather than a collection of individual *symptoms* or risk factors. These data have been used to substantiate the presence of biological “phenotypes” of metabolically-healthy obesity (MHO) – as well as metabolically-unhealthy individuals *without* obesity – which have been theorized since the onset of the obesity epidemic in the United States (Ruderman et al. 1981). This research finds that individuals' BMIs and the label of obesity are not definitive measures of their underlying health, as approximately one-third of adults with obesity do not exhibit cardiometabolic impairment (Smith et al. 2019).

Yet, in focusing on MHO as a *biological* phenotype and construct, researchers often neglect to consider how heterogeneity in body size and health is a function of social mechanisms, with MHO or

similar constructs representing *social* phenotypes as well. For example, socioeconomic disparities in obesity and cardiometabolic health are well-documented (Braveman et al. 2010; Krueger and Reither 2015; Pampel et al. 2010), and often singled out as a key contributor to educational gradients in mortality and life expectancy in the United States (Cutler et al. 2011; Elo 2009; Vierboom 2017). To the extent that various combinations of these measures – such as metabolic syndrome and allostatic load – are socially-patterned on the basis of education (Dowd et al. 2009; Loucks et al. 2007; Montez et al. 2016; Richardson et al. 2021; Seeman et al. 2010), we would anticipate that different profiles of body size and cardiometabolic health are not exclusively biologically-determined, and thus not randomly distributed throughout the population.

The goal of this chapter is to assess the clustering, or co-occurrence, of obesity with other measures of adiposity and relevant cardiometabolic health risks and provide a better understanding of variation in body size and health at the population level. Rather than describe obesity and its threat to future health in monolithic terms, this study helps illustrate the importance of allowing for heterogeneity in how we think about and assess obesity and its risks for population health. Critically, extant research on education as a fundamental determinant of health – and key correlate of both obesity and cardiometabolic risk – provides a useful entrée for illustrating how a systems-wide, multimorbidity approach can provide insights on health disparities and educational gradients in mortality. Thus, rather than solely identifying groupings of body size and cardiometabolic risk, we can also examine their social patterning and estimate their contribution to educational disparities in premature mortality.

The chapter begins by providing needed context on cardiometabolic health in the U.S. and the role of obesity, as well as reviewing extant research on heterogeneity in obesity and cardiometabolic risk and what past work suggests about educational attainment as a determinant of this heterogeneity. Using data from the National Health and Nutrition Examination Survey-Linked Mortality Files from 1988 through 2015, I ask three key questions emergent from this literature. First, *which body size and cardiometabolic health profiles best characterize U.S. adult population over the past decades?* Past approaches tend to follow an *a priori* conceptualization of individuals as having or not having obesity and



having or not having elevated cardiometabolic risk – or just focus on MHO – without considering alternate possibilities, especially in using a more expansive set of comorbidities. Second, *what is the association between these profiles and premature adult mortality risk in the United States?* Though one may infer whether a given profile represents good or bad health based on the distribution of various comorbidities, *validating* these profiles based on their association with mortality risk allows for a more definitive assessment of their association with future health. Finally, *how does the social patterning of these profiles explain educational gradients in mortality risk?* These profiles of body size are likely to be unevenly distributed based on individuals' educational attainment; knowledge of this broader spectrum of risk can identify which profiles are most consequential in accounting for higher mortality risk among adults with less education.

## **Background**

### *Cardiometabolic Health in the United States and the Role of Obesity*

Despite recent downturns in U.S. life expectancy associated with opioid-related deaths and the COVID-19 pandemic, cardiometabolic dysfunction and disease has been the driving force behind improving trends in morbidity and mortality over the past 60 years (Ma et al. 2015). The broad spectrum of conditions associated with impaired cardiovascular and metabolic health account for the overwhelming majority of chronic diseases and leading causes of death in the U.S (National Center for Health Statistics [NCHS] 2021). While past decades have seen progress in declining rates of diabetes and cardiovascular disease-related mortality (Gregg et al. 2018; Koton et al. 2014; Ma et al. 2015; Mensah et al. 2017), as well as declining – or at least, non-increasing – prevalence of cardiovascular conditions like heart disease and stroke (Ford et al. 2014), the burden of poor cardiometabolic health remains considerable and may increase in the coming years (Masters et al. 2018; NASEM 2021).

Over 30% of U.S. adults age 20 and above have hypertension (Fryar et al. 2017), approximately 27% have some form of hypercholesterolemia (NCHS 2018), and over 26% of men and approximately 9% of women have low levels of “good” high-density lipoprotein (Carroll and Fryar 2020), with increasing prevalence by age. Diabetes prevalence has seen a sharp increase in past decades as well;

whereas diabetes prevalence was under 1% in 1958 (Centers for Disease Control [CDC] 2017a), the current rate is over 9%, accounting for 30 million U.S. adults (CDC 2017b). Combined with an additional 84 million considered “prediabetic”, over one-third of adults exhibit signs of impaired glucose regulation (CDC 2017b). Moreover, the prevalence of metabolic syndrome – a medical term for the clustering of cardiometabolic risk factors – is at an all-time high; anywhere from one-in-four to one-in-three U.S. adults present with *multiple* cardiometabolic morbidities, potentially indicative of an overall state of physiological dysregulation (Aguilar et al. 2015; Beltrán-Sánchez et al. 2013; Moore et al. 2017).

Poor cardiometabolic health is seen as the consequence of the many unhealthy behaviors and lifestyles shaping contemporary society, which have replaced infectious disease, violence, and accidents as the major threats to population health (Olshansky and Ault 1986). In keeping with this perspective, obesity – or an “unhealthy” body size, more broadly – is typically the *key* factor implicated in the high prevalence of poor cardiometabolic health in the United States (Olshansky et al. 2005; Preston et al. 2014; Preston et al. 2018). This conclusion is not entirely surprising given the strong associations between BMI and many cardiometabolic diseases and associated causes of death (Mokdad et al. 2003; Sowers et al. 2003; Steele et al. 2017; Van Gaal et al. 2006). Moreover, obesity and multiple cardiometabolic risk factors have seen parallel growth over past decades; while not indicative of causal associations, the correlation is concerning in its implications for the future of population health in the United States.

Yet the overwhelming emphasis on body size as the singular determinant of poor cardiometabolic health has been challenged by recent efforts to promote a more holistic evaluation of health, recognizing *multiple* sources of risk and treating individuals as “systems” of comorbidities (Barnett et al. 2014; Bierman and Tinetti 2016; Salisbury 2012). For instance, a number of medical researchers and practitioners actively advocate against the conflation of individuals’ body size and their cardiometabolic health (Guo et al. 2014), recognizing that many adults who have obesity from a clinical perspective are not sick or diseased. This argument is rooted in the fact that “cardiometabolic risk factors confer much higher risk of diabetes, cardiovascular disease, stroke, and mortality than obesity *per se*” and a large proportion of obese adults are “devoid of metabolic syndrome risk factors” and thus “at markedly reduced

risk of cardiometabolic disease” (Guo and Garvey 2016: 524). Even the American Association of Clinical Endocrinologists and the American College of Endocrinology – which recognize obesity as a disease – emphasize that the binary classification of BMI as obese versus not obese is incommensurate with medical knowledge about the considerable heterogeneity in how it is observed throughout the population (Garvey et al. 2014). Specifically, they propose a more “medically-meaningful” diagnostic strategy that better recognizes variation and severity in “pathogenesis of obesity as a chronic disease”, allowing for greater “effectiveness of public health initiatives” (Garvey et al. 2014: 980).

### *Health at Every Size*

Critically, most researchers and scholars expressing skepticism about obesity do not deny that there is a point at which body weight poses an issue; rather, the lack of a clear universal and biological threshold underscores the uncertainty in categorizing obesity as a state of poor health. For instance, almost a *quarter* of U.S. adults ages 30-74 in 2017-2018 fall within *two BMI points* of the clinical cutoff for obesity (based on author’s calculations), which is an approximately 13-pound range for an average adult. This not an insignificant amount of weight to gain or lose; but it is also not an immediately clear criteria by which to assess a meaningful change in health. Such narrow constraints on how we define health have significant implications, as millions of Americans are perpetually on the border between “good” and “bad” weight. In turn, relatively minor tweaks to BMI guidelines have substantial repercussions for how physicians, insurers, and the public at large perceive their health (Flegal 2010; Jutel 2011; Kuczmarski 2007; Kuczmarski and Flegal 2000; Nicholls 2013).

Recognizing these limitations, and better acknowledging diversity in weight and health, the central goals of the Health At Every Size movement (HAES), and similar initiatives, strive to promote good health rather than healthy weight (Bombak 2014; Bombak et al. 2019; Miller 2005; Penney and Kirk 2015; Robison 2005; Tylka et al. 2014). There is a growing body of evidence to suggest that weight-targeted interventions are often ineffective and unsustainable, emphasizing dietary and exercise regimes for which the only metric of success is a purely *quantitative* reduction in weight (Bacon and Aphramor 2011; Kraschnewski et al. 2010; Mann et al. 2007). Many adults successfully and sustainably improve

many other cardiometabolic indicators that allow for better overall health and longevity (Bacon and Aphramor 2011; Mann et al. 2007; Tylka et al. 2014), suggesting that public health efforts may benefit from transitioning away from a “one size fits all” approach that treats weight loss as a panacea (Phillips 2013). The discordance between individuals’ having an “unhealthy” body weight despite otherwise “healthy” measures of cardiometabolic functioning introduces doubt and dissatisfaction as to the overall utility of a clinical encounter or medical evaluation (Greenhalgh 2015; Saguy 2012). In turn, researchers’ and clinicians’ focus on weight can bias them towards ignoring other important signs and symptoms of both good and bad health (Puhl and Heuer 2009; Puhl and Heuer 2010), leading to skewed assessments of individuals’ health which only become magnified at the population level.

Recent research sheds empirical light on these calls for a more holistic evaluation of weight and health, questioning the value of an exclusively body size-based measure of obesity as a measure of health. More detailed assessments of cardiometabolic health consistently find that a significant number of adults classified as obese are just as healthy, if not healthier, than their normal weight counterparts. Studies and meta-analyses of “metabolically-healthy obesity” (MHO) and “cardiometabolic risk clustering” among obese and non-obese adults finds that up to 40% of U.S. adults can be described as in *good* cardiometabolic health despite their having a BMI greater than 30 (Blüher 2020; Primeau et al. 2011; Stefan et al. 2008; Wildman et al. 2008). Likewise, many “normal” or “healthy” weight adults exceed one or more of the criteria for such risk factors as hypertension, hyperglycemia, and dyslipidemia, as well as more severe conditions like cardiovascular disease (CVD) and diabetes (Ahima and Lazar 2013; Aung et al. 2014). Granted, more long-term validation of the risk associated with MHO is mixed; some studies find that the relative risk of developing CVD, diabetes, and/or mortality (both all-cause and CVD-related) for metabolically-healthy obese adults is on-par or lower than their normal-weight counterparts (Appleton et al. 2013; Guo et al. 2014; Hamer and Stamakis 2012; Roberson et al. 2014), while others continue to find a small increased risk among otherwise healthy obese adults (Aung et al. 2014; Bell et al. 2014; Kramer et al. 2013; Kuk and Ardern 2009; Roberson et al. 2014). Critically, there appears to be no added risk associated with obesity when individuals already show signs of poor health (Kramer et al. 2013;

Roberson et al. 2014), reflecting how a narrow focus on obesity often ignores the large proportion of U.S. adults whose cardiometabolic profile portends higher risk of worse health and early death, even if their weight is considered “healthy” or “ideal.”

Consensus on these studies is difficult to achieve because definitions of “metabolic healthiness” vary (Hinnouho et al. 2013; Primeau et al. 2011; Magkos 2019; Roberson et al. 2014). Despite their demonstrating broad variation in cardiometabolic health co-occurring with obesity, most studies continue to rely on fairly strict – albeit relatively arbitrary – definitions of poor cardiometabolic health, such as exceeding a risk threshold for at least one or two risk factors, or *all* risk factors, or based on a summed risk factor score/index (Blüher 2020; Magkos 2019; Phillips 2013). Critically, these approaches implicitly assume that these diverse indicators of cardiometabolic impairment are equivalent in their impact on individuals’ health. Yet, research has found that decisions about which combinations of measures to include in the definition of both obesity and cardiometabolic health are consequential for subsequent estimates of risk for worse health or mortality (Durward et al. 2012; Hinnouho et al. 2013; Pataky et al. 2011), suggesting that subjectivity in how MHO is defined remains a key source of uncertainty in understanding the salience of this concept.

#### *Educational Attainment and Cardiometabolic Risk*

Research on MHO and similar concepts has been critical for broadening understanding of how body weight and cardiometabolic health co-occur in the population; however, the extent to which obesity is considered metabolically healthy or not is often framed as a biologically-preordained phenomenon that is randomly distributed throughout the population (Huang et al. 2017; Navarro et al. 2015; Telle-Hansen et al. 2013). Perhaps as a function of this research often being confined to medical and clinical settings, individuals’ “predisposition” for different phenotypes of obesity is described in terms of physiological, microbiotic, and genetic mechanisms underlying different body types, with additional considerations for the role of exercise and physical activity (Iacobini et al. 2019; Phillips 2013; Primeau et a. 2011). Yet, the role of larger social determinants underlying these processes cannot be ignored. While researchers are often careful to control for sociodemographic factors or note key correlates of MHO (Al-kaidi et al. 2019;

Wildman et al. 2008), to date there has been less attention to the social explanations for how these body size and health phenotypes are distributed throughout the population.

Decades of sociological and social demographic research consistently find that population heterogeneity in health is not random, as fundamental causes of health like individuals' socioeconomic status (SES) give rise to myriad beneficial resources and mechanisms that predispose highly-educated adults to have more favorable risk profiles (Link and Phelan 1995; Phelan et al. 2010). Though all educational groups have experienced increasing rates of obesity over past decades (Ljungvall and Zimmerman 2012), highly-educated adults consistently have the lowest rates of obesity in the U.S. population, with less than three-in-ten adults with a college degree classified as obese as compared to over four-in-ten adults with lower educational attainment (Ogden et al. 2017). The many advantages that highly-educated adults tend to have – such as more disposable income, greater leisure time, better availability of healthy foods, and a higher probability of living in neighborhoods and communities that more easily facilitate physical activity – are all key mechanisms linking educational attainment to the types of health behaviors and lifestyles that we associate with individuals' ability to maintain a 'normal' weight (Braveman et al. 2010; Krueger and Reither 2015; Pampel et al. 2010). Many of these same mechanisms underlie educational gradients in individual cardiometabolic risks and conditions like hypertension, hyperglycemia, and dyslipidemia (Kanjilal et al. 2006; Mensah et al. 2005; O'Rand and Lynch 2018). Recent evidence suggests that educational disparities across a broad array of cardiometabolic indicators emerge early in the life course, often many years before they present as clearly diagnosable conditions (Lawrence et al. 2018; Noppert et al. 2021).

Given the associations between educational attainment and body size and cardiometabolic health – and the aforementioned links between obesity and a broad range of cardiometabolic conditions – it is unsurprising that a number of studies find educational disparities in metabolic syndrome and allostatic load. Though these constructs reflect different conceptual models of how and why individuals' experience physiological decline, they are similar in using multiple measures of body size and cardiometabolic risks to present a more comprehensive profile of individuals' health. Both Loucks et al. (2007) and Montez et

al. (2016) find anywhere from 25-75% higher probability of metabolic syndrome among adults with a high school degree or less as compared to those with greater educational attainment. Likewise, Dowd et al. (2009) and Seeman et al. (2010) both note a consistent, graded relationship between lower education and higher allostatic load in their reviews of past literature – though Dowd et al. contend that the cardiometabolic components of the allostatic load measure appear to be the key source of disparities. Once again, emerging evidence on emerging cohorts of young adults suggests that educational disparities in both metabolic syndrome and allostatic load present in early adulthood (Kane et al. 2018; Richardson et al. 2021), despite these constructs traditionally being measures associated with ‘aging’ and chronic ‘wear-and-tear’ on the body.

Despite the persistence of educational disparities in obesity and cardiometabolic risk across multiple indicators and dimensions of health – and focus on these disparities as an explanation of socioeconomic disparities in life expectancy (Elo 2009) – relatively few researchers have explicitly examined their role as mediators for educational gradients in mortality in the United States (Cutler et al. 2011; Seeman et al. 2004; Vierboom 2017). Vierboom’s (2017) finding that approximately 10% of educational disparities in mortality risk are explained by differences in weight status is consistent with the minimal-to-modest contributions of individual cardiometabolic risk factors observed in non-U.S. data (Dégano et al. 2017; Dowd and Goldman 2006; Gleib et al. 2013; Kershaw et al. 2013). Critically, there has been less research on multifactorial constructs like allostatic load, despite recognition that a failure to account for “multi-systems” approach to studying health has been a limitation of past research (Seeman et al. 2004; Seeman et al. 2008), and acknowledgement that health risks typically cluster together (Kershaw et al. 2013). Seeman et al. (2004) find that over a third of educational disparities in mortality (based on more or less than a high school education) were explained by an allostatic load index; however, their sample consisted exclusively adults in the 70-79 age range. Moreover, results from non-U.S. samples yield mixed results. For instance, Kim et al. (2018) find that allostatic load mediated less than 7% of the relationship between socioeconomic status and mortality in a sample of Korean adults, while Gleib et al. (2013) observe ~20% mediation between education and general health in a sample of older Russian

adults. To my knowledge, there is no research examining the how the clustering of multiple cardiometabolic risks accounts for educational disparities in mortality for a more broadly representative sample of U.S. adults.

In summary, though the formal, clinical definition of obesity is entirely based on individuals' having a BMI of 30 or higher, recent evidence points to multiple “phenotypes” of obesity given heterogeneity in how body size co-occurs with other cardiometabolic risk factors of concern. Recognition of these phenotypes is critical in challenging the paradigmatic conceptualization of obesity as a binary state of health (Blundell et al. 2014), further emphasizing the importance of looking at a more expansive set of risk factors and biomarkers in helping to make assessments of how body size and cardiometabolic health are distributed throughout the population (Al-kaidi et al. 2019). However, past research and theory has largely focused on population heterogeneity in obesity as a function of biological and genetic mechanisms, failing to consider how a key social attribute like educational attainment structures individuals' abilities to have a ‘healthy’ body size or a ‘healthy’ cardiometabolic risk profile, or both, or neither.

Specifically, I examine broad sets of anthropometric and cardiometabolic measures used in this line of research to document probabilistic – rather than deterministic – groupings of body size and health in the US adult population. Rather than strictly rely on cross-sectional assumptions about the risk associated with these groupings, I examine their relationship with premature mortality risk across different causes of death to help validate this probabilistic approach and provide a substantive interpretation of what these groupings mean for long-term health. Finally, I examine how individuals' educational attainment intersects with these groupings in demonstrating the “social” – rather than biological – patterning of these phenotypes and how they help to explain educational disparities in adult mortality risk.

## **Data**

The data for these analyses come from the National Health and Nutrition Examination Survey (NHANES), a nationally-representative survey of U.S. adults combining extensive sociodemographic and



health questionnaires with clinically-assessed physiological and anthropometric measurements across a broad range of health outcomes (NCHS 2017). NHANES uses a stratified, multistage probability sample that is representative of the civilian noninstitutionalized U.S. population. Each survey participant is subject to a household interview with questions on their sociodemographic characteristics and multiple domains of health and health-related behaviors. Participants also undergo a comprehensive physical examination by trained health technicians in a mobile examination center, who collect data on multiple anthropometric and physiological metrics using standardized measuring procedures and equipment. Due to these rigorous data collection procedures, NHANES data are often considered the “gold standard” for assessing population health in the United States (Dillon et al. 2020).

Critical to this study, NHANES data have been merged with mortality data, pooling from a variety of databases such as the National Death Index and the Social Security Administration, with a high probability of successful matches (NCHS 2019). In order to maximize the number of cases for identifying variation in body size and cardiometabolic health, as well as to ensure a sufficient sample size for subsequent analyses of mortality risk, this project pools data from NHANES III (1988-1994) and continuous NHANES, collected biennially from 1999 through 2014. Due to concerns about respondents’ privacy in publicly-available data, information on cause of death is limited to ten broad categories through December 31st, 2015. This study focuses on all-cause mortality risk, but also presents results for causes of death where (1) hypertension or (2) diabetes is noted as a contributing cause, as well as (3) only heart disease-related deaths, and (4) a final fourth category of causes of death including heart disease, diabetes, and cancer.

The primary variables of interest – i.e., the measures of body size and cardiometabolic health that I use to identify the different “phenotypes” – consist of self-reported and physical measurements, reflecting both diagnosed and undiagnosed conditions. There are numerous biomarkers of health in the NHANES data; I focus on measures that are comparable to those used in past research on metabolic syndrome and allostatic load, or that are plausibly associated with body size, rather than including any and all measures of individuals’ health risk (e.g., heavy metal blood tests). Additionally, these measures

were chosen to ensure as much harmonization across the survey cycles as possible given that NHANES varies testing protocols and measures across years. Specifically, the measures fall into four broad categories:

- (1) Body size and history, including: the “standard” measure of obesity based on a BMI of 30.0 and higher; whether individuals ever were considered obese based on BMI calculated from their highest ever reported weight; whether individuals were classified as obese 10 years ago or at age 25, also calculated from retrospective reports of weight; whether their waist circumference to hip circumference exceeds gender-specific ratios associated with an unhealthy distribution of body fat (0.9 for males; 0.85 for females [World Health Organization 2011]); and whether the ratio of their waist circumference to height ratio exceeds 0.5, which has been proposed as a more generalizable measure than waist circumference on its own in suggesting increased risk for central adiposity (Ashwell and Gibson 2012; Baioumi 2019; Schneider et al. 2010). The inclusion of retrospective weight measures is a key addition to research on multimorbidity, as individuals’ BMI at time of survey provides a limited snapshot of their body size and does not allow researchers to understand individuals’ weight history. The latter point has been a key area of concern in recent years, with a number of studies suggesting that retrospective measures provide more accurate assessments of the population burden of obesity by correcting for biases due to illness-related weight loss among individuals who previously were obese (Stokes and Preston 2016).
- (2) Cardiovascular health, including: a resting pulse rate exceeding 100 beats per minute, as indicative of tachycardia (Mayo Clinic 2020); measured pre-hypertension, based on an elevated mean blood pressure reading at examination ( $\geq 120$  mm Hg for systolic or 80 for diastolic [Muntner et al. 2018]); and diagnosed hypertension based on individuals’ having been given a diagnosis of hypertension by a physician or currently using antihypertensive medication.

- (3) Dyslipidemia, including: measured high cholesterol, based on elevated total cholesterol at examination ( $\geq 200$  mg/dL [Davidson 2020]); diagnosed high cholesterol based on individuals' having been given a diagnosis of high cholesterol by a physician or currently using cholesterol-lowering medications; measured high triglycerides at examination ( $\geq 150$  mg/dL [Davidson 2020]); and measured high apolipoprotein B at examination ( $\geq 100$  mg/dL [Paredes et al. 2019]).
- (4) Hyperglycemia, including: measured hemoglobin A1c percentage (providing a ~three-month average of blood sugar based on what percentage of hemoglobin proteins in the blood are glycosylated, or coated with sugar) greater than or equal to 5.7%, indicative of a pre-diabetic state (Dansinger 2020); measured high blood glucose in serum or plasma ( $\geq 100$  mg/dL [fasting] or  $\geq 200$  mg/dL [non-fasting] [Khatri 2019]); measured high fasting insulin level ( $\geq 25$  mIU/L [Melmed et al. 2015]); and diagnosed diabetes based on individuals' having been given a diagnosis of diabetes by a physician or currently using antidiabetic medication.
- (5) Other relevant measures, including: measured high C-reactive protein at examination ( $\geq 3.0$  mg/L), indicative of a high-risk, elevated inflammatory state (Pearson et al. 2003); and evidence of albuminuria or kidney damage, defined as albumin-to-creatinine ratio in urine greater than or equal to 30 at examination (Mayo Clinic 2021).

NHANES provides continuous versions of the biomarker and anthropometric measures used in the analysis; however, I choose to dichotomize the indicators for two key reasons. The first is to maintain comparability between these analyses and extant work focusing on the co-occurrence of distinct “comorbidities” as measures of underlying health (i.e., the aforementioned literature on metabolic syndrome and allostatic load). Though scholars have noted that clinical cutoffs are often a function of arbitrary – and/or bureaucratic – decisions about how to operationalize health (Jutel 2011; Timmermans and Epstein 2010), they allow for easier interpretability of the various measures and what they imply about individuals' risk. Secondly, dichotomized categories are well-suited to this study's overarching goal of explicitly *challenging* core assumptions about obesity as a binary state, in demonstrating that the same

category of “unhealthy” body size corresponds with different levels of underlying risk. Using a binary conceptualization of health across these measures can also help to reveal the limitations of said categories for understanding heterogeneity in population health. Nevertheless, I consider alternate specifications and combinations of measures as part of the sensitivity analyses.

Educational attainment is the key non-health measure used in the analyses, as a general indicator of individuals’ SES. While there are many ways to operationalize SES, education is the most common measure in research on health because it typically precedes attained occupation or income (Elo 2009), and is less prone to reverse causation bias (Seeman et al. 2008). This is not to suggest that educational attainment is the most important social factor associated with obesity and cardiometabolic health; past studies consistently document important gender and racial/ethnic disparities in body size and various indicators of cardiometabolic dysregulation, as well as at the intersection of the two (Borrell et al. 2010; Geronimus et al. 2006; Geronimus et al. 2010; Hargrove 2018; Levine and Crimmins 2014). Rather, this study focuses on educational attainment in response to the mixed findings identified in past research on the role of obesity and cardiometabolic health as a mediator between SES and subsequent morbidity and mortality. I acknowledge that the meaning of educational attainment for health exhibits considerable variation on the basis of gender and race/ethnicity (Goldman et al. 2006; Kimbro et al. 2008; Ross et al. 2012; Ross and Mirowsky 2010), but these additional analyses are challenging with the more limited sample sizes in the NHANES Linked Mortality Files, as described later.

Educational attainment is categorized as less than a high school education, a high school degree or GED equivalent, some college education or an associate’s degree or equivalent, or a college education or greater. I also control for survey year, individuals’ age, gender, and race/ethnicity based on NHANES-defined categories (“White” [non-Hispanic], “Black” [non-Hispanic], “Mexican-American,” “Other”), nativity, income-to-needs ratio based on federal poverty thresholds adjusted for inflation and family size (0-0.99; 1.00-1.99; 2.00-3.99; 4.00+), smoking status (“Never,” “Former” [ever smoked 100 cigarettes, but currently does not], “Current”), and health insurance coverage. These controls were chosen because

they are measured relatively consistently across the NHANES years I used and they help address issues of confounding often neglected in research on adiposity and mortality (Stokes and Preston 2016).

The final analytic sample is limited to adults ages 30-74 at time of survey/examination, with the exclusion criteria being pregnant women, adults who did not participate in the clinical examination, and those who are not eligible for mortality follow-up because of very poor identifying information. Mortality is assessed through age 85 or the end of the calendar year of 2015. The sample includes 40,095 adults, with 7,106 deaths during the follow-up period. In sum, this represents 417,076 person-years, with an average follow-up duration of about 10.4 years.

## **Methods**

Using the above-mentioned measures of body size and cardiometabolic risk, I first use Latent Class Analysis (LCA) to identify different profiles of health based on the probabilistic co-occurrence of these measures across the adult population. LCA is a commonly used form of finite mixture modeling, allowing researchers to identify “unobserved” groupings or relationships among variables given a broad array of possible combinations and no clear *a priori* theoretical or empirical guidance on how these variables may be clustered (Masyn 2013). It is an increasingly popular technique in health research, allowing researchers to identify meaningful groupings of health behaviors and outcomes amid the numerous measures available in contemporary health surveys (Collins and Lanza 2010; Lanza and Rhoades 2013; Kongsted and Neilsen 2017), such as in identifying patterns of multimorbidity among adults (Larsen et al. 2017; Olaya et al. 2017; Schüz et al. 2009; Whitson et al. 2016). Indeed, Larsen et al. (2017: 2) note that “multimorbidity is a highly complex phenomenon, and the vast variety of disease combinations makes it a difficult phenomenon to analyze”, hence “[i]t is hardly practical to describe the prevalence and health outcomes of every conceivable disease combination, and much information is lost if multimorbidity is explored solely by counting disorders or applying one of several disease severity indices.”

Consequently, LCA is a less reductive approach as it divides the population into groups representing distinct and meaningful patterns based on individuals in the same group “shar[ing] a

common joint probability distribution among the observed variables” (Larsen et al. 2017: 3). Unlike its close analogue of confirmatory factor analysis, LCA draws on a covariance matrix of individuals to uncover latent groups of *individuals*, rather than to uncover latent *constructs* drawn from a matrix of items or measures (Bauer and Curran 2004). In other words, the focus is on identifying logical relationships and patterns among respondents which might otherwise be missed with interindividual, variable-centered analyses (Ferguson et al. 2020).

LCA is particularly useful for this study because extant research makes it difficult to anticipate *how many* classes one might expect to observe based on this set of indicators, or exactly what they would look like in terms of their composition. Based on the consistency in the health profiles observed in past research on MHO, I would not be surprised to observe the emergence of four groupings of body size and cardiometabolic health that are consistent with four-way categorization implied by dichotomizing both body size and cardiometabolic health as either healthy or unhealthy. However, I do not rule out the possibility that the groupings proposed in past literature are limited by researchers’ focus on identifying these exact combinations of body size and health in their data (i.e., pre-defining and then identifying “phenotypes”). While concern about the data, rather than hypothesis, driven nature of LCA is warranted (Schmiege et al. 2018), this approach can be instructive when there is a lack of clear theory, as in this case. Thus, LCA can help confirm extant theory on how body size and cardiometabolic health co-occur, refute these theories outright, or – as is likely the case with something as complex as health – augment extant theories with novel and interesting categorizations that may not be anticipated.

An additional useful aspect of LCA is its ability to efficiently handle missing data given the many indicators being used in these analyses. LCA uses a maximum likelihood estimation, assuming data are missing at random (Muthén and Muthén 2017). Indeed, actual missingness due to nonresponse is very low in NHANES; in these analyses, the majority of missing cases are attributable to their systematically not being asked in a given year or among a given portion of respondents (e.g., only those respondents being examined in the morning session to ensure fasting). For missingness among covariates in subsequent analyses, multiple imputation with chained equation is used, creating 10 imputed data sets

corresponding with the 10% missingness on the full set of covariates (White et al. 2011). Throughout all stages of the analyses, NCHS-provided survey weights are used per the method suggested by NHANES when pooling across multiple NHANES cycles of data (NCHS 2021).

In identifying the optimal number of classes that describe these NHANES data – and U.S. adults more generally – I examine changes in model fit statistics (AIC, BIC) with an increasing number of classes, where lower values are preferred, as well as conduct likelihood ratio tests which compare the nested  $k$  and  $k+1$  class solutions, with a significant p-value suggesting the  $k+1$  solution is not necessarily a better fit (Nylund et al. 2007). I also consider how well-differentiated these classes are, indicative of how accurately the indicators identify distinct groupings of individuals (Masyn 2013). A measure of entropy is often used as an “omnibus index” where values greater than 0.8 suggest individuals are accurately sorted into individual classes (Clark and Muthén 2009; Nylund-Gibson and Choi 2018). The average posterior probabilities (AvePP) provide additional information about how well a hypothesized model categorizes individuals into a given class as compared to one of the other options; values greater than 0.7 for the most-likely class indicate good separation (Nylund-Gibson and Choi 2018). Ensuring that the classes are well-differentiated is critical for these analyses, as evidence of a poorly-separated model would suggest considerable measurement error in the assignment of classes, and thus biased estimates in any subsequent analyses predicting membership into classes and estimating the association between classes and a distal outcome (Asparouhov and Muthén 2014; Bray et al. 2015).

Finally, the substantive interpretation of a given class – based on conditional probabilities – is important in identifying meaningful and plausible groupings, as is the relative size of the class within the population (Nylund-Gibson and Choi 2018). Focusing on the indicators of body size and cardiometabolic risk used in the analyses, LCA assigns an individual membership to a certain class based on a maximum likelihood estimate of their inclusion probability. These classes are in turn defined by conditional probabilities representing the likelihood that an individual *within* that class is likely to be characterized by a specific measure of body size or cardiometabolic risk. For instance, individuals in a class similar to the “metabolically-healthy obesity” phenotype noted in past research may have a >80% conditional

probability of having obesity but relatively low conditional probabilities (<40%) of having hypertension or dyslipidemia. Conversely, a “metabolically-unhealthy” normal-weight individual may have a <20% conditional probability of having obesity but higher conditional probabilities (>60%) of other measures of poor cardiometabolic health.

Having identified the best-fitting and substantively-meaningful number of latent classes describing body size and cardiometabolic health risk, I estimate the association between these classes and premature mortality risk. Survival time is measured using detailed information on individuals’ month of birth and death: discrete time-to-event Poisson regression models are used to obtain estimates of relative mortality risk (i.e., odds ratios) for different latent classes, relative to the referent group (or whichever would appear to be the lowest-risk, in this study). Specifically, I construct person-year file, wherein each individual has a record for each full or fraction of a year contributed at a specific age between 30 and 85 (Keyes et al. 2018).

Pursuant of the objective of understanding how these groupings of body size and cardiometabolic health are related to individuals’ educational attainment, I predict membership into the latent classes using multinomial logistic regression models, with latent classes as the outcome and educational attainment as the focal independent variable. To facilitate easier interpretation, I present these results as marginal probabilities and average marginal effects (Williams 2012), showing how the distribution of the different latent classes varies based on individuals’ educational attainment in both unadjusted and adjusted models.

In the final analyses, I combine information on educational attainment and mortality into a single model to estimate how much of the educational gradient in mortality is explained when accounting for the different distribution of latent groupings of body size and cardiometabolic risk across individuals. As death is defined as a binary outcome – and the resultant model assumes a logistic regression – simply comparing the change in coefficients after adding covariates, as in an OLS model, is likely to lead to biased results. Karlson, Holm, and Breen (2011) refer to this as an issue of “rescaling,” in the sense that the underlying latent variable corresponding with the probability of death is unobserved and thus differs between models as a function of the other covariates that included/excluded. Thus, standard comparisons



across models are misleading in failing to account for the confounding introduced by the outcome variable having a different underlying scale in each model (Breen et al. 2018). Their proposed solution – which can be applied using the “kfb” command in Stata (Kohler et al. 2011) – distinguishes changes in the coefficients due to this “rescaling” from the changes that occur due to adding variables to the model (i.e., the substantive changes of interest).

Given that these are cross-sectional data linked to longitudinal death records, I cannot definitively claim that these models represent a true “mediation” analysis, in the causal sense. However, this approach is consistent with extant research assessing how educational disparities in mortality risk change when accounting for plausible intermediate health mechanisms like body size and various biomarkers for cardiometabolic health. The Karlson, Holm, and Breen (KHB) method outlined above provides a more formal framework for making these comparisons of coefficients across models as compared to manually calculating the difference, as in past studies (Seeman et al. 2004). Moreover, this method not only estimates how much these latent classes mediate educational disparities in mortality, but it also estimates how much the *individual* classes contribute to these differences (Breen et al. 2013), providing novel insight on which groupings appear to be most influential.

## Results

As seen in the weighted, descriptive statistics in Table 3.1, NHANES is a nationally-representative sample of the U.S. adult population over the last three decades. Given that 50% of the sample is drawn from the NHANES III cycle – which is the largest single NHANES data collection on record – the overall sociodemographic profile skews slightly towards the earlier portion of this period. The average age is 49, 51% of respondents identify as female, and about 15% are foreign born. The sample is majority non-Hispanic White (74%); non-Hispanic Black adults represent 11% of respondents, Mexican-American adults (who are over-sampled in NHANES), represent approximately 6% of respondents, and the remaining 9% are a broad category of respondents with a racial/ethnic background that is not represented by the above categories. Educational attainment is fairly evenly distributed among the four categories, with 26% adults having a four-year college degree or more, 24% having some college

education or an associate’s degree (or equivalent), 29% having a high school degree or equivalent, and approximately 21% having less than a high school degree. The modal income-to-needs category is a ratio of 4.00 or higher (35.1%), followed by 2.00-3.99 (34.5%), 1.00-1.99 (18.7%), and 0-0.99 (11.7%). About 86% of respondents had health insurance coverage, and just under half had never smoked (47%), while approximately a quarter of respondents were former or current smokers (28% and 26%, respectively). Finally, 18.7% of respondents were determined to be deceased based on NCHS data linkages during the follow-up period.

**Table 3.1** Descriptive Statistics, National Health and Nutrition Examination Survey-Linked Mortality Files, 1988-2014, Ages 30-75

	48.8	95% C.I.	
		48.5	49.2
Age ( $\mu$ )	48.8	48.5	49.2
Survey Cycle			
1988-1994	50.0%	47.7%	52.2%
1999-2000	5.44%	4.99%	5.88%
2001-2002	6.23%	5.62%	6.84%
2003-2004	6.03%	5.29%	6.77%
2005-2006	6.17%	5.33%	7.01%
2007-2008	6.31%	5.54%	7.08%
2009-2010	6.44%	5.67%	7.20%
2011-2012	6.62%	5.64%	7.61%
2013-2014	6.79%	5.93%	7.65%
Female	51.4%	50.7%	52.0%
Race/ethnicity			
NH White	73.8%	71.9%	75.7%
NH Black	10.9%	9.94%	11.8%
MX-American	5.84%	5.15%	6.54%
Other	9.45%	8.33%	10.56%
Foreign-born	15.2%	13.7%	16.6%
Education			
Less than HS	20.8%	19.5%	22.1%
HS or equal	28.9%	27.8%	30.0%
Some college	24.3%	23.4%	25.1%
BA or higher	26.0%	24.7%	27.4%
Income-to-needs ratio			
0-0.99	11.7%	10.8%	12.7%
1.00-1.99	18.7%	17.8%	19.6%
2.00-3.99	34.5%	33.2%	35.8%
4.00+	35.1%	33.3%	36.8%

Health insurance	86.3%	85.2%	87.3%
Smoking status			
<i>Never</i>	46.7%	45.5%	47.9%
<i>Former</i>	27.8%	27.1%	28.6%
<i>Current</i>	25.5%	24.5%	26.5%
Proportion "determined deceased"	18.7%	17.6%	19.7%
<hr/>			
<b>Sample size</b>	40,095		
<hr/>			

*Notes:*

Estimates and associated confidence intervals account for NCHS-provided survey weights.

Estimates and associated confidence intervals based on multiple imputation to account for missing data.

Table 3.2 presents the weighted distributions of the body size and cardiometabolic health measures used to construct latent classes, as well as notes on the number of respondents with available data, which years the data come from, and how the measures are constructed (corresponding with the earlier explanation in the “Data” section). Once again, these indicators are representative of the entire time period covered by the NHANES data, and are thus slightly skewed towards population health patterns from the earlier years of the data range. For instance, approximately 31% of respondents have obesity at time of survey, which is lower than the current estimate of 40% referenced earlier (Hales et al. 2020). However, 42% of adults were ever considered obese based on retrospective measures. Approximately one-in-five adults (19%) report having obesity 10 years ago, and only 7.5% report having obesity at age 25. In terms of other anthropometric measures, the majority of adults are at risk for central obesity based on the waist-to-height ratio (78.4%), as well as having an unhealthy distribution of body fat based on their waist-to-hip ratio (71.9%), though the latter measure is only available in the NHANES III data from 1988 to 1994.

With respect to cardiovascular health, just over 1% of respondents had a dangerously elevated pulse rate at time of examination; however, 57% of adults had a blood pressure reading consistent with elevated risk of poor cardiovascular health, and approximately three-in-ten adults reported having

received a diagnosis of hypertension or were using antihypertensive medication. Similarly, 54% of respondents had elevated levels of total cholesterol, and three-in-ten were told they had high cholesterol by a doctor or were currently on cholesterol-lowering medications. Approximately 37% of adults had high triglyceride readings, and 39% had elevated levels of Apolipoprotein B, though the latter measure was only collected among morning session participants from 2005 through 2014. Under a quarter (23%) of respondents had low levels of the “good” high-density lipoprotein cholesterol.

**Table 3.2** Distribution of Latent Class Indicators, National Health and Nutrition Examination Survey-Linked Mortality Files, 1988-2014, Ages 30-75

		95% C.I.		N used in LCA	Availability	Notes on measures
<b>Body Size and History</b>						
<i>Obesity</i>	30.8%	29.9%	31.8%	39567	1988-2014	Obesity defined as BMI $\geq 30.0$ ; Body Mass Index (BMI [kg/m <sup>2</sup> ]) based on measured height and weight at examination.
<i>Ever obese</i>	41.7%	40.6%	42.7%	38847	1988-2014	Ever obese defined as maximum BMI $\geq 30.0$ ; Maximum BMI based on measured height at examination and highest ever recalled weight.
<i>Obesity 10 years ago</i>	18.8%	18.0%	19.7%	32586	1988-2014	Obesity 10 years ago defined as BMI 10 years ago $\geq 30.0$ ; BMI 10 years ago based on measured height at examination and recalled weight from 10 years ago.
<i>Obesity at age 25</i>	7.54%	7.07%	8.03%	37691	1988-2014	Obesity at age 25 defined as BMI at age 25 $\geq 30.0$ ; BMI at age 25 based on measured height at examination and recalled weight from age 25.
<i>Waist-to-hip</i>	71.9%	69.7%	74.0%	10714	1988-1994	High waist-to-hip ratio defined as 0.9 for males and 0.85 for females; Waist-to-hip ratio based on measured waist circumference and hip circumference at examination.
<i>Waist-to-height</i>	78.4%	77.4%	79.3%	38189	1988-2014	High waist-to-height ratio defined as $\geq 0.5$ ; Waist-to-height ratio based on measured waist circumference and measured height at examination.
<b>Cardiovascular</b>						
<i>Pulse</i>	1.28%	1.14%	1.44%	38663	1988-2014	High pulse rate, or tachycardia, defined as pulse rate $> 100$ beats per minute at examination.

<i>Hypertension (M)</i>	57.4%	56.4%	58.3%	38724	1988-2014	High blood pressure, in the pre-hypertensive range, defined as mean systolic blood pressure $\geq$ 120 mm Hg or diastolic blood pressure $\geq$ 80 mm Hg at examination.
<i>Hypertension (Dx)</i>	29.5%	28.6%	30.3%	39938	1988-2014	Hypertension based on received diagnosis or use of antihypertensive medication.
<b>Dyslipidemia</b>						
<i>High Chol. (M)</i>	53.5%	52.3%	54.7%	37933	1988-2014	High cholesterol, in the "abnormal" range, defined as measured blood cholesterol $\geq$ 200 mg/dL at examination.
<i>High Chol. (Dx)</i>	29.6%	28.8%	30.5%	39133	1988-2014	High cholesterol based on received diagnosis or use of cholesterol-lowering medication (e.g., statins).
<i>High Trigly.</i>	36.9%	35.6%	38.2%	37862	1988-2014	High triglycerides, in the "abnormal" range, defined as measured blood triglycerides $\geq$ 150 mg/dL at examination.
<i>High Apob</i>	39.2%	37.5%	41.0%	8951	2005-2014; Morning session	High ApoB (Apolipoprotein B), in the "abnormal" range, defined as $\geq$ 100 mg/dL at examination.
<i>Low HDL</i>	22.6%	21.6%	23.6%	37841	1988-2014	Low HDL (high-density lipoprotein), in the "abnormal" range, defined as measured blood HDL $<$ 40 mg/dL at examination.
<b>Hyperglycemia</b>						
<i>High HbA1c</i>	20.6%	19.5%	21.6%	38338	1988-2014	High HbA1c (hemoglobin A1c), in the "prediabetic" range, defined as $\geq$ 5.7% at examination.
<i>High Glucose</i>	42.5%	41.3%	43.6%	38157	1988-2014	High glucose, in the "prediabetic/insulin resistance" range, defined as glucose in serum or plasma $\geq$ 100 mg/dL (fasting) or $\geq$ 200 mg/dL (non-fasting) at examination.

<i>High Insulin</i>	7.21%	6.64%	7.83%	23930	1988-2014; Morning session	High insulin level, indicative of "insulin resistance," defined as fasting insulin $\geq 25$ mIU/L; Morning examination to help ensure fasting levels among participants.
<i>Diabetes (Dx)</i>	7.66%	7.27%	8.07%	40062	1988-2014	Diabetes based on received diagnosis or use of antidiabetic medication.
<b>Other</b>						
<i>High CRP</i>	31.0%	29.6 %	32.4%	30476	1988-2010	High CRP (C-reactive protein), indicative of a high-risk "inflammatory state/response," defined as $\geq 3.0$ mg/L at examination.
<i>High Alb.-to-Creat.</i>	1.26%	1.13%	1.42%	39223	1988-2014	High albumin-to-creatinine ratio in urine, indicative of microalbuminuria or kidney damage, defined as $\geq 30$ at examination.

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*Notes:*

Estimates and associated confidence intervals account for NCHS-provided survey weights.

Turning to indicators of hyperglycemia, approximately one-in-five adults had a high hemoglobin A1c reading suggesting consistently elevated levels of blood sugar for the past three months. Likewise, over 40% of respondents had elevated levels of blood glucose, and 7% of respondents had high insulin levels, though insulin was only collected for fasting participants in the morning session across all survey years. Approximately 8% of respondents had received a diagnosis of diabetes or were currently taking antidiabetic medication. Finally, approximately three-in-ten adults had a level of C-reactive protein consistent with an elevated inflammatory state, though these data were not collected in the 2011-2012 and 2013-2014 NHANES cycles. Just over 1% of respondents had an albumin-to-creatinine ratio in their blood consistent with evidence of microalbuminuria or kidney damage.

Based on this set of body size and cardiometabolic indicators, I address the first research aim by using LCA to examine how these measures co-occur within the U.S. adult population. Table 3.3 summarizes changes in fit statistics as the estimated number of latent classes increases from two to six across models. In general, all of the models appear to be well-differentiated based on the entropy and AvePP for classification: the entropy is above or near 0.8, and the AvePP consistently exceeds 0.7. Both the entropy and AvePP decline across classes, but these measures have to be contextualized among other measures of fit in helping to determine the best model. Namely, the likelihood ratio tests (Vong-Lo-Mendell-Rubin and Lo-Mendell-Rubin) comparing adjacent class solutions help to rule out the six-class solution based on the non-significant test result and the emergence of a fairly small class (5%). Likewise, the likelihood ratio tests help to rule out the two- and three-class solutions, as the significant test result for the  $k+1$  classes (three and four, respectively) suggest that a four-class solution is preferred to both.



**Table 3.3** Fit Assessment for Models with 2-6 Latent Class Solutions

	Measures of Fit			Likelihood Ratio Test of K/K-1 Solution		Additional Diagnostics		
	<i>AIC</i>	<i>BIC</i>	<i>SSA BIC</i>	<i>VLMR LRT</i>	<i>LMR A-LRT</i>	<i>Entropy</i>	<i>Average Posterior Probability for Classification</i>	<i>Smallest Class</i>
2 Classes	611376	611729	611599	0.000	0.000	0.880	0.964	39%
3 Classes	593315	593848	593651	0.000	0.000	0.862	0.909	30%
4 Classes	584432	585146	584882	0.000	0.000	0.847	0.869	12%
5 Classes	581173	582067	581737	0.062	0.063	0.807	0.840	10%
6 Classes	577952	579027	578630	0.184	0.186	0.794	0.817	5%

*Notes:*

National Health and Nutrition Examination Survey, 1988-2014, Ages 30-75.

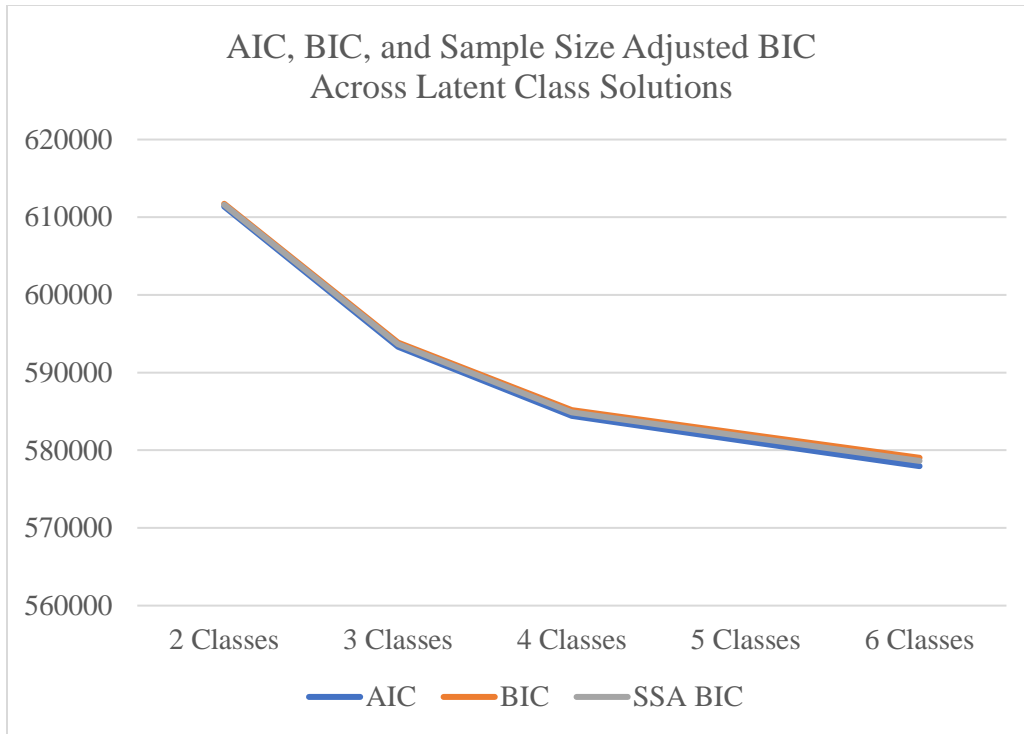
Estimates account for NCHS-provided survey weights.

Non-significant LRT indicates *K-1* class solution preferred over *K* class solution.

Entropy >0.8 indicative of good separation of individuals into classes.

Average posterior probabilities >0.7 indicative of well-separated classes.

By contrast, there is some ambiguity in deciding between the four- and five-class solution. As seen in Figure 3.1, the AIC, BIC, and SSA-BIC values decrease across models, but there is no clear “elbow” in the trend at either the four- or five-class solution to suggest a large improvement after which there is leveling-off in fit (Nylund-Gibson and Choi 2018). The advantages of the four-class solution are a slightly higher entropy and AvePP, as well as a marginally non-significant likelihood ratio test compared to the five-class solution. However, Chen et al. (2017) warn that likelihood ratio tests have a tendency to “over-extract” the correct number of classes in large-N samples, such as these data. Consequently, I examine estimates from the conditional item probabilities to see if the five-class solution offers substantively interesting information beyond that provided by the four-class solution.



**Figure 3.1** AIC, BIC, and Sample Size Adjusted BIC Across Latent Class Solutions

As seen in Table 3.4, the conditional probabilities from the five-class solution suggest five fairly distinct groupings of body size and cardiometabolic health. Approximately three-in-ten U.S. adults (28%) are described by an “Ideal” latent class, in reference to their very low conditional probabilities of having any of the body size or cardiometabolic risks considered in these analyses. Indeed, the highest conditional probabilities are for waist-to-hip, waist-to-height, measured hypertension, and measured high cholesterol, but none of them exceed 34%. On the opposing end of the risk spectrum, one-in-ten adults (10%) are represented by a High Risk group, based on their high conditional probabilities of key risk factors across a majority of measures. These individuals have a greater than 80% conditional probability of current obesity, ever having obesity, and being at risk for central obesity, as well as the highest probability of having obesity 10 years ago (65%). They also have higher probabilities of measured or diagnosed hypertension (~75%) and high hbA1c or glucose (>80%); while the conditional probabilities for high insulin and diagnosed diabetes are lower (40% and 53%, respectively), they are higher than any other class. Notably, this group has the highest probability of dangerously elevated C-reactive protein at 65%.

**Table 3.4** Conditional Item Probabilities for 5-Class Solution

	"Ideal" (28%)	"Fat but Fit" (13%)	Mixed Health w/o Obesity (35%)	Mixed Health w/ Obesity (14%)	High Risk (10%)
<b>Body Size and History</b>					
<i>Obesity</i>	0.50%	68.10%	4.10%	85.2%	83.5%
<i>Ever obese</i>	2.20%	100%	11.7%	100%	100%
<i>Obesity 10 years ago</i>	0.00%	42.6%	0.00%	41.8%	65.4%
<i>Obesity at age 25</i>	0.00%	22.9%	0.00%	15.0%	24.4%
<i>Waist-to-hip</i>	30.0%	73.6%	93.3%	96.9%	97.1%
<i>Waist-to-height</i>	33.9%	97.9%	92.4%	100.0%	99.7%
<b>Cardiovascular</b>					
<i>Pulse</i>	0.70%	1.10%	1.30%	1.20%	3.40%
<i>Hypertension (M)</i>	30.3%	51.5%	68.1%	75.6%	79.0%
<i>Hypertension (Dx)</i>	7.40%	27.6%	31.6%	42.4%	68.0%
<b>Dyslipidemia</b>					
<i>High Chol. (M)</i>	33.6%	29.7%	69.2%	80.7%	48.6%
<i>High Chol. (Dx)</i>	11.2%	15.4%	38.7%	43.1%	49.8%
<i>High Trigly.</i>	5.80%	12.9%	49.7%	72.3%	61.7%
<i>High Apob</i>	10.1%	5.10%	60.1%	93.5%	32.5%
<i>Low HDL</i>	6.20%	17.1%	26.2%	39.6%	39.0%
<b>Hyperglycemia</b>					
<i>High HbA1c</i>	4.10%	8.30%	20.8%	18.0%	84.7%
<i>High Glucose</i>	19.7%	28.2%	50.9%	48.5%	87.1%
<i>High Insulin</i>	0.40%	3.70%	3.80%	10.9%	40.0%
<i>Diabetes (Dx)</i>	0.60%	1.00%	5.50%	0.10%	53.2%
<b>Other</b>					
<i>High CRP</i>	12.4%	45.6%	28.8%	42.8%	65.5%
<i>High Alb.-to-Creat.</i>	0.20%	0.40%	1.10%	0.60%	6.80%

*Notes:*

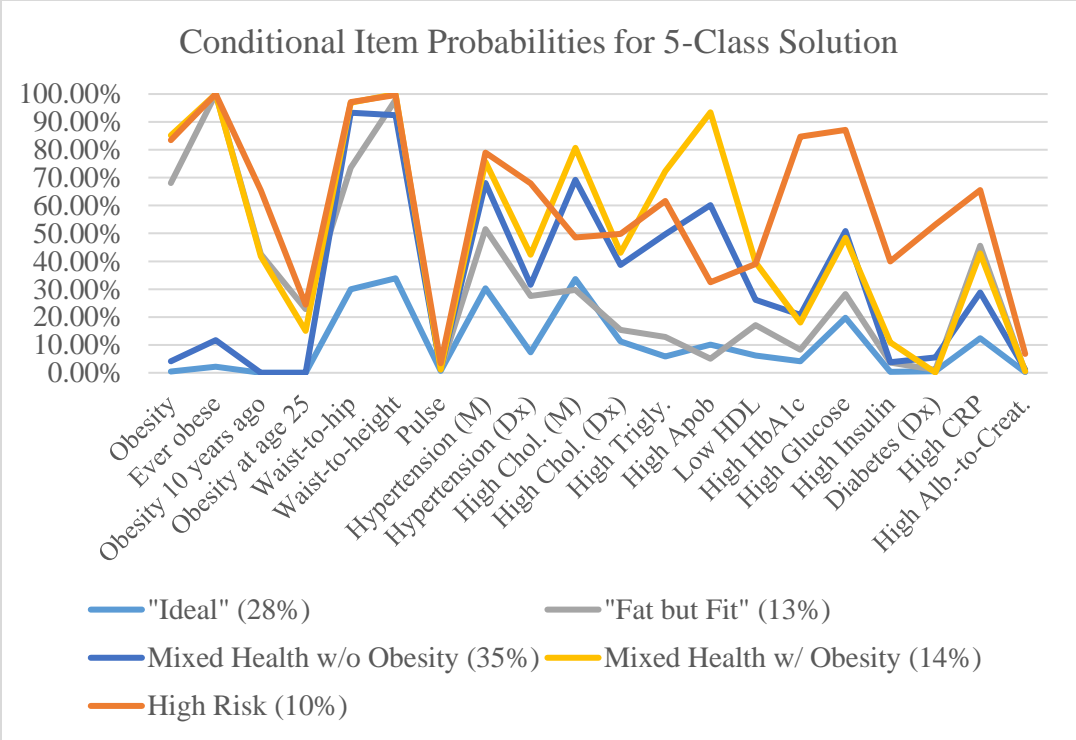
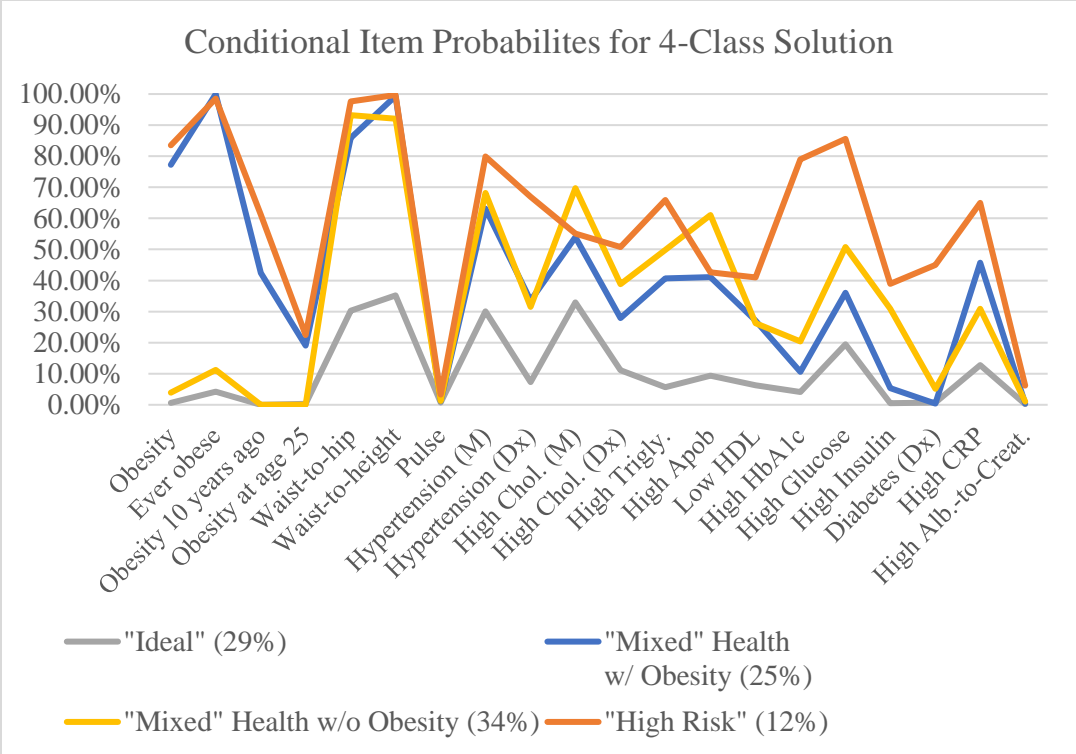
National Health and Nutrition Examination Survey, 1988-2014, Ages 30-74.

Estimates account for NCHS-provided survey weights.

Interestingly, between the “Ideal” and High Risk group, I find that a majority of adults (62%), fall into what might be described as *intermediate* risk classes. Closer to the “Ideal” class, 13% of adults can be described as “Fat but Fit” – to use the language of past research on MHO – in the sense that they are virtually indistinguishable in their cardiometabolic health from adults without obesity. As seen in the

conditional probabilities, this group is more likely to have obesity (68%) or to have ever had obesity (100%), as well as to have risks of central adiposity (>70%). Yet, with the exception of measured hypertension (52%), only high C-reactive protein exceeds 30%. The Mixed Health without Obesity class is the modal group (35%), representing the inverse of the “Fat but Fit” class. Their probability of ever having had obesity is very low, yet they have elevated probabilities of a number of cardiometabolic risks, like measured hypertension (68%), measured high cholesterol (69%), high glucose (50.9%), and a high level of apolipoprotein B (60%). Granted, their conditional probabilities for high waist-to-height and waist-to-hip ratios are elevated as well, exceeding 90%. Finally, there is a Mixed Health with Obesity group, resembling the former group in terms of their cardiometabolic risk, but not quite having the consistently elevated risk profile associated with the High Risk group. The conditional probabilities of obesity, ever having obesity, or being at risk for central obesity exceed 85%, while the conditional probabilities for measured hypertension, measured high cholesterol, and high triglycerides exceed 70%. This group also stands out for having the highest conditional probabilities of high levels of apolipoprotein B (93%) and low levels of HDL (40%).

Compared to the four-class solution (Table A.1 in Appendix), there are key similarities with regards to the “Ideal,” High Risk, and Mixed Health groups. However, the five-class solution offers an important substantive addition of the aforementioned “Fat but Fit” class, as well as greater differentiation in how cardiometabolic risk is distributed among the other intermediate groups. Per the recommendation by Nylund et al. (2007), Figure 3.2 presents a linear plot comparing the conditional probabilities across the four- and five-class solutions to help differentiate between the two. Both models echo some of the basic findings about metabolically-healthy/unhealthy obesity/non-obesity in past research; however, the added nuance provided by the five-class solution without an appreciable decrease in fit leads me to conclude that it is the appropriate choice for subsequent analyses.



**Figure 3.2** Conditional Item Probabilities for 4-Class Solutions

Having identified the five-class solution, I address the second research aim of “validating” these classes in estimating their associated mortality risk and, thus, their implications for long-term health. In these models, I use the “Ideal” class as the baseline, anticipating that this group of individuals should have the lowest mortality risk due to the substantially lower conditional probabilities of both high body size and worse cardiometabolic health. As seen in Table 3.5, this assumption is accurate; all of the four other classes have significantly higher mortality risk relative to the “Ideal” group. In these unadjusted models, the “Fat but Fit” group still has higher relative risk (OR 1.57 [1.34-1.84]), but this risk is lower than both of the other Mixed Health classes, whose relative risk is similar to one another (OR ~2.65). Unsurprisingly, the High Risk group has the highest relative risk of early death, more than *sixfold* higher than the “Ideal” comparison (OR 6.11 [5.30-7.05]). This pattern is repeated across all causes of death, though the smaller counts of death in the underlying diabetes, underlying hypertension, and heart disease categories result in very large confidence intervals and less reliable risk estimates for the smaller High Risk class. Even after adjusting for the additional covariates described earlier, all classes are associated with increased risk relative to the “Ideal” group (Table A.2 in Appendix). However, the magnitude of the risk is substantially reduced, such that the High Risk group is associated with a twofold increase in risk (OR 2.04 [1.77-2.35]) and the three other groups are very similar to each other, indicating a ~30% increase in mortality risk relative to the “Ideal” group. Interestingly, the “Fat but Fit” group is not associated with increased risk relative to the “Ideal” group for a number of causes of death (Table A.3 in Appendix).

**Table 3.5** Cause-specific Mortality Risk Across Latent Classes

	All Cause			Underlying Diabetes			Underlying Hypertension			Heart Disease			Heart Disease, Diabetes, or Cancer		
	<i>Odds Ratio</i>	<i>95% CI</i>		<i>Odds Ratio</i>	<i>95% CI</i>		<i>Odds Ratio</i>	<i>95% CI</i>		<i>Odds Ratio</i>	<i>95% CI</i>		<i>Odds Ratio</i>	<i>95% CI</i>	
<b>5-Class Solution</b> <i>(ref. "Ideal")</i>															
"Fat but Fit"	1.57	1.34	1.84	1.52	0.62	3.74	1.88	1.04	3.40	1.35	0.84	2.15	1.35	1.10	1.65
Mixed Health w/ Obesity	2.72	2.36	3.14	5.52	2.74	11.1	4.28	2.53	7.24	4.11	2.95	5.72	2.76	2.31	3.29
Mixed Health w/o Obesity	2.57	2.22	2.98	7.55	3.77	15.1	5.61	3.08	10.2	4.18	2.99	5.85	2.61	2.16	3.16
High Risk	6.11	5.30	7.05	51.6	26.4	101	18.0	10.4	31.1	11.88	7.82	18.1	6.24	5.02	7.77
<b>Sample size</b>	40,095			40,095			40,095			40,095			40,095		
<b>Number of deaths</b>	7,106			1,017			1,014			1,395			3,502		

*Notes:*

National Health and Nutrition Examination Survey, 1988-2014, Ages 30-75.

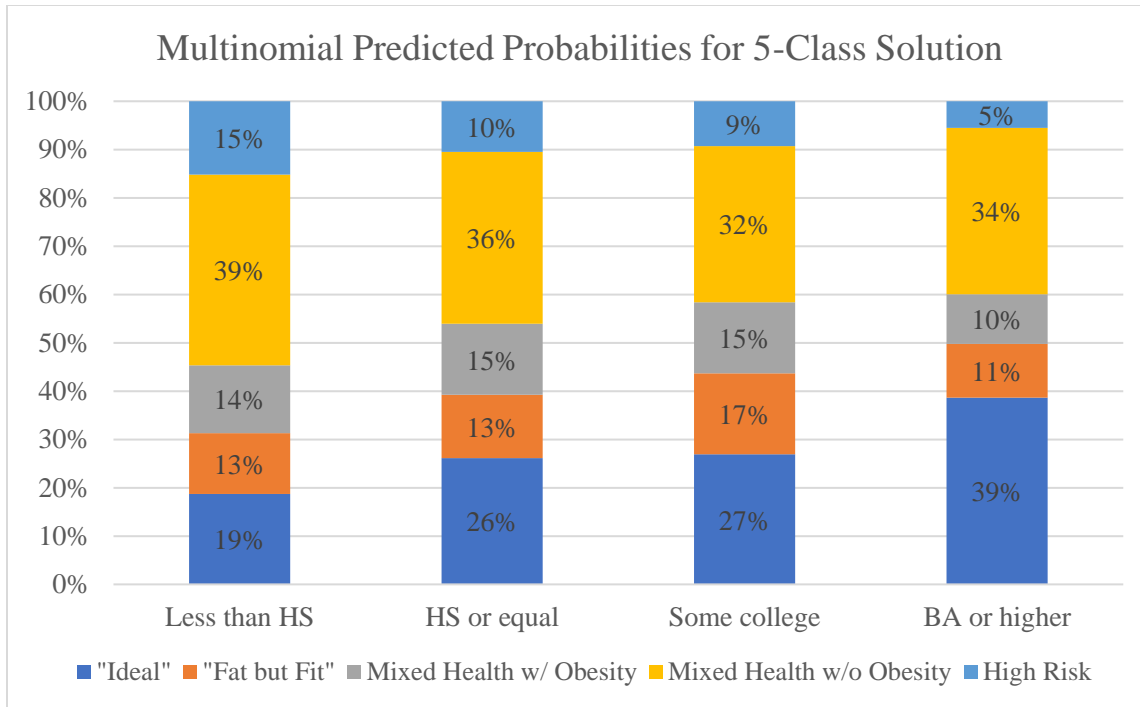
Deaths restricted to ages 30-85.

Estimates account for NCHS-provided survey weights.

Estimates based on multiple imputation to account for missing data.

In the final section of the analyses, I address the goals of the third research aim by examining the association between individuals' educational attainment and how these different latent classes of body size and cardiometabolic health are distributed throughout the population. As seen in the predicted probabilities from a multinomial logistic regression in Figure 3.3, the distribution of these “phenotypes” is far from random, and clearly socially-patterned on the basis of individuals' educational attainment. The overall distribution of classes is more favorable among higher-educated adults as compared to those with less education – especially those with less than a high school degree, who are nearly half as likely to be in the “Ideal” class (19% vs. 39%), and three times more likely to be in the High Risk group (15% vs. 5%). Those with a high school degree or some college education are fairly similar to one another, with a distribution in between the two ends of the education spectrum (~27% in “Ideal” and ~10% in High Risk). Interestingly, however, the three intermediate risk groups are fairly evenly distributed across all four levels of educational attainment. College-educated adults are least likely to be “Fat but Fit” (11%), whereas those with some college education are most likely to be represented by this group (17%). Mixed Health with Obesity is least prevalent among the highly educated (10%), but fairly similar across other groups (~15%). Finally, the fairly high risk set of individuals with elevated cardiometabolic health but not considered obese constitute about a third of all four educational groups, albeit being more highly represented among those with a high school degree (36%) or less than a high school degree (39%).





**Figure 3.3** Multinomial Predicted Probabilities for 5-Class Solution

Table 3.6 provides a more formal comparison of these predicted probabilities based on average marginal effects (AME) from both unadjusted models (the same as the results in Figure 3.3) and adjusted models, where all covariates are held at their means across individuals. While the AMEs decline in the adjusted models, the general pattern noted above remains the same, as seen in the much larger differences in the lowest and highest risk groups contrasted with small and non-significant differences across the other latent classes. For example, there is a graded relationship between educational attainment and membership in “Ideal” group, ranging from an 11.1% lower predicted probability among those with some college education relative to a college degree, to a 15.5% lower probability among those with less than a high school education compared to those with a college degree. The inverse pattern is observed for the High Risk group. Differences in the intermediate groups are less apparent, with no significant difference in predicted probabilities for the large Mixed Health without Obesity class.

**Table 3.6** Average Marginal Effects for Educational Attainment Across Latent Classes

		<i>"Ideal"</i>					
		Unadjusted			Adjusted		
		95% C.I.			95% C.I.		
<b>Education (ref. BA or higher)</b>							
<i>Less than HS</i>	-19.9%	-22.5%	-17.4%	-15.5%	-18.1%	-12.9%	
<i>HS or equal</i>	-12.5%	-14.6%	-10.4%	-12.3%	-14.3%	-10.2%	
<i>Some college</i>	-11.7%	-14.7%	-8.74%	-11.1%	-13.8%	-8.29%	
		<i>"Fat but Fit"</i>					
		Unadjusted			Adjusted		
		95% C.I.			95% C.I.		
<b>Education (ref. BA or higher)</b>							
<i>Less than HS</i>	1.46%	-0.16%	3.08%	2.69%	0.85%	4.52%	
<i>HS or equal</i>	1.96%	0.61%	3.31%	2.19%	0.81%	3.57%	
<i>Some college</i>	5.63%	4.15%	7.11%	4.14%	2.68%	5.60%	
		<i>Mixed Health w/ Obesity</i>					
		Unadjusted			Adjusted		
		95% C.I.			95% C.I.		
<b>Education (ref. BA or higher)</b>							
<i>Less than HS</i>	3.80%	2.16%	5.44%	4.29%	2.11%	6.48%	
<i>HS or equal</i>	4.45%	2.80%	6.09%	4.57%	2.79%	6.35%	
<i>Some college</i>	4.45%	2.65%	6.3%	4.16%	2.32%	5.99%	
		<i>Mixed Health w/o Obesity</i>					
		Unadjusted			Adjusted		
		95% C.I.			95% C.I.		
<b>Education (ref. BA or higher)</b>							
<i>Less than HS</i>	4.95%	2.33%	7.56%	1.77%	-0.99%	4.54%	
<i>HS or equal</i>	1.13%	-1.31%	3.56%	0.87%	-1.63%	3.37%	
<i>Some college</i>	-2.17%	-4.65%	0.32%	-0.29%	-2.66%	2.09%	
		<i>High Risk</i>					
		Unadjusted			Adjusted		
		95% C.I.			95% C.I.		
<b>Education (ref. BA or higher)</b>							
<i>Less than HS</i>	9.71%	8.53%	10.9%	6.71%	5.27%	8.15%	
<i>HS or equal</i>	4.97%	3.77%	6.17%	4.64%	3.34%	5.93%	
<i>Some college</i>	3.80%	2.91%	4.68%	3.04%	2.11%	3.96%	

*Notes:*

National Health and Nutrition Examination Survey, 1988-2014, Ages 30-75.

Estimates account for NCHS-provided survey weights.

Estimates based on multiple imputation to account for missing data.

Adjusted for educational attainment, age, survey year, gender, race/ethnicity, nativity, income-to-needs ratio, health insurance, and smoking status.

The full set of AMEs for all covariates used in the adjusted models is included in the Appendix (Table A.4). Briefly, the AMEs generally follow expected patterns based on past research on

sociodemographic measures and health. Age is inversely related with the probability of being in a higher-risk class; female respondents are more likely to be represented in the lower-risk “Ideal” and “Fat but Fit” groups; non-Hispanic Black and Mexican-American adults are less likely to be in the “Ideal” group and more likely to be in the High Risk group relative to non-Hispanic White adults. Interestingly, being a current smoker relative to never smoking is associated with a higher probability of being in the “Ideal” group and a lower probability of being in the High Risk group; however, it is important to note that smoking is associated with weight loss and a lower BMI (Stokes and Preston 2016), and not necessarily positively associated with all of the specific cardiometabolic risk indicators under consideration (e.g., measures of hyperglycemia).

For the final set of analyses, I consider how differences in the educational distribution of these latent classes of body size and cardiometabolic health can help to explain educational disparities in mortality risk. Namely, while the distribution of the high- and low-risk classes is clearly stratified by education, the fact that intermediate classes of risk are more evenly represented across all levels of education makes it hard to anticipate how these different groupings contribute to overall educational disparities in mortality risk. Based on the KHB results in Table 3.7, I find that accounting for these groupings of body size and cardiometabolic risk alone mediates 17% of the differences in mortality risk for those with a college degree or higher relative to their less than high school-educated counterparts, 16% of the differences compared those with only a high school degree, and 18.5% compared to those with some college education or the equivalent. In the models controlling for additional covariates, the average percent of the gradient mediated decreases to 14%. The estimated mediation is largely unchanged in the adjusted models for the high school only and less than high school groups, though only 11.7% of differences are explained when comparing some college educated adults to their college educated counterparts.

**Table 3.7** Percent Contribution of Latent Classes to Educational Disparities in All-Cause Mortality

	Percent Mediated	
	<i>Unadjusted</i>	<i>Adjusted</i>
<b>Education (ref. BA or higher)</b>		
<i>Less than HS (ref. "Ideal")</i>	17.3	16.2
"Fat but Fit"	1.06	1.79
Mixed Health w/ Obesity	2.35	2.97
Mixed Health w/o Obesity	3.99	1.67
High Risk	9.92	9.72
<i>HS or equal (ref. "Ideal")</i>	15.8	14.8
"Fat but Fit"	1.70	2.43
Mixed Health w/ Obesity	4.10	3.72
Mixed Health w/o Obesity	1.48	1.37
High Risk	8.55	7.27
<i>Some college (ref. "Ideal")</i>	18.5	11.7
"Fat but Fit"	5.33	4.25
Mixed Health w/ Obesity	5.80	2.99
Mixed Health w/o Obesity	-1.54	0.79
High Risk	8.91	3.72

*Notes:*

National Health and Nutrition Examination Survey, 1988-2014, Ages 30-75.

Deaths restricted to ages 30-85.

Estimates account for NCHS-provided survey weights.

Adjusted for age, age-squared, survey year, gender, race/ethnicity, nativity, income-to-needs ratio, health insurance, and smoking status.

Critically, the KHB method allows for additional insights on the individual contributions of the latent classes themselves, estimating the percent mediation of each class as compared to the “Ideal” group. This additional “disentangling” feature of the KHB method is only possible in non-imputed data, so I checked that the overall estimated proportion of mediation is the same in both the imputed and non-imputed samples before proceeding with analyses; fortunately, the results were nearly identical. Similar to the clear educational disparities in the High Risk group observed earlier, these results show that this class accounts for the majority of the overall mediation, whereas the intermediate risk classes individually account for a much smaller proportion of the overall differences. This pattern is less clear among those

with some college education, where the Mixed Health without Obesity group accounts for the smallest proportion in the adjusted model, while the other three groups are relatively similar in the magnitude of their contribution (~3.5%).

I also ran these adjusted KHB mediation models for the other cause of death categories, as seen in Table 3.8. Overall, there is a very similar pattern of mediation, albeit with considerably higher estimates for underlying diabetes, underlying hypertension, and heart disease – as might be expected for causes of death more closely linked with these latent class indicators of body size and cardiometabolic health. Indeed, the latent classes explain approximately *one-third* of the educational gradient in mortality risk among deaths where diabetes or hypertension were contributing causes. They also account for approximately *one-quarter* of the educational gradient in heart disease mortality risk when comparing college educated adults to those with a high school education or less. However, the percent mediation is closer to the all-cause mortality estimates when examining deaths from heart disease, diabetes, or cancer. Once again, the High Risk group accounts for a very large proportion of the difference, close to or greater than half of the total mediation across the different causes of death. There is a fairly minimal contribution from the “Fat but Fit”, and greater influence for the two Mixed Health groups, especially with obesity.

**Table 3.8** Percent Contribution of Latent Classes to Educational Disparities in Cause-specific Mortality

	Percent Mediated			
	<i>Underlying Diabetes</i>	<i>Underlying Hypertension</i>	<i>Heart Disease</i>	<i>Heart Disease, Diabetes, or Cancer</i>
<b>Education (ref. BA or higher)</b>				
<i>Less than HS (ref. "Ideal")</i>	35.6	28.5	24.4	16.5
"Fat but Fit"	1.00	2.00	0.48	1.15
Mixed Health w/ Obesity	8.44	7.00	6.07	3.14
Mixed Health w/o Obesity	5.15	3.39	3.60	1.83
High Risk	23.0	16.1	14.2	10.4
<i>HS or equal (ref. "Ideal")</i>	31.7	21.8	25.2	13.9
"Fat but Fit"	1.30	2.25	0.75	1.47
Mixed Health w/ Obesity	10.06	7.27	8.76	3.72
Mixed Health w/o Obesity	4.01	2.30	3.40	1.42
High Risk	16.32	10.0	12.3	7.31
<i>Some college (ref. "Ideal")</i>	37.2	41.9	13.7	9.84
"Fat but Fit"	4.02	10.2	1.08	2.51
Mixed Health w/ Obesity	14.3	15.1	5.80	2.90
Mixed Health w/o Obesity	4.08	3.43	1.61	0.79
High Risk	14.8	13.2	5.17	3.64

*Notes:*

National Health and Nutrition Examination Survey, 1988-2014, Ages 30-75.

Deaths restricted to ages 30-85.

Estimates account for NCHS-provided survey weights.

Adjusted for age, age-squared, survey year, gender, race/ethnicity, nativity, income-to-needs ratio, health insurance, and smoking status.

*Sensitivity Analyses*

As noted in the description of the methods, this analytic approach is what researchers describe as the “one step” method for LCA, wherein individuals are assigned to a specific latent class that are then used in subsequent analyses with predictors and outcomes. Though the estimates of entropy and average posterior probabilities in these analyses suggest well-differentiated models, there is still a possibility that the decision to assign individuals to a specific class as opposed to an alternate option could change the

results. Recent years have seen the development of new methods that better account for uncertainty in the latent class assignment (Bakk et al. 2013); however, they are not well-suited for the discrete-time survival models with mortality as the distal outcome. Indeed, there was some ambiguity in the results concerning the choice of the four- or five-class model. Even though the literature on LCA suggests that researchers have some discretion in identifying the most appropriate model, I wanted to make sure that the choice of five rather than four classes did not alter key substantive takeaways. Thus, I conducted a robustness check to see if the key results of these analyses are upheld when using the four-class solution. As seen in the Appendix (Tables A.5-A.8), these sensitivity analyses provide results that are very similar to those in the primary analyses, both in terms of how they characterize the population with respect to body size and cardiometabolic health and how these groupings are related to mortality. These analyses also result in similar estimates of educational differences in the groupings, as well as comparable estimates of the educational gradient in mortality risk explained by the latent classes.

I also ran Latent Profile Analyses (LPA) using continuous versions of the same set of indicators, finding similar patterns of results up through the four-class solution. However, these models consistently encountered greater convergence and replication issues, leading me to question the reliability of these estimates; indeed, convergence and replication issues are common given the sensitivity of LPA models to the different scales and distributions of indicators (Berlin et al. 2014), as in these analyses. More broadly, throughout the analysis I examined the sensitivity of the latent class solutions to alternate specifications of indicators with respect to the cut-point for ‘high’ vs. ‘low’ risk, finding that the results were very consistent in terms of both distribution and substantive interpretation of the latent classes. I also considered different groupings of variables – such as limiting the analyses to only those variables with data on all respondents across all years, or avoiding multiple measures of a given domain of health, or including only measures with conditional probabilities <30% or >70%, among others – again finding that the emergent latent class solutions were largely unchanged. Importantly, the set of variables used in the main analyses showed the best evidence of well-separated classes across the various combinations of indicators examined.

Finally, I also ran gender-stratified models, finding the four- and five-class LCA solutions for both females and males were fairly similar, albeit with slightly different conditional probabilities among indicators. Moreover, subsequent analyses of both educational distribution and mediation were also comparable, largely mirroring the findings noted above; generally, the proportion of educational disparities in mortality mediated by the latent classes was higher for women than men. However, the issue of unstable estimates associated with some of the latent groupings was magnified when running stratified models; the intersection of gender, education, latent classes, and specific causes of death resulted in cells with relatively few cases. Given the lack of major differences in findings, and to avoid any ambiguity in the interpretation of the results, I limit my discussion to the sample as a whole.

### **Discussion and Conclusion**

There is compelling evidence that obesity is a key driving force underlying patterns and trends of morbidity and mortality in the United States (NASEM 2021; NHLBI 2013), yet research based on biomarker data from large national surveys suggests that body size is not a monolith when it comes to individual and population health (Tomiya et al. 2016; Wildman et al. 2008). Examining heterogeneity in body size and health is an important consideration in the United States, where over four in ten adults are considered obese (Hales et al. 2020). While excess body size can be harmful, it is not a definitive marker of health (Gutin 2018); thus, a large proportion of adults with obesity, and the population as a whole, may be able to enjoy “good” health. More importantly, knowledge of this heterogeneity is critical for reevaluating population health priorities centered on weight loss and maintaining a “normal” weight given the abundance of research highlighting the failures of weight-focused interventions (Bacon and Aphrarmor 2011; Kraschnewski et al. 2010; Mann et al. 2007). However extant research on this subject has focused on predefined categories of body size and cardiometabolic health – often with a fairly limited set of indicators – rather than identifying patterns emerging from the rich population health data at our disposal (Blüher 2020; Magkos 2019; Phillips 2013). This research tends to focus on identifying “phenotypes” of metabolically-healthy obesity, but does not fully consider the population health relevance of other body size and health groupings. Moreover, this research has adopted a largely



biological and genetic perspective on these phenotypes (Huang et al. 2017; Navarro et al. 2015; Telle-Hansen et al. 2013), giving less attention to key social factors – like individuals’ educational attainment – as a determinant of these health profiles.

Drawing on clinical and epidemiological research on body size and health, as well as sociological and social demographic work examining the role of educational attainment disparities in health, this study identifies probabilistic groupings of body size and cardiometabolic health in the U.S. adult population and examines their contribution to educational gradients in mortality. Based on the results of latent class analysis and survival analysis with National Health and Nutrition Examination Survey data from 1988-2015, approximately one-third of adults have a clinically “Ideal” health profile, in terms of having a medically healthy body size and not exhibiting any major cardiometabolic dysregulation. However, the remaining two-thirds of adults show some degree of ‘unhealthiness’ on the basis of either body size, cardiometabolic health, or both, having body size and cardiometabolic health profiles associated with increased all-cause mortality risk relative to their “Ideal” health counterparts. The five latent groupings of body size and cardiometabolic health identified in the data are consistent with past research on MHO, wherein adults are categorized as either metabolically-healthy or unhealthy and either having obesity or not. However, the results document greater heterogeneity in obesity as a risk group, to the extent that adults with obesity exhibit relatively low, medium, and high levels of risk relative to the “Ideal” group. As expected, the High Risk group – consisting of adults with a higher probability of both obesity and impaired cardiometabolic health – have the highest relative mortality risk, and represent one-in-ten adults. By contrast, a “Fat but Fit” group – conceptually similar to MHO – represents 13% of adults, with a slightly elevated mortality risk relative to the “Ideal” group. The remaining two groups consist of adults that are very similar in terms of their cardiometabolic health profiles, yet one has obesity (14%) and the other does not (35%). Indeed, the latter is the modal group, accounting for over a third of the sample, whose mortality risk is significantly higher than both the “Ideal” and “Fat but Fit” groups across all causes of death, and on par with their nearly identical cardiometabolic counterparts who have obesity.

Moreover, these are not exclusively biological phenotypes, as college-educated adults generally have a more advantageous distribution of these latent groupings relative to their less-educated counterparts. The most apparent differences are in the low-risk, “Ideal” group, where college-educated adults are more than twice as likely to be represented relative to their less than high school educated counterparts, and about one-and-a-half times more likely to be represented than those with a high school but not college degree. The High Risk group shows an inverse pattern, as less than high school educated adults are three times more likely to be represented than college educated adults, and one-and-a-half times more likely to be represented than those with a high school but not college degree. However, these educational differences are far less pronounced among the intermediate risk groups, which is where the majority of adults are classified across all educational groups. When combining information on education, latent groupings, and mortality, these educational differences in groupings of body size and cardiometabolic health account for approximately 17% of educational disparities in mortality when using only latent classes as a mediator, and 14% of differences when adjusting for a comprehensive set of covariates. This mediation is larger for causes of death more closely associated with obesity and cardiometabolic health (20-40%). Notably, the High Risk group accounts for the majority of this mediation relative to the “Ideal” counterfactual; nevertheless, differences across all latent groupings tend to favor highly-educated adults.

First and foremost, this study reaffirms past research suggesting that body size and cardiometabolic health exhibit considerable variation in the United States, with various combinations of body size-related and other clinically significant cardiometabolic risks. As noted in past work, treating obesity as a singular category of poor health represents a false binary from a population health perspective, as evidenced by the more nuanced configurations seen in these results. Nearly one-in-eight adults are represented by the “Fat but Fit” group similar to MHO, which is associated with increased mortality risk in the bivariate models but is not significantly associated with increased risk relative to the “Ideal” group in cause-specific models adjusted for covariates. While not addressing the psychosocial implications of HAES, these findings confirm the perspective that individuals can maintain relatively

good health without a narrow focus on weight and weight loss (Bombak 2014; Penney and Kirk 2015). Critically, high body weight or excess adiposity is not the only factor associated with elevated mortality risk, as nearly one-third of adults do not have obesity yet exhibit signs of cardiometabolic dysfunction that places them at higher risk of early death, relative to both the “Ideal” and “Fat-but-Fit” groups. Moreover, their cardiometabolic profile and risk level is nearly identical to the latent grouping of individuals who share the same cardiometabolic profile but have obesity.

These findings echo extant concerns that a narrow-minded focus on obesity and its associated cardiometabolic implications leads us to falsely equate thinness with good health (Saguy 2012; Shugart 2016; Tomiyama et al. 2016). As seen in these data, and found in past research, the proportion of U.S. adults who *do not* have obesity but are not necessarily ‘healthy,’ is nearly equivalent to the proportion of adults who have obesity. While parsimony is valuable in population health, and there are many instances where researchers have a legitimate interest in examining a singular risk factor, this study emphasizes how the broader agenda of improving population health is incomplete without considering the *multiplicity* of risk factors affecting individuals and how they may present themselves in distinct, but substantively important, configurations. Even though existing theories and related measurement schemes on health and aging provide some guidance on how to conceptualize different combinations of comorbidities – like MHO, metabolic syndrome, and allostatic load – methods like LCA can be used to scrutinize these theories/methods or to find ways that they can be refined, such as looking at probabilistic groupings rather than using sum scores or indices in identifying latent classes with unique substantive interpretations.

On the subject of multiplicity in risk and morbidity, this study has key implications for our understanding of educational disparities in mortality and the different mechanisms, or combinations of mechanisms, that have the greatest impact. Indeed, a central contribution of fundamental cause theory (FCT) is that population health researchers observe the “net” effect of SES on health via “massively multiple mechanisms” (Freese and Lutfey 2011: 69). Critically, all that is required to sustain the relationship is that net effect is consistently positive over time and place, and across many changing mechanisms and outcomes; i.e., not that all of the mechanisms linking education to health and mortality

have to follow the same causal direction or have the same causal impact (Lutfey and Freese 2005; Freese and Lutfey 2011). This point is best illustrated in Lutfey and Freese's (2005) ethnographic investigation of *how* FCT is observed in the context of diabetes – a key source of cardiometabolic morbidity and mortality in the United States. One of their key findings is that better adherence and outcomes among high-SES diabetic patients is a function of multiple systemic advantages, given there were many instances of *both* low-SES and high-SES patients failing to follow through with management or care protocols. Along these lines, a recent report from the NCHS finds that the percentage of U.S. adults regularly consuming fast food *increased* based on their income level (Fryar et al. 2018), even though rates of obesity and poor cardiometabolic health are generally lower among more affluent adults (O'Rand and Lynch 2018). Perhaps most broadly, Cockerham's (2005) framework for the formation of "health lifestyles" across different socioeconomic groups theorizes that not all of the behaviors and beliefs embodied by higher-status individuals are universally salubrious on account of both class- and individual-based preferences.

In the case of this study, the examination of heterogeneity and identification of distinct groupings of body size and cardiometabolic health helps identify particular clusters of concern contributing to educational disparities in mortality. Just as there is heterogeneity in the groupings of body size and cardiometabolic health, there is heterogeneity in how these risk profiles are represented across educational groups. College-educated individuals have a more favorable proportion of the "Ideal" type and a lower proportion of higher-risk classes, reaffirming the fact that the health-advantages enjoyed by higher-SES adults are not limited to a single source of risk, or even multiple measures (Link and Phelan 1995; Phelan et al. 2010). However, cardiometabolic health is fairly evenly distributed among intermediate risk profiles; while this does not quite rise to the level "countervailing mechanisms" suggested by Lutfey and Freese (2005), the more equal representation likely mitigates the percentage of educational disparities in mortality attributable to this set of body size-related and cardiometabolic risks. Namely, higher-educated adults are not immune from poor health across a broad set of cardiometabolic health profiles, having prevalence on par with their lower-educated counterparts. More broadly, this reinforces the notion that

individuals' SES does not make them a homogenous group in all aspects of their health and wellbeing (Cockerham 2005).

Empirically speaking, the differential impact of the latent classes in these analyses can help explain some of the inconsistencies in past research on the role of obesity and other cardiometabolic risks as mediators of socioeconomic disparities in health and mortality. More critically, knowledge of the contribution of these intermediate risk profiles to observed disparities can be instructive in identifying which risk profiles should be prioritized for intervention. FCT's core argument is that we can achieve the broadest possible impact on improving population health by intervening on distal determinants of health like education; yet, the practical reality is that meso-level processes and mechanisms continue to be the focus of many population health initiatives (Goldberg 2014). In turn, better knowledge of which mechanisms merit the most concern can be informative, even if these approaches do not represent the desired macro-level intervention. Indeed, Phelan et al. (2010) recognize that that the focus on addressing fundamental causes of mortality need not come at the expense of understanding and intervening on intermediate mechanisms; rather, they stress that these intermediate mechanisms need to be targeted in a way that does not further exacerbate social disparities in health and mortality.

Consequently, this study addresses the issue of what it means to focus solely on obesity in the hopes of mitigating educational disparities in mortality, given that obesity and poor cardiometabolic health do not co-occur identically across different levels of educational attainment. In turn, these different groupings of body size and health do not equally contribute to the educational gradient in mortality; this may be less apparent in the case of all-cause mortality, but it is clearer when examining disparities in heart disease-related deaths, and those where hypertension or diabetes are contributing causes. Among the different latent classes of body size and cardiometabolic health where obesity is a defining attribute, the group represented by both obesity and high cardiometabolic risk accounts for a majority of the disparities in mortality. Conversely, the fact that the "Fat but Fit" class accounts for a smaller share of mortality disparities suggests that obesity in and of itself is a more limited concern when it comes to disparities. The more "medium" level of risk associated with obesity in the Mixed Health group accounts for a larger

proportion of disparities, but is still less than the “High Risk” group for those with a high school education or less. Though its contribution varies across different levels of educational attainment, the Mixed Health without Obesity group accounts for a greater proportion than of disparities than the “Fat but Fit” group (and the Mixed Health without Obesity group in some cases), but might be neglected given a singular focus on obesity as the driving mechanism. These results underscore that contextualizing population health risks is important: a relatively small group of high-risk adults – accounting for only 10% of adults as a whole, and 5-15% across educational groups – accounts for the majority of educational disparities in mortality in this study. Yet the contribution of intermediate risk groups, representing different combinations of risk factors, is more variable. This confirms, and potentially helps explain, mixed findings from extant research suggesting that not all cardiometabolic risks have the same explanatory power when it comes to educational gradients in health (Dégano et al. 2017; Dowd and Goldman 2006; Kershaw et al. 2013; Kim et al. 2018).

Returning to the question of fundamental causes, it is clear that the reducing educational disparities at a societal level would be the most effective intervention in reducing social disparities in mortality. Though looking at individual mechanisms is informative, the results of this study ultimately reaffirm the motivating principles of FCT, as even this broad set of body size and cardiometabolic measures only explains ~15% of educational disparities in mortality risk. This serves as an important reminder of how even a more holistic or multi-systems view of health does not account for numerous other risks shaping social disparities in health. Even with causes of death more tightly-linked to cardiometabolic health, one can imagine numerous unmeasured, subclinical, and accumulated stressors that account for increased mortality (Gutin 2020), as posited by the large sociological body of literature on individuals’ SES and the overall burden of stress in their lives (Elo 2009; Lantz et al. 2005; Mirowsky and Ross 2003).

Moreover, it is clear that no single combination of body size and cardiometabolic mechanisms accounts for the entirety of the mediation, as adults with a college education tend to have lower mortality on account of a more favorable profile across all latent classes. Examining these intervening mechanisms

demonstrates how certain pathways – such as the High Risk minority – might take precedent over a broad-based approach focused on obesity as the single source of social disparities in mortality. Population health researchers interested in disparities might draw on recent research identifying “super-utilizers” in health care, wherein a very small proportion of patients accounts for a disproportionate amount of healthcare spending (Aldrige and Kelley 2015; Johnson et al. 2015; Mitchell 2020). Efforts to identify and directly target this group via social programs and services has been successful in both lowering costs and improving overall population health (Kangovi and Grande 2020); this may have implications for targeting especially high-risk populations with the goal of reducing disparities. More generally, however, the key takeaway is recognizing that obesity is not the only mechanism by which we observe poor cardiometabolic health among U.S. adults, regardless of their level of education. Thus, when evaluating the target or outcome of a given intervention population researchers and policymakers should invest in a more holistic understanding of how individuals’ health has changed, given that a focus on body size can be misleading and/or incomplete.

### *Limitations*

Prior to concluding, it is important to note the limitations of these analyses and how they may be addressed in future research. First and foremost, there are valid concerns related to potential sources of measurement error in two respects. One important source of measurement error pertains to the various indicators of body size and cardiometabolic health used in the analysis. Earlier, I discussed the similarities between LCA and confirmatory factor analysis (CFA), wherein a key distinction is an emphasis on individuals rather than the measures themselves (Bauer and Curran 2004). In this study the choice of LCA is strategic in identifying meaningful groupings among respondents, as befitting the research aims. However, a key limitation of LCA relative to CFA is that the focus on individuals does not allow researchers to correct for measurement error in the individual measures themselves by modeling them as indicators of a shared latent construct. Even though most of the NHANES measures used in the analyses are based on examination data collected by trained professionals using validated methods and techniques,

there is still likely to be measurement error from random sources – such as user error or poorly calibrated instruments – that is not accounted for (Bollen 1989: 151-178).

A secondary source of measurement error is in the assignment of individuals to specific latent classes. Making the latent class an ‘observed’ variable in the analysis requires a strong assumption of high reliability in classification (i.e., minimal classification bias [Clark and Muthén 2009]). Researchers have developed methods to estimate measurement error in assignment, and then incorporate this uncertainty into the subsequent regressions using the latent classes as variables (Asparouhov and Muthén 2014); unfortunately, these methods are not validated for survival analysis and lead to convergence issues in a sample as large as the NHANES. The high entropy and AvePP for the five-class solution in this study mitigates these concerns – as does the robustness check based on the four-class solution and alternate specifications of measures – but they cannot be entirely discounted. Based on past research, the estimates and standard errors presented in these analyses may be conservative due to misclassification bias (Bolck et al. 2004).

Relatedly, one may expect some variation in the results based on the choice of measures used in the LCA, with respect to both the types of measures included and how they are categorized in terms of risk. In the present study, different combinations of variables and cutoffs did not alter the substantive latent classifications; thus, I used a set of variables and cutoffs exhibiting the best classification criteria and separation among respondents. A dataset as rich as the NHANES provides numerous other biomarkers and self-reports that have been used in past research on socioeconomic disparities in health (Dowd and Zajacova 2009); future research may consider how including an even broader set of measures affects the conclusions. However, researchers should be careful in providing some theoretical and/or empirical basis for how and why they might expect the various measures to co-occur, especially in making arguments about the interpretability and validity of the resulting latent classes.

Further work with more recent restricted-use mortality data would allow researchers to examine additional cause-specific analyses and better address issues of temporality, given the many period- and cohort-based influences on obesity, cardiometabolic risk, and their associations with mortality (An and



Xiang 2016; Bell and Jones 2014; Keyes et al. 2010; Masters et al. 2013; Reither et al. 2009; St-Onge et al. 2010). With public-use data I was limited to the ten broad categories of leading causes of death in the United States, along with deaths where hypertension or diabetes are contributing causes. As demonstrated in the results, the cause of death under consideration greatly influences the mortality risk associated with classes as well as their estimated mediation of educational disparities in mortality. Critically, a longer mortality follow-up period would help address some of the small cell sizes that produce such unstable estimates. This would then facilitate additional group-specific analyses and/or looking at trends over time, as would be of interest across this broad range of years. As noted, the intersection of gender and/or race/ethnicity with education is a key area of future work. Past work consistently documents lower educational returns to health for both women and non-White adults, with a particularly notable disadvantage among non-Hispanic Black females (Borrell et al. 2010; Geronimus et al. 2006; Geronimus et al. 2010; Hargrove 2018; Levine and Crimmins 2014). The most appropriate course of action would be to conduct a more systematic examination of group differences, estimating separate latent class models across these different categories of gender and race/ethnicity. In addition to better understanding the groupings of body size and cardiometabolic health that describe these demographic groups, we would also gain valuable insight on group differences in which latent groups appear to have the greatest explanatory power when it comes to SES gradients in mortality.

Finally, on a more abstract level, this study represents one of many approaches researchers could use in trying to add nuance to our understanding of the relationship between body size and health. Namely, proponents of HAES might argue that even this multimorbidity, multi-systems view of body size and health provides limited insight on individuals' experiences and understanding of their health as a function of their body size. Even if LCA captures important nuances in how body size and cardiometabolic health co-occur, individuals are still being categorized on the basis of health, thus obscuring additional levels of heterogeneity. There are also many aspects of individuals' health and wellbeing that are not addressed in these data, such as key dimensions of psychosocial and emotional health that also factor into a HAES-based understanding of how our bodies affect our health (Bombak

2014; Penney and Kirk 2015). Thus, in arguing that I provide importance evidence of variation in how body size and health co-occur, I concede that this variation may not rise to the level of nuance advocated for by the HAES framework, or by other scholars seeking to diminish the categorical power afforded to BMI and obesity as measures of health.

### *Conclusion*

The growth of biosocial data in the past 20 years provides researchers with a novel opportunity to use a systems-level approach in the study of population health (Seeman et al. 2004), integrating multiple dimensions and measures of health to attain a better and more comprehensive understanding of underlying risk (Harris 2010). Indeed, the *lack* of a more integrative approach has been a key issue in the study of obesity, wherein the fairly imprecise measure of BMI – and the corresponding categorization of obesity – provides limited insight on individuals’ overall health. This is not to suggest that BMI and obesity are uninformative in understanding population health and identifying important social disparities; rather, orthodoxy in their use as unequivocal measures of overall health often biases researchers, policy makers, and the public at large from adopting a more comprehensive view of the full spectrum of individuals’ cardiometabolic health (Jutel 2011; Saguy 2012; Shugart 2016). Fortunately, the aforementioned availability of innovative data and methods has allowed researchers to document the complexity and multidimensionality of population health, as seen in recent work on metabolically-healthy obesity, HAES, and in the broader call for understanding heterogeneity in the relationship between body size and health.

Thus, rather than continuing to *substantiate* a biomedical view of obesity as a homogeneous risk, population researchers have unprecedented access to the kinds of rich biosocial data and novel methodological tools that allow them to *challenge* and *improve* the conceptualization, definition, and measurement of health and healthiness at the population level. As this analysis shows, by situating obesity among many other indicators of cardiometabolic health, researchers can employ a more comprehensive and holistic systems-level view that allows for a better understanding of the nuances in how body size and

cardiometabolic risk are distributed across the population, and where we might best direct efforts to improving health and reducing disparities.

Indeed, this chapter underscores the utility of these multi-systems, multimorbidity approaches when examining the *social* patterning of health. Applying a sociological and social demographic lens to the same questions explored in clinical and epidemiologic research on body size and cardiometabolic health shows that the different combinations and ‘phenotypes’ observed throughout the population are not random – as befitting biological or genetic perspectives – but instead highly stratified based on social factors, like education. In turn, we obtain a better understanding of *which* health risks help explain educational disparities in mortality, and identify combinations of risk factors that merit greater attention. Moreover, this multi-systems perspective and analytic approach provides empirical support for extant sociological concepts like fundamental cause theory, demonstrating how the multiple mechanisms connecting social determinants to disparities in mortality work in concert with one another to shape individuals’ health. Critically, while fundamental cause theory emphasizes the upstream determinants of health, the arguments it makes about meso-level processes have important implications as well by highlighting how not all of the mechanisms linking education to mortality have the same impact. In turn, this knowledge can and should be leveraged to better understand where the limited time and resources available for intervening on population health should be directed.

More broadly, this study contributes to the growing body of research advocating for a more comprehensive approach to studying population health in a world where multiple risks and conditions simultaneously influence premature aging and mortality (Belsky et al. 2015; Moffitt et al. 2017). Defining health on the basis of individual measures, and along strict binaries of healthiness and unhealthiness, continues to provide important population health knowledge; however, there is more to be learned in

broadening these definitions. Leveraging all available tools and methods facilitates a better understanding of not only the full spectrum of health, but also how individuals' social attributes and environments influence where their health is located along this spectrum.

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## **CHAPTER 4: DOUBLE JEOPARDY: PATHWAYS OF OBJECTIVE AND SUBJECTIVE WEIGHT STATUS THROUGHOUT THE LIFE COURSE AND HEALTH IN ADULTHOOD**

### **Introduction**

Since the early 1960s, the mean weight of U.S. adults has increased by more than 24 pounds and obesity rates have more than tripled for men and doubled for women, with much of the growth occurring in just the last three decades (Fryar et al. 2012). At present, over one-in-four of the U.S. adult population is considered to have obesity (Hales et al. 2020), with some projections suggesting half of the population will have obesity by 2030 (Finkelstein et al. 2012). Most worryingly, the rise in obesity among adolescents and young adults has been especially pronounced, with obesity rates quadrupling among those ages 12-19 since the 1980s (from ~5% to 20%) – a growth rate higher than any other age group (Ogden et al. 2014; Ogden et al. 2016). High and raising rates among more recent cohorts of U.S. adults raise well-founded concerns about reductions in life expectancy and quality of life for future generations of Americans (Jia and Lubetkin 2010; Olshansky et al. 2005; Reither et al. 2011), many of whom may experience the cumulative effects of having spent the majority of their lives living with obesity (Ferraro and Kelley-Moore 2003; National Academies of Sciences, Engineering, and Medicine [NASEM] 2021).

While no single factor is responsible for the sharp increase in adolescent and adult obesity, its rapid onset underscores the urgency of understanding potential precursors to obesity and related diseases. One line of recent inquiry has emphasized the critical role of individuals' "self-perception" of their weight as a potential explanatory and/or mediating factor underlying obesity and health outcomes. Researchers posit individuals' satisfaction with their weight (i.e. an individual's belief that their current weight is appropriate, or just right, or does not require change) influences the degree to which they perceive excess body weight as "unhealthy", thus mitigating the extent to which they then view their overall health and wellbeing as a function of their weight status. The misperception of weight (i.e.,

“discordance between an individual's actual weight status and the perception of his/her weight status” [Duncan et al. 2011: 2]) can lead individuals to downplay or be unaware of the consequences of excess body weight, giving rise to an ongoing cycle of weight gain.

Indeed, a growing body of evidence demonstrates that individuals’ perceptions of themselves as “overweight” do not necessarily align with their “clinical” weight status on the basis of body mass index (Burke et al. 2010; Chang and Christakis 2003; Maximova et al. 2008; Robinson and Kirkham 2014). These findings are consistent with previous research on subjective evaluations of overall health, as reports of “poor” or “excellent” self-rated health are not necessarily concordant with more objective indicators of individuals’ health on the basis of diagnosed conditions, limitations, and measured physiological impairment (DeSalvo et al. 2006; Dowd and Zajacova 2010; Franks et al. 2003; Garbarski 2016; Layes et al. 2012). Yet, the same benefit of doubt granted to self-rated health as an independent and valid measure of health has not been extended to self-perceived weight. Researchers frame the ambiguity and uncertainty in the measurement of self-rated health as one of its key strengths; namely, self-rated health has strong predictive validity for health outcomes net of “objective” measures of health, as it potentially captures unmeasured (or unmeasurable) aspects of health and other experiences that are implicitly factored into individuals’ subjective evaluations (Huisman and Deeg 2010; Jylhä 2009; Jylhä 2010). The aforementioned objective-subjective discordance is not framed as an issue of incorrect reporting, but instead an acknowledgement that subjective and objective measures can represent different dimensions of health, complementing one another in trying to account for the complexity of health as a multi-dimensional and holistic construct.

Critically, subjective measures speak to the value of understanding the *experiences* associated with health and the body, which are distinct from the direct physiological processes underlying health and disease (Lupton 2012). Much like with self-rated health, perceiving oneself as overweight can provide valuable insight on the psychosocial insults associated with the stress due to having a negative self-image or being stigmatized on the basis of one’s physical appearance (Puhl and Huerer 2009; Puhl and Huerer 2010), especially in a highly weight-conscious and body-normative society like the United States

(Greenhalgh 2015; Oliver 2006; Saguy 2012). This is a particularly important issue with respect to sociological theory on the body and what it *represents* in a given social context (Bourdieu 1984); bodies are defined both biologically and socially (Fox 2012), often as a function of what they do or what individuals assume they can do. Considerable social meaning is projected onto body weight and size and what it communicates about a person's health status (Jutel and Buetow 2007; Shugart 2016), beyond their physiological implications for health.

Indeed, there is increased recognition of body size as a key axis of inequality in a weight-conscious society like the United States (Gutin 2021); “overweight” – as an identity – is one of the earliest examples cited in foundational research on stigma (Cahnman 1968; Maddox et al. 1968), and continues to be a socially acceptable form of bias (Puhh and Heuer 2010). In turn, stigma is a fundamental cause of health (Hatzenbuehler et al. 2013), wherein the aspect of body weight as an embodied social identity, and social source of stress, can be uniquely consequential. Thus, subjective weight might be highly predictive or better associated with certain health outcomes than objective weight in capturing individuals' lived experience of weight – such as poor psychosocial health stemming from institutional and interpersonal discrimination (Hatzenbuehler et al. 2009; Papadopoulos and Brennan 2015; Puhl and Heuer 2010; Puhl and Suh 2015; Schafer and Ferraro 2011). This may be especially pertinent at younger ages, where the more “objective”, (pre)disease-related symptoms and evidence of poor weight-related health have yet to manifest (Altman et al. 2016), or are largely subclinical.

Thus, rather than focusing on discordance in the objective and subjective reality of weight, it is important to recognize how these measures reflect body weight as both a physical and social identity, with differing implications for individuals' health. Such is the focus of this study, which seeks to better understand the relationship between these two dimensions. Both are likely subject to unique trajectories, potentially categorized by different processes of stability and change, requiring systematic examination. Rather than simply noting the binary occurrence of “misperceptions,” this work shows how subjective and objective weight influence one another in complex and dynamic ways. Consequently, this study uses longitudinal data from adolescence to adulthood and structural equation modeling with latent variables to

assess the measurement and predictive properties of subjective weight, as well as its associations with objective weight and health. Specifically, the research questions addressed in this project are twofold:

- (1) How do subjective and objective weight affect one another over time? Although prior research finds considerable discordance between perceived and measured weight, this neglects the possibility that subjective and objective weight influence one another in complex ways over time. There may be cross-lagged effects between these measures, as well as cross-sectional associations and correlations, potentially across all points in the life course or only at specific points in time. Identifying an appropriate longitudinal trajectory will also allow for better models in subsequent analyses.
- (2) What is the influence of both subjective weight and objective on later life health outcomes? Having identified the parallel, and potentially intersecting, trajectories of subjective and objective weight, I assess the extent to which they are both predictive of later life health outcomes – demonstrating how both the subjective experience of feeling overweight and the physiological condition of being overweight represent distinct lifelong “exposures” that are associated with physical and mental health in adulthood.

Given the highly gendered-nature of weight identity and body image in the United States (Fikkan and Rothblum 2012; Puhl et al. 2008 Saguy 2012), I also compare the overall results to gender-stratified models to see if the trajectories, and their associations with health outcomes in adulthood, differ between female and male respondents.

The study begins with an overview of research on weight perception in the context of the U.S. obesity epidemic, the role of subjective measures in health research, and emerging work on individuals’ perceived weight as a distinct construct separate from objective body weight and size. I then proceed with a detailed description of the data and methods used in the analyses, explaining the step-wise approach to model building and comparisons used throughout the analysis. Following the results, I discuss the implications of the findings for knowledge of subjective and objective weight over the life course and how they relate to adult health. I close by emphasizing the importance of distinguishing between

subjective and objective dimensions of health in population research, especially when studying body weight size as both a physical characteristic and social identity.

## **Background**

### *Weight Perception and the Obesity Epidemic*

Many studies point to steady changes in individuals' standards for a "normal" body weight as a potential explanation for the dramatic increase in obesity from the 1980s onward, suggesting that individuals' accuracy in self-perception of their weight has been negatively influenced by increases in average body size across the communities and social networks in which they are embedded (i.e. social comparison framework: Burke and Heiland 2007; Christakis and Fowler 2007; Wedow et al. 2018). Empirical work testing the theory that increases in average body size have led to more individuals viewing their weight as "just right" rather than "overweight" finds evidence in favor of a "generational shift in social norms related to body weight... such that people may be less likely to desire weight loss than previously" which has potentially "limit[ed] the effectiveness of public health campaigns aimed at weight reduction" (Burke et al. 2010: 1226).

Indeed, the "misperception" of overweight – i.e., believing oneself to be "normal" weight when, objectively, one's BMI puts them in the overweight or obese categories – is negatively associated with weight management and key health-promoting behaviors like eating healthy foods and performing physical activity (Duncan et al. 2011). This discordance has been implicated as a significant barrier to many interventions aiming to raise awareness about obesity as a health issue and triggering more positive health-related decision-making (Johnson-Taylor et al. 2008; Kuchler and Variyam 2003), as the individuals targeted by these interventions may not perceive themselves as unhealthy to begin with.

Moreover, the aforementioned "generational shift" in attitudes towards weight suggests that adolescence and early adulthood represent critical points in the life course for better understanding the effects of weight perception on future weight and health. These early life attitudes and feelings towards weight and health are often the foundation for weight-related behaviors and beliefs well into later life (Bauldry et al. 2012; Harris 2010; Harris et al. 2006), as is shown in past research examining the



persistence of obesity and other weight-related behaviors from adolescence into adulthood (Laska et al. 2012; Nelson et al. 2008; Viner et al. 2012). Individuals' early perceptions of their weight may be particularly influential in crystallizing certain assumptions about the relationship between weight and health that persist through their adult years.

However, the lack of many "objective" measures of physiological dysregulation at younger ages suggests that subjective assessments or perceptions of health are particularly valuable indicators of poor health and wellbeing (Bauldry et al. 2012; Boardman 2006; Sokol et al. 2017). This is an especially key issue among younger adults, given that the most serious and obvious health consequences of excess body weight and obesity (e.g., chronic disease and disability) often do not manifest until later life (Altman et al. 2016; Zajacova and Burgard 2010). Thus, rather than exclusively focusing on individuals' subjective weight status as an obstacle in health promotion efforts, medical, epidemiologic, and public health research could stand to benefit from a better understanding of subjective weight as a meaningful measure in and of itself.

### *Subjective Measures in Health Research*

Researchers valorize subjective reports of health for their parsimony and predictive power, summarizing complicated and potentially unobservable health processes that cannot be comprehensively documented in survey research due to "practical limitations of empirical studies, or... the inadequacy of our present knowledge to appropriately measure these aspects" (Jylhä 2009: 312). Subjective or perceived measures of health have shown considerable predictive value in social and health research over past decades, as most clearly evidenced by the large body of work using self-rated health or similar assessments of general health. Namely, individuals' self-perceptions of their health status are strongly associated with a greater frequency and probability of numerous health outcomes, including medical care, disability and functional ability, physiological dysregulation, chronic diseases (such as coronary heart disease, diabetes, stroke, lung disease, arthritis, and cancer), and premature mortality across multiple causes of death (Benamins et al. 2004; DeSalvo et al. 2006; Dowd and Zajacova 2010; Goldman et al. 2004; Idler and Benyamini 1997; Halford et al. 2012; Idler and Kasl 1995; Jylhä et al. 2006; Latham et al.

2012). In nearly all studies, the strong associations between negative assessments of one's health and negative health outcomes persist even after accounting for more detailed questions about one's health and various morbidities, physician evaluations, and, increasingly, direct biophysiological measures of health.

More narrowly-focused subjective assessments of health – such as self-rated mental health (Fleishman and Zuvekas 2007; Lee 2000) and oral health (Benyamini et al. 2004) – have similar predictive properties, lending further empirical support to the utility of subjective and more 'holistic' assessments of health in survey research. Even perceived physical activity has been documented as a significant predictor of premature mortality (Zahrt and Crum 2017). This growing body of research has spurred researchers to think more critically about the relationship between subjective assessments of health and the objective health conditions that they are proposed to reflect. While primarily focused on better understanding the predictive power of self-rated health, emergent theories provide a much broader framework for recognizing the complex social, psychological, and biophysiological processes underlying subjective measures of health.

Namely, extant theory and research underscores the importance of conceptualizing subjective reports as *separate* health constructs, rather than just reports of the "true" objective health they supposedly measure (Goldman et al. 2004; Huisman and Deeg 2010; Jylhä 2010; Layes et al. 2012; Quesnel–Vallée 2007). As Quesnel–Vallée (2007) notes, much of the research on self-rated health operates "under the broad assumption that 'true' health is defined as the absence of diseases and especially those that are life-threatening," which often implies that "'true' health is equated with objective measures of health" (p.1161). However, the complexity and multidimensionality of individuals' health suggests that this idea of 'true' health is "a non-existent, impossible, ultimate, total entity" and thus not very useful in seeking to unpack subjective assessments of health (Jylhä 2010: 657). Consequently, researchers are better-served by shifting the focus away from trying to "validate" subjective measures against their objective counterparts (Huisman and Deeg 2010), and toward recognizing perceptions of health as "a valid measure of *those aspects of health* that are related to the likelihood of survival and mortality" (Jylhä 2010: 657, emphasis mine), or positive health and well-being more broadly. As Bombak

(2013) notes, “individuals are capable of recognizing their own state of wellbeing, regardless of whether this reflects the views held by practitioners and researchers” (p.2); thus, an overwhelming focus on discrepancies or inaccuracies in subjective and objective health is inappropriate, as it implicitly expects that “individuals... rate their health according to others’ standards, identify deficiencies, and correct their behaviors to achieve “better” health” (p.2), rather than consider the individual merits of these subjective measures.

### *Perceived Weight as a Distinct Construct*

Even though subjective weight status has been less thoroughly examined in extant theoretical and empirical research on subjective health, the arguments presented above extend to individuals’ self-assessments of their weight. To date, relatively few studies have assessed the predictive power of subjective weight on future health outcomes. Recent work by Daly et al. (2017) and Unger et al. (2017) challenges the assertion that “misperception” of one’s weight is necessarily harmful to one’s health, shifting the focus to negative self-assessments of one’s weight as having a negative impact on health independent of objective weight. Specifically, both studies find that perceiving oneself as overweight – irrespective of the accuracy of this assessment – is associated with a significant increase in worse subjective health, depressive symptomology, and a broad set of indicators representing physiological dysregulation, including blood pressure, C-reactive protein, waist circumference, the ratio of total blood cholesterol to levels of high-density lipoprotein cholesterol, total triglycerides, glucose, and glycated hemoglobin (Daly et al. 2017: 877; Daly et al. 2019; Frisco et al. 2010; Haynes et al. 2019; Unger et al. 2017).

These studies, as well as reviews of the physiological and psychological health consequences associated with negative perceptions of weight, increasingly point to the deeply stigmatizing aspects of being “overweight” in contemporary society as a driving force underlying the association between high body weight and poor health (Daly et al. 2019; Haynes et al. 2018; Robinson et al. 2017; Tomiyama et al. 2018). Having “knowledge that you possess a characteristic devalued and derogated by society is likely to be psychologically damaging”, leading to deterioration in one’s mental and physical health (Robinson et

al. 2017: 1160). Thus, “the stigma attached to identifying as being a person with overweight or obesity may ironically exacerbate these conditions” (Robinson et al. 2017: 1160), as it engenders stress and continued weight gain, calling into question the efficacy of clinical and public health messaging intended to raise awareness about one’s weight status.

Past studies have suggested that this stigma is especially consequential for girls and women, given the highly-gendered environment for body size and beauty norms in the United States, and many other societies (Fikkan and Rothblum 2012; Puhl et al. 2008). Namely, the “thin ideal” type for bodies and health is most directly applicable to young women, while there is more flexibility in the range of body types and sizes that is considered “acceptable” for men (Bordo 2004; Grogan 2007). For instance, women are far more likely to express dissatisfaction with their weight throughout the entirety of the life course, such that concern about body image does not begin to attenuate until they are elderly (Tiggemann 2004). Thus, the stigma attached to having the “wrong” body or weight – or *feeling* like you have the wrong body size – is likely to be exacerbated among women, who are regularly exposed to messages in mass media, advertising, and popular culture promoting a body image valuing thinness and low weight (Arciszewski et al. 2012; Bordo 2004; Homan et al. 2012).

The psychological aspects of one’s weight-related health are likely to be particularly influential in early life and into adulthood. Body weight-related stigma is commonplace in contemporary society, manifest as chronic discrimination and bias against overweight individuals who experience social ostracism, verbal and physical abuse, bullying, harassment, and the internalization of this negative self-imagery due to their bodies not conforming to social and medical standards for “healthiness” or “normality” (Bucchianeri et al. 2013; Durso and Latner 2008; Lewis et al. 2011; Puhl and Brownell 2001; Puhl and Brownell 2006; Puhl and Heuer 2009; Puhl et al. 2007). Many researchers in this area emphasize the impact of these chronic insults on youth, who experience some of the harshest encounters with size-based discrimination on a daily basis (Puhl and Latner 2007); moreover, poor mental health is likely to be the earliest “symptom” of poor health associated with one’s weight, prior to the onset of either physical or functional declines. Indeed, the *cumulative* toll of weight-based stigmatization and

discrimination throughout the life course may be particularly damaging (Puhl 2011), as implied by the strong association between weight-related stigma and premature mortality (Sutin et al. 2015). As noted above, rigid and gendered standards for body size throughout the life course help explain why these negative health outcomes – especially with respect to psychosocial and mental health – are consistently more pronounced among girls and women (Ciciurkaite and Perry 2018; Frisco et al. 2010; Hilbert et al. 2014; Puhl et al. 2008; Yuan 2010).

Thus, subjective weight constitutes an important predictor of future health outcomes, representing key psychosocial mechanisms independent of objective weight. The aforementioned studies by Daly et al. (2017) and Unger et al. (2017) support this claim, albeit limiting the scope of subjective weight's predictive validity to its influence on psychosomatic mechanisms related to weight stigmatization and (paradoxical) weight gain. These mechanisms are important to emphasize in a society where body weight and size are heralded as measures of one's health and moral character (Brownell et al. 2010; Mata and Hertwig 2018; Oliver and Lee 2005; Saguy and Gruys 2010; Saguy and Riley 2005). Indeed, there is compelling evidence that psychosocial mechanisms constitute some of the *primary* pathways through which individuals' body size negatively impacts their health (Pearl and Puhl 2018; Puhl et al. 2020; Tomiyama et al. 2018); in turn, studying subjective and objective weight as separate constructs can help distinguish these important physiological and psychosocial mechanisms.

#### *Limitations of Past Longitudinal Research on Perceived Weight*

On a final note, while the research discussed above is important in acknowledging the experience and perception of one's weight as an important aspect of health, this work has not explicitly acknowledged the issue of measurement error in subjective weight – especially as it relates to individuals' trajectories of subjective weight over time. First and foremost, subjective weight status is undoubtedly susceptible to measurement error, as individuals' feelings about their weight are shaped by numerous contextual factors in their social environments that influence their responses at a given point in time (Wedow et al. 2018). Secondly, an outstanding issue in the subjective health literature is the extent to which subjective measures, such as self-rated health, reflect a relatively “stable” baseline assessment of

health that varies across individuals as compared to a more dynamic assessment of health that changes with respect to “new” information about health (Bailis et al. 2003; Boardman 2006; Bollen et al. 2021; Dowd and Zajacova 2011; Huisman and Deeg 2010; Jylhä 2009). Often framed as an issue of “reliability,” a number of studies have examined the extent to which subjective assessments of health vary over time despite “objective” changes to individuals’ health, finding support for both static and dynamic processes indicative of individuals “self-enduring” conceptualization of their health, as well as more “spontaneous” assessments based on near-present circumstances (Bailis et al. 2003; Boardman 2006; Dowd and Zajacova 2011). Recent work suggests that more complex processes may be at work, wherein these subjective measures are governed by multiple longitudinal processes operating in tandem with one another (Bollen and Gutin [Forthcoming]).

This simultaneously dynamic and stable conceptualization of health is most effectively documented in research on self-rated health, yet extant research suggests similar processes with respect to individuals’ perceptions of their weight. Scholars have written extensively on how individuals’ life and health experiences lead to the formation of certain “health identities”, and even “weight identities”, which are fairly static over time and have a strong influence on specific health beliefs and behaviors throughout the life course (Blaxter 2004; Fox and Ward 2008; Sobal and Maurer 2017; Whyte 2009). While few studies have empirically assessed these theories, Wedow et al.’s (2016) study on adolescent and young adult weight identity finds evidence of both stability and change. Namely, a large proportion of the variance in subjective weight status can be explained due to differences and changes in individuals’ objective weight; nevertheless, a significant proportion of the stability in subjective weight remains unaccounted for, strongly suggestive of stable genetic or “heritable” traits influencing this measure, especially among females. Thus, identifying the appropriate longitudinal model for perceived weight is critical for understanding its association with both objective weight and later life health.

## **Data**

Data for this project come from the National Longitudinal Study of Adolescent to Adult Health (Add Health), a nationally-representative survey of adolescents (grades 7-12) who, along with their

parents, were initially interviewed in 1994-1995, with additional respondent interviews in 1996 (Wave II: grades 8-12), 2001-2002 (Wave III: ages 18-26), 2008 (Wave IV: ages 24-32), and 2016-2018 (Wave V: ages 32-42). A key strength of the initial study design is its use of a complex, stratified sampling strategy that accounts for the region, urbanicity, size, type, and racial composition of schools from which students were recruited, thus maintaining the national representativeness of the data at the initial wave and through the follow-up (Harris et al. 2019). Further, Add Health is ideally suited for examining both the measurement properties of subjective weight and its associations with other aspects of health; while questions about individuals' perceptions of/feelings about their weight are relatively common in survey research (e.g., National Health and Nutrition Examination Survey, Jackson Heart Study), they are far less frequent in longitudinal data sets that span critical points in the life course (e.g., National Longitudinal Survey of Youth 1997 [NLSY97]), let alone in conjunction with many other measures of health in adulthood, as is crucial for this analysis. Additionally, Add Health provides longitudinal survey weights that ensure the sample is representative of U.S. adults in this cohort, while accounting for attrition over time.

Individuals' subjective weight status is the focal variable in this project, based on respondents' answer to the question, "How do you think of yourself in terms of weight?", with "very underweight", "slightly underweight", "about right", "slightly overweight", and "very overweight" as possible options. This measure is asked in the first four waves of Add Health, providing a comprehensive history of individuals' perceptions of their weight over nearly 20 years and across multiple important stages in the life course. I treat subjective weight as a continuous measure, influenced by an underlying continuous latent variable of perceived weight. Past research suggests that this is a plausible assumption for a five-category ordinal measure (Rhemtulla et al. 2012), especially as it facilitates easier estimation in what are already demanding models; however, I make sure to test alternate specifications in case the choice of measurement introduces bias. Given the focus on overweight and obesity in health research, more positive values indicate a greater propensity to view oneself as overweight (i.e., subjective weight). Thus, in the

first part of the analysis, I focus on establishing the measurement properties of subjective weight as indicative of stability or change in individuals' latent beliefs about their current body weight.

Though I return to this in the limitations, I would be remiss to ignore the large body of literature in psychology examining body image, body satisfaction, and weight bias or stigma, often using multi-item scales to capture different dimensions of these complex latent constructs (Lillis et al. 2010; Sandoz et al. 2013). The perceived weight measure used in these analyses (or a close analogue) can be found in these scales (Durso and Latner 2008), suggesting that these perceptions are reflective of multiple latent constructs surrounding one's body, weight, and how individuals feel about it. While perceived weight is an imperfect measure, it is the best available option in a longitudinal, nationally-representative data set like Add Health; thus, in referring to subjective weight, I focus on perception and subjective evaluation, but consider its implications for broader constructs like body image, body satisfaction, and weight bias or stigma.

I also account for changes in individuals' trajectories of "objective" weight, in estimating how much of the variation in subjective weight is based on actual changes in body size over time. In defining "objective" weight, I use the established measure of individuals' body mass index (BMI:  $\text{mass}[\text{kg}]/\text{height}[\text{cm}]^2$ ). The focus is on BMI as a relative estimate of body size – wherein higher values suggest one's weight is increasingly disproportionate to one's height – rather than using clinical categories of "normality" or "healthiness" into which BMI is sorted, as is often the case in past research on individuals' perceptions of weight. Given the large age range under consideration, a continuous measure of BMI helps reduce any additional measurement error introduced by categorization, especially with respondents still growing in the early waves of the data. BMI is collected in all five waves of Add Health, but only self-reported measures are available in Wave I when respondents are adolescents. There is evidence to suggest reporting bias based on self-reported, rather than measured, height and weight in this age group (Sherry et al. 2007); however, this bias is fairly low and this study takes steps to correct for measurement error in the analysis.



Pursuant of the second research goal, I examine the association between intersecting trajectories of subjective and objective weight and a diverse set of health outcomes. These outcomes represent a mix of both physical and mental health measures examined in past research on perceived weight and BMI; however, this study focuses on subjective and objective weight as indicative of individuals' exposures to physiological and psychosocial "stress" – both of which may be captured by the same health outcomes – rather than examining a broader set of indicators associated with all domains of health (e.g., blood lipids and blood sugar [Daly et al. 2017]). Consequently, the measures noted below emphasize this notion of underlying stress across various domains:

1. Wave V BMI: Extant research on misperception of weight implicitly assumes that individuals' accurate perception of themselves as overweight is key to instigating the kinds of weight-related behaviors that engender weight loss. However, there is evidence of a "paradoxical" relationship between perceived weight and BMI, such that individuals who perceive themselves as overweight experience *further* weight gain, possibly on account of unhealthy dieting behaviors (e.g., yo-yo dieting, where short-term weight loss precedes further weight gain) and/or other coping mechanisms brought on by the stress of overweight as a social identity (Tomiyama et al. 2018). Wave V BMI is already used in modeling the trajectory of objective weight, but I can further examine how this relationship does or does not change in accounting for subjective weight.
2. Blood pressure: Decades of research show higher blood pressure is associated with the physiological consequences of a higher body weight and the stresses this weight places on the body by requiring the heart and cardiovascular system to work harder in maintaining homeostasis (Kotchen 2010). However, higher blood pressure is also thought to be associated with the increased stress of having a negative body image or greater body dissatisfaction, stemming from the social factors noted prior. Drawing on past research, I leverage the fact that Add Health provides three reports of blood pressure to estimate a latent variable associated with both systolic and diastolic blood pressure (SBP and DBP: Bauldry et al. 2015).

3. Measured hypertension and/or taking medication: This is a constructed variable in Add Health, based on clinical cutoffs for hypertension from measured blood pressure (130/80 SBP/DBP), while also taking into account individuals' use of antihypertensive medications that can affect these measurements (Whistles et al. 2020).
4. C-reactive protein (CRP): While this is not a measure available in all data sets, it is increasingly used as a marker of "chronic" (or sustained) stress on the body, to the extent that this stress is reflected in a sustained inflammatory response (Harris and Schorpp 2018). As with blood pressure, CRP doubles as both a physiological and psychosocial marker of stress associated with objective and subjective body size, respectively.
5. Depression: Many studies have found robust longitudinal associations between depression and obesity or a higher BMI, but the proposed explanations typically favor psychosocial mechanisms (Frisco et al. 2010; Luppino et al. 2010). In using depression as an outcome, this study assumes that the subjective weight variable is likely to capture this psychosocial process identified in past research, as a function of the worse mental health typically associated with negative body image and stigma (Friedman et al. 2005; Harriger and Thompson 2012; Stevens et al. 2017). Rather than using a sum-score or index approach to measuring depression, I estimate a latent variable model based on indicators with the highest reliability (Perreira et al. 2005). Namely, this measure is constructed from reports of how many times respondents felt depressed in the last week, how many times they had the blues, and how many times they felt sad, with responses recoded as ever or rarely (0), sometimes (1), a lot of the time (2), or most or all of the time (3).
6. Diagnosed depression: As with blood pressure, I include this measure to account for any formal diagnosis of depression that may not be reflected in the previous latent measure of depression, especially if it is influenced by a diagnosis.
7. Diagnosed anxiety: Like depression, this is another common measure of mental health examined in conjunction with obesity and BMI, and yet largely explained as a function of psychosocial

mechanisms (Garipey et al. 2010). Thus, it presents another opportunity to assess the role of perceived weight – or the social aspects of overweight – rather than body size itself.

8. Sleep trouble: I include this final measure given emerging research on the importance of high-quality sleep for many of the health outcomes listed above. Individuals' quality of sleep is not necessarily a definitive "outcome" in this analysis, but it is likely correlated with the other measures and represents an important health issue in and of itself. Explanations for the association between poor sleep and obesity/BMI reflect psychosocial mechanisms as well – wherein the role of perception is once again highly salient – but there are many reasons to believe that a high body size is positively associated with trouble sleeping on account of physiological mechanisms, such as breathing difficulties. Indeed, evidence on the association between individuals' quality of sleep and obesity varies based on the measures being used (Rahe et al. 2015). In Add Health, respondents are asked how often they experience trouble sleeping, with responses recoded as never (0), less than once a week (1), one or two times a week (2), three or four times a week (3), or five or more times a week (4).

Finally, I include key sociodemographic variables in the analysis. As noted, I consider gender-stratified models, checking for configural invariance in the fit of the intersecting trajectories of subjective and objective weight between female and male respondents. I also include basic controls for respondents' age and their race/ethnicity, coded as non-Hispanic (NH) White, NH Black, Hispanic, and NH Other. Educational attainment is allowed to vary over time. Since most respondents have not yet completed their education at Waves I and II, it is coded as 0 or 1 based on whether a parent had at least a college degree. At Wave III, educational attainment is coded as 0 or 1 based on whether respondents are in college or have completed a college degree, given the mixed age range in the sample. At Waves IV and V, educational attainment is coded as 0 or 1 based on whether respondents have completed a four-year college degree. Ideally, the analysis would include more detailed categories for educational attainment, but this dichotomization was a necessary modification to avoid small variances and facilitate model convergence.

## Methods

This analysis uses structural equation models (SEM) with latent variables to assess the measurement properties of subjective weight status over time, as well as its relationship with other measures of health. Critically, by fitting a single hypothesized model that accounts for trajectories of perceived weight, BMI, health outcomes, and covariates – while accounting for measurement error in key variables – I can explicitly account for important relationships among variables that would be neglected using a more traditional OLS approach, or other methods where researchers cannot specify specific pathways of interest. A distinct advantage of SEM is the ability to assess and compare multiple fit statistics across nested models in identifying the hypothesized model structure (i.e., the interrelations among latent and observed variables specified in the latent and measurement models) most closely corresponding to the relationships and covariance among variables observed in the Add Health data, thus providing important context for assessing the veracity of the estimates. The current study uses a systematic approach to model-building, making sure that the individual components of the larger structural model demonstrate good fit before proceeding with testing additional components.

The first step of the analysis involves identifying the appropriate trajectories of both perceived weight and BMI over time. This work builds on recent literature emphasizing the utility of SEM for testing nested longitudinal modeling frameworks (Bauldry and Bollen 2018; Bianconcini and Bollen 2018; Bollen and Curran 2004; Bollen and Gutin [Forthcoming]), especially when clear guidance on a choice of model is lacking or unavailable, as is the case for both measures. Indeed, there is evidence of many different trajectories in research on BMI, though the lack of systematic testing of these trajectories does not provide researchers with clear guidance as to which models are most appropriate in a given context. Many studies invoke some kind of “growth” trajectory over the life course – including past studies of the Add Health data (Burdette and Needham 2012; Hargrove 2018; Sokol et al. 2019) – while other research suggests more straightforward autoregressive frameworks (Konttinen et al. 2014; Sokol et al. 2020), or more complex trajectories that incorporate both random intercepts and slopes and autoregressive relations (Aitkin and Alfö 2003). There are compelling reasons to suggest one longitudinal

approach is more or less appropriate than another, and a full review of these explanations is beyond the scope of the current study; however, these models can be tested and compared to one another in identifying the best choice for these analyses.

Likewise, there is limited knowledge of what categorizes longitudinal trajectories of subjective weight, beyond what researchers might infer based on how well it does (or does not) track with individuals' objective weight. On the one hand, if subjective weight is largely a reflection of individuals' objective weight, then we might expect to see the same “growth,” or life course, patterns as those seen in past work on BMI. However, recent studies suggest that weight identities may be relatively “sticky,” such that individuals exhibit a fair degree of stability in assessments of their weight despite changes that occur in their BMI over time (Wedow et al. 2016; Wedow et al. 2018). These findings would suggest the presence of some kind of time-invariant or enduring influence on perceived weight (Bollen and Gutin [Forthcoming]), and/or the presence of a strong lagged effect. In general, many measures used in health and social research can be described as path dependent, to the extent that we might anticipate strong lagged effects, where the best predictor of a measure is its prior value, as in an autoregressive model (Adachi and Willoughby 2015; Biesanz 2012; Bollen and Gutin [Forthcoming]). Critically, none of these models are mutually exclusive, as it is possible to integrate multiple longitudinal properties reflecting different underlying assumptions about the longitudinal processes at work (Bauldry and Bollen 2018; Bianconcini and Bollen 2018; Bollen and Curran 2004; Bollen and Gutin [Forthcoming]). Finally, both subjective and objective weight can be categorized by some degree of “spontaneity” in their measurement, such that they are largely a function of momentary contextual influences – as hypothesized in research on self-rated health (Gunaseraka et al. 2012; Peruccio et al. 2010). In this case, there is no overarching longitudinal trajectory or pattern that is appropriate, as would be evidenced by the poor fit of all models under consideration (Bollen and Gutin [Forthcoming]). Once again, SEM provides the opportunity to consider multiple plausible options and identify the most appropriate model.

Furthermore, these longitudinal models allow researchers to correct for measurement error in the observed measures by modeling them as single indicators of an underlying latent variable. Typically, such

a model would not be identified; however, researchers can make reasonable assumptions about the error variance of the observed measures as being the same over adjacent repeated measures (or fixed over the entire observation period), thereby providing an estimate of the reliability of the measure in the form of the R-squared value (Bollen and Gutin [Forthcoming]; Heise 1969; Werts et al. 1971; Wiley and Wiley 1970). Specifically, by estimating how much of the error variance in the observed measure is explained by the latent variable (as the observed measure is regressed on the latent variable), the remaining unexplained variance provides an estimate of the random measurement error.

One can think of this as a way of assessing how much variance might be expected in the observed response by virtue of asking respondents the same question while erasing their memory of past responses or, alternatively, if individuals were asked the same question in slightly different ways. For instance, there is likely to be considerable measurement error in perceived weight; inherent differences across individuals' frame of mind or emotional state, as well as their interpretation of the question and the underlying ideas or beliefs that it triggers, may limit the reliability of perceived weight as an accurate measure of individuals' subjective weight. This can lead to biased estimates in the relationship between subjective weight and other variables, such as later life physical health and well-being (Daly et al. 2017; Unger et al. 2017). Likewise, random measurement error in BMI may occur from issues in self-reporting, the instruments being used to measure height and weight, and user-error on the part of those using the instruments (Bollen 1989). Thus, correcting for measurement error allows for longitudinal analyses using the underlying error-free latent constructs rather than observed variables (Bianconcini and Bollen 2018), while also providing insight on the reliability of these observed variables over time. In this first step of the analysis, I use the full sample of the 12,300 Add Health respondents who participated in Wave V and at least one other wave, thus allowing maximum flexibility in identifying the appropriate trajectories without further limiting the sample due to the absence of survey weights.

The second step of the analysis builds on the prior in testing plausible models of how the trajectories of subjective and objective weight are associated over time. Depending on the nature of the best-fitting models for both measures, one may expect direct associations between the measures

themselves – such as cross-lagged relationships across waves and direct relationships within waves – or relationships among the latent intercepts and slopes for these measures – as consistent with latent growth models – as well as different assumptions about correlated errors (Bollen and Curran 2004). Critically, the different models may also incorporate various combinations of these features, all of which are testable assumptions, as seen in past research (Kane et al. 2018). These models are initially estimated using the unweighted sample of 12,300 Add Health respondents, which are compared to weighted estimates from the 7,105 Add Health respondents with valid longitudinal survey weights. This weighted sample is used in subsequent analyses because I want to make sure the estimated associations with the Wave V health outcomes are nationally-representative and account for potential selection bias among adults without weights.

The third and final step incorporates Wave V health outcomes into the best-fitting model identified in the prior step: thus, the estimated associations may be between Wave IV subjective and objective weight and Wave V health; between the intercepts and slopes explaining the longitudinal patterns of subjective and objective weight; or both the latent variables and latent intercepts and slopes, depending on the nature of the trajectories and their intersections.

Throughout the analysis, I examine how well the model fits for female and male respondents, as one should verify that the structure is well-fitting for both groups prior to making comparisons in the nature of the trajectories or their associations with health outcomes. One cannot reliably make claims about gender differences if a given model does not appear to work equally well for both groups.

Model fit is examined across multiple criteria commonly used in SEM. Specifically, I use chi-square tests and other measures to assess how closely the hypothesized models fit the Add Health data. Given the large sample size in Add Health, it is likely that even minor specification errors could lead to statistically significant chi-square ( $\chi^2$ ) tests; thus, I also use a BIC comparison statistic that compares the fit of the saturated and hypothesized models, which is obtained by subtracting the degrees of freedom times the natural log of the sample size from the chi-square value (Raftery 1995). Negative values – ideally, greater than 10 – provide evidence favoring the hypothesized over the saturated model (Raftery

1995). The same logic can be applied to nested models, where models with larger negative BIC values are favored. The CFI, TLI, and RMSEA (or 1-RMSEA, in this case) are other common fit statistics (Bentler 1990; Steiger and Lind 1980; Tucker and Lewis 1973). Across all three, values closer to 1 represent better fit, while values less than 0.9 are considered inadequate.

I use Casewise Maximum Likelihood (also called FIML) estimation to account for missing data among endogenous variables, which assumes data are Missing Completely at Random or the less restrictive Missing at Random. Critically, this approach allows individuals to contribute any available information on observed variables at any wave, which is especially useful in longitudinal data where researchers often limit their sample to only those adults meeting a minimum number of waves for inclusion. Overall model fit is then derived from fitting equations across all of the individual cases (Arbuckle 1996). In the models with endogenous categorical outcomes – such as those testing alternate specifications for perceived weight and models with binary health outcomes – I instead use the weighted least square mean and variance adjusted (WLSMV) estimator, with the theta parametrization. Both the R package “lavaan” (Roseel 2012) and Mplus are used to estimate models (Muthén and Muthén 2018); lavaan allows for more flexibility in estimating the different longitudinal models in the first step of the analysis, as convergence issues are less frequent, while Mplus is better suited for incorporating survey weights in the latter part of the analysis.

## **Results**

Table 4.1 provides a descriptive summary of all variables used in the analysis, both for the overall sample and stratified by gender, accounting for complex survey weights. The average age for respondents at Wave I is 15.4, half are female, and approximately 70% identify as non-Hispanic White, 16% as non-Hispanic Black, 9% as Hispanic, and 5% as a different non-Hispanic race or ethnic group. About one-third of respondents had at least one college-educated parent, and just under half were in college or had completed college at Wave III (50% among females, compared to 45% among males). However, many



respondents did not complete college, as the percentage with at least a college degree is 32% in Wave IV and 36% by Wave V, with a higher proportion among females at both waves (34% vs. 29% at Wave IV; 39% vs. 32% at Wave V).

**Table 4.1** Descriptive Statistics for Perceived Weight, Body Mass Index, and Sociodemographic Covariates and Health Outcomes

	<i>Overall</i>		<i>Female</i>		<i>Male</i>	
	Mean/Prop.	Std. Dev.	Mean/Prop.	Std. Dev.	Mean/Prop.	Std. Dev.
<i>Perceived Weight</i>						
Wave I	3.180	0.803	3.356	0.785	3.007	0.782
Wave II	3.195	0.789	3.375	0.771	3.018	0.766
Wave III	3.337	0.809	3.538	0.784	3.140	0.785
Wave IV	3.625	0.823	3.804	0.804	3.448	0.802
<i>Body Mass Index (BMI)</i>						
Wave I	22.565	4.590	22.440	4.585	22.686	4.591
Wave II	23.276	5.197	23.292	5.367	23.260	5.026
Wave III	26.791	6.426	26.973	7.016	26.612	5.782
Wave IV	29.223	7.631	29.416	8.242	29.033	6.969
<i>Sociodemographic</i>						
Age at Wave I	15.396	1.761	15.317	1.726	15.475	1.791
Female	0.497	0.500	-	-	-	-
Race/Ethnicity						
NH-White	0.700	0.458	0.699	0.458	0.702	0.457
NH-Black	0.159	0.366	0.164	0.370	0.154	0.361
Hispanic	0.093	0.290	0.088	0.285	0.097	0.295
NH-Other	0.048	0.214	0.049	0.214	0.048	0.214
College-educated Parent	0.320	0.466	0.313	0.464	0.326	0.469
Wave III In-College +	0.476	0.499	0.499	0.500	0.453	0.498
Wave IV B.A. Degree +	0.316	0.465	0.337	0.473	0.294	0.456
Wave V B.A. Degree +	0.357	0.479	0.389	0.488	0.324	0.468
<i>Wave V Outcomes</i>						
BMI	30.969	7.958	31.349	8.613	30.517	7.073
Syst. BP (1)	124.969	15.978	120.631	15.191	129.891	15.413
Syst. BP (2)	123.689	15.464	119.614	14.714	128.314	14.985

Syst. BP (3)	123.090	15.042	119.263	14.230	127.444	14.755
Diast. BP (1)	80.628	11.270	78.194	10.785	83.389	11.171
Diast. BP (2)	80.243	11.162	77.862	10.775	82.947	10.977
Diast. BP (3)	79.755	10.924	77.415	10.464	82.417	10.826
Measured						
Hypertension/Rx	0.334	0.471	0.261	0.439	0.416	0.493
C-reactive Protein	3.947	5.650	4.900	6.555	2.821	4.067
Felt Depressed Freq.	1.419	0.699	1.453	0.725	1.385	0.669
Felt Blues Freq.	1.372	0.691	1.388	0.694	1.357	0.687
Felt Sad Freq.	1.585	0.672	1.638	0.690	1.532	0.649
Depression Dx	0.256	0.436	0.328	0.469	0.185	0.389
Anxiety Dx	0.234	0.423	0.300	0.458	0.169	0.374
Sleep Trouble Freq.	1.774	1.345	1.957	1.355	1.592	1.309

N(Overall)=7,105; N(Female)=4,152; N(Male)=2,953.

Results based on casewise maximum likelihood (also called FIML) estimation to account for missing data, with robust (Huber-White) standard errors.

Weighted estimates account for person-level longitudinal survey weights (WI-V), school-level clustering, and regional strata.

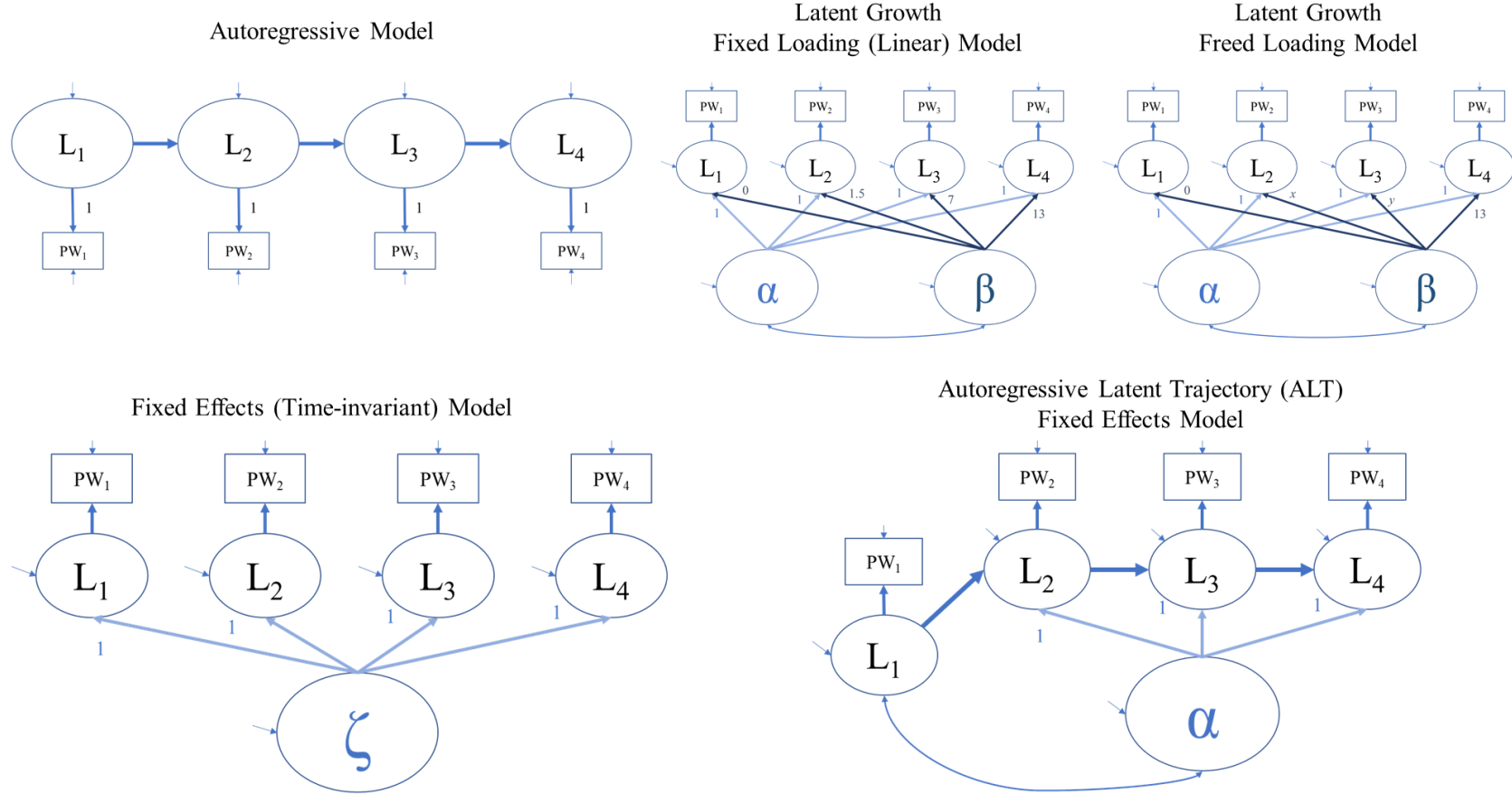
The focal variable of interest, perceived weight, has an increasing trend over time, following the same trend in BMI across the first four waves. Namely, while respondents have a mean perceived weight of 3.19 in Waves I and II – slightly above a value corresponding with perceiving oneself as at about the right weight – this value increases to 3.63 by Wave IV, closer to the “slightly overweight” threshold. However, there are clear gender differences in this pattern between women and men. Women perceive themselves as more overweight across all four waves, with a consistent gap of 0.35 in the mean value over time; indeed, not until Wave IV does the mean value among men (3.45), exceed the mean value for women at Waves I and II (3.36). By contrast, BMI means are much closer for female and male respondents, and track more closely over time, shifting from a BMI (~23) that is considered “normal” based on BMI categories in Waves I and II (albeit these categories are not necessarily appropriate for the age range represented in Wave I), to overweight in Wave III (26.8), and being close to the cutoff for obesity by Wave IV (29.2).

Finally, mean values and proportions for Wave V health outcomes show clear evidence of gender differences in both physical and mental health. BMI has increased further and is above the cutoff for obesity at mean value of 31, which is slightly higher for females (31.3) than males (30.5). Conversely, systolic blood pressure is higher for males (128.6) than females (119.8), with a pattern of decreasing values across repeated measures. Likewise, diastolic blood pressure is higher for males than females (82.9 vs. 77.8), also with a pattern of decreasing values across measures. Unsurprisingly, over two-in-five males have a prevalence of high blood pressure or report using anti-hypertensive medication, as compared to just over one-quarter of female respondents. The overall mean for systolic over diastolic blood pressure is 123.9/80.2, and about one-third of adults have measured hypertension or are on medication. Looking at C-reactive protein, the mean value for the sample is 3.9 mg/L, which exceeds the “high” risk threshold of 3.0 often used in medicine and research (Ridker 2003); however, female respondents have much higher average CRP (4.9) compared to males (2.8). Female respondents are also more likely to report poor mental health symptoms than their male counterparts, as seen in the measures used to estimate latent depression (Depressed frequency 1.45 vs. 1.38 [1.42 Overall]; Blues frequency 1.39 vs. 1.36 [1.37

Overall]; Sad frequency 1.64 vs. 1.53 [1.59 Overall]), as well as prevalence of diagnosed depression (33% vs. 18.5% [26% Overall]) and diagnosed anxiety (30% vs. 17% [23% Overall]). Finally, the overall mean score of 1.77 on sleep trouble frequency suggests the average respondent had trouble falling asleep closer to one or two times a week as compared to less than once a week, though this average is closer to the one or two times threshold for females (1.96) as compared to male respondents (1.59).

Since I use non-weighted estimates in some of the analyses, the descriptive statistics for the unweighted sample of 12,300 Wave V adults are shown in Table A.9 in the Appendix. The most notable differences in this sample are the higher proportion of female respondents (57%) and lower percentage of non-Hispanic White adults (60%). However, most of the other estimates and patterns are largely the same.

As discussed earlier, there is nothing in the literature on perceived weight to suggest a specific longitudinal model best-suited for the measure; thus, I test plausible options consistent with different assumptions about lagged effects, enduring influences, and growth trajectories, as well as possible combinations therein. With only four waves of data for perceived weight, one cannot test all possible longitudinal model types and trajectories. In some cases, constraints need to be imposed (Bollen and Curran 2004). However, I am still able to examine five distinct models consistent with the different plausible longitudinal processes at work, as seen in Figure 4.1. The five models are consistent with a “lagged effects” perspective on perceived weight (as represented by an autoregressive model), an “enduring influence” framework (equivalent to a traditional fixed effects model [Bollen and Brand 2010]), two different growth trajectories assuming either linear slopes or freed loading slopes that vary over time (Bauldry and Bollen 2018), or a final “hybrid” model where the enduring influence is complemented by lagged effects over time (Bollen and Gutin [Forthcoming]).



$L_j$  = Latent subjective weight at Wave  $j$ ;  $j = 1,2,3,4$ .  
 $PW_j$  = Measures of Perceived Weight at Wave  $j$ ;  $j = 1,2,3,4$ .  
 $\alpha, \beta, \zeta$  = Latent intercepts and slopes.  
 Open arrows indicate error terms.

**Figure 4.1** Plausible Longitudinal Trajectories for Subjective Weight

Table 4.2 presents fit statistics for these different longitudinal models, as well as any additional notes on the estimation. The best fitting model is clearly the autoregressive trajectory, wherein the best predictor of individuals' subjective weight is its prior value. This model has a non-significant chi-square value and a negative BIC, providing evidence that the hypothesized model fits the data well. Moreover, the CFI, TLI, and 1-RMSEA values are either at the perfect value of 1, or very close to it, with no estimation issues or additional constraints. With the exception of the fixed effects model, all of the other models show good fit on the basis of CFI, TLI, and 1-RMSEA; however, the positive BIC values suggest they are not appropriate for modeling subjective weight. These models also produce negative variances for some of the latent variables, suggesting misspecification issues.

**Table 4.2** Comparison of Longitudinal Models for Subjective Weight

<b>Model</b>	<b><math>\chi^2</math></b>	<b>DF</b>	<b>BIC</b>	<b>CFI</b>	<b>TLI</b>	<b>1-RMSEA</b>	<b>Notes</b>
Autoregressive	1.841	1	-7.576	1.000	1.000	0.991	<i>None</i>
Fixed Effects	437.891	3	409.639	0.933	0.865	0.838	<i>Negative variances</i>
Linear Growth	146.229	3	117.977	0.981	0.962	0.914	<i>Negative variances</i>
Freed Loading Growth	33.333	1	23.916	0.992	0.953	0.905	<i>Negative variances</i>
ALT-Fixed Effects	28.314	1	18.897	0.997	0.984	0.945	<i>Regressions constrained to be equal</i>

N=12,300.

Results based on casewise maximum likelihood (also called FIML) estimation to account for missing data, with robust (Huber-White) standard errors.

Variance on first and last two observed measures of perceived weight constrained to be equal, respectively.



The models above use a continuous specification for perceived weight, based on a five-point scale from very underweight to very overweight. To ensure that the choice of specification does not result in different conclusions about the choice of the most appropriate model, I present a comparison of all the aforementioned models based on alternate specifications for perceived weight in Table A.10 in the Appendix. Namely, I compare models with only three categories where underweight and about right responses are combined into a single category; three categories where respondents are either underweight, about right, or overweight; a dichotomous indicator of overweight or not; and ordinal specifications of the five and three category specifications. These additional analyses provide clear evidence that the autoregressive (AR) model outperforms the other longitudinal models across most specifications. There is some ambiguity when treating subjective weight status as a five-category ordinal measure, wherein the ALT-fixed effects (ALT-FE) model (combining an enduring and lagged effects perspective) and freed loading growth models also have good fit. However, the general pattern across specifications favors the AR model, and thus I feel confident using a continuous version of the perceived weight measure.

Following the same approach described above, I proceed with identifying the appropriate longitudinal model to describe the trajectory of BMI in the Add Health sample. Though I am primarily focused on Wave V BMI as an outcome, I include the measure in this trajectory to have more flexibility in estimating the models over five rather than four waves. As seen in Table 4.3, the autoregressive model once again has excellent fit, with a nonsignificant chi-square and a large, negative BIC, exceeding the recommendation for “strong” evidence of good fit (Raftery 1995). As before, there is evidence of relatively good fit for a number of the models based on CFI, TLI, and 1-RMSEA, but most have large and positive BIC values, as well as possible issues in the estimation or require many additional constraints to converge or be identified. There is good support for the ALT-FE model; a likelihood ratio test comparing the ALT-FE and AR model is nonsignificant, favoring the ALT-FE model. However, the BIC difference of 16 between these models favors the AR model (Raftery 1995). In assessing the resulting parameters, there appear to be no major differences; AR coefficients are consistently larger than 1.000 in the ALT-FE model, albeit with larger standard errors. Given that the substantive interpretation of the models is similar,

I proceed with the AR model, which also allows for a more straightforward interpretation of how the trajectory of objective weight intersects with subjective weight over time. Finally, the AR models for both subjective and objective weight demonstrate configural invariance between female and male respondents, suggesting the model structure is appropriate for both groups.

**Table 4.3** Comparison of Longitudinal Models for Objective Weight

Model	$\chi^2$	DF	BIC	CFI	TLI	1-RMSEA	Notes
Autoregressive	2.561	3	-25.691	1.000	1.000	1.000	<i>None</i>
Fixed Effects	1627.872	6	1571.368	0.822	0.703	0.689	<i>Negative variances</i>
Linear Growth	1263.366	7	1197.445	0.907	0.868	0.792	<i>Negative variances</i>
Quadratic Growth	118.473	4	80.804	0.992	0.980	0.919	<i>Negative variances</i>
Freed Loading Growth	142.254	4	104.585	0.988	0.970	0.901	<i>Negative variances</i>
ALT-Fixed Effects	0.087	1	-9.330	1.000	1.000	1.000	<i>None</i>
ALT-Linear Growth	30.279	2	11.444	0.999	0.988	0.954	<i>None</i>
ALT-Quadratic Growth	365.480	2	346.645	0.980	0.980	0.819	<i>Regressions constrained to be equal; Observed BMI error variances constrained to be equal; Covariance of latent Obj. with Intercept and Slopes constrained to be equal.</i>
ALT-Freed Loading Growth	13.371	2	-5.464	0.999	0.996	0.965	<i>Regressions constrained to be equal; Observed BMI error variances constrained to be equal.</i>

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N=12,300.

Results based on casewise maximum likelihood (also called FIML) estimation to account for missing data, with robust (Huber-White) standard errors.

Variance on first and last two observed measures of Body Mass Index constrained to be equal, respectively.

Table 4.4 shows coefficient estimates from the AR model for both measures, demonstrating how both subjective and objective weight exhibit a high degree of inertia or *path dependency* on the basis of the autoregressive coefficients. Namely, the subjective weight model has relatively high autoregressive coefficients between the latent variables for subjective weight ( $W1 \rightarrow W2$  0.921;  $W2 \rightarrow W3$  0.775;  $W3 \rightarrow W4$  0.878), as well as fairly high R-square values for the endogenous latent variables ( $W2$  0.870;  $W3$  0.620;  $W4$  0.758) suggesting that the majority of variance in subjective weight status is explained by its previous value. The R-squared for the observed measures of perceived weight provide an estimate their reliability; there is clear evidence of nonnegligible measurement error, as approximately one-third to one-quarter of the variation in the measure (depending on the wave) is attributable to random error.

**Table 4.4** Coefficient Estimates for Autoregressive Models of Subjective Weight (SUBJ.) and Objective Weight (OBJ.)

Parameters	Perceived Weight			Body Mass Index			
	Estimate	Std. Err.	P-value	Estimate	Std. Err.	P-value	
<i>Regressions</i>				<i>Regressions</i>			
Wave IV SUBJ. ←				Wave V OBJ. ←			
Wave III SUBJ.	0.921	0.017	0.000	Wave IV OBJ.	0.936	0.015	0.000
Wave III SUBJ. ←				Wave IV OBJ. ←			
Wave II SUBJ.	0.775	0.015	0.000	Wave III OBJ.	1.072	0.013	0.000
Wave II SUBJ. ←				Wave III OBJ. ←			
Wave I SUBJ.	0.878	0.016	0.000	Wave II OBJ.	1.104	0.013	0.000
				Wave II OBJ. ←			
				Wave I OBJ.	1.086	0.013	0.000
<i>Intercepts</i>				<i>Intercepts</i>			
Wave I SUBJ.	3.190	0.007	0.000	Wave I OBJ.	22.610	0.041	0.000
Wave II SUBJ.	0.399	0.052	0.000	Wave II OBJ.	-1.332	0.286	0.000
Wave III SUBJ.	0.870	0.048	0.000	Wave III OBJ.	1.154	0.290	0.000
Wave IV SUBJ.	0.560	0.058	0.000	Wave IV OBJ.	0.409	0.340	0.229
				Wave V OBJ.	3.494	0.410	0.000
<i>Variances</i>				<i>Variances</i>			
Wave I PW	0.161	0.007	0.000	Wave I BMI	1.907	0.171	0.000
Wave II PW	0.161	0.007	0.000	Wave II BMI	1.907	0.171	0.000
Wave III PW	0.233	0.009	0.000	Wave III BMI	4.112	0.392	0.000
Wave IV PW	0.233	0.009	0.000	Wave IV BMI	5.158	0.753	0.000
				Wave V BMI	5.158	0.753	0.000
Wave I SUBJ.	0.474	0.011	0.000	Wave I OBJ.	18.544	0.458	0.000
Wave II SUBJ.	0.054	0.011	0.000	Wave II OBJ.	2.147	0.310	0.000
Wave III SUBJ.	0.154	0.007	0.000	Wave III OBJ.	8.667	0.355	0.000
Wave IV SUBJ.	0.110	0.014	0.000	Wave IV OBJ.	9.203	0.624	0.000
				Wave V OBJ.	10.280	1.128	0.000

*R-Square*

Wave I PW	0.746
Wave II PW	0.722
Wave III PW	0.636
Wave IV PW	0.661
Wave II SUBJ.	0.870
Wave III SUBJ.	0.620
Wave IV SUBJ.	0.758

*R-Square*

Wave I BMI	0.907
Wave II BMI	0.926
Wave III BMI	0.902
Wave IV BMI	0.911
Wave V BMI	0.916
Wave II OBJ.	0.911
Wave III OBJ.	0.771
Wave IV OBJ.	0.826
Wave V OBJ.	0.818

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N=12,300.

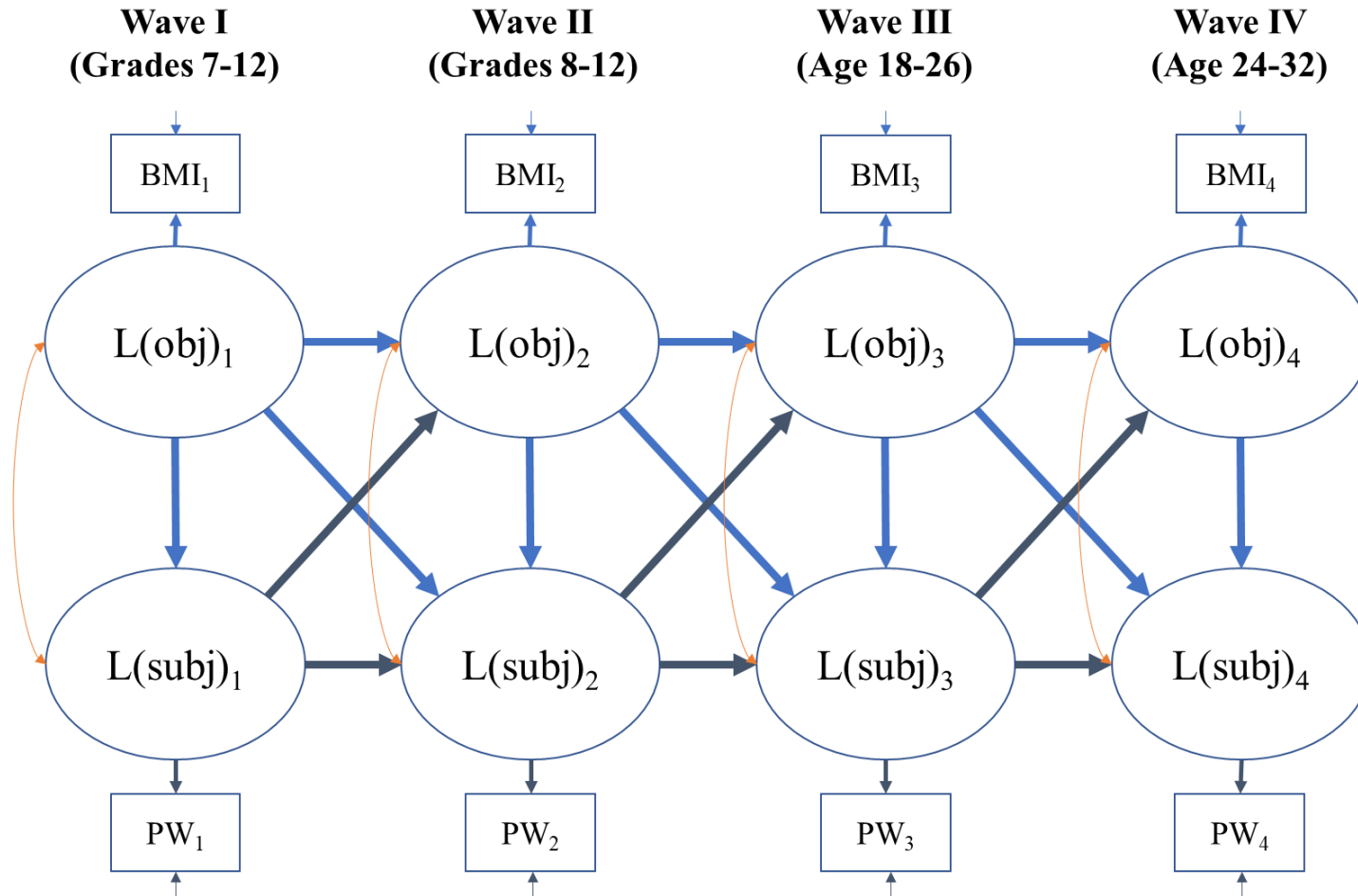
Results based on casewise maximum likelihood (also called FIML) estimation to account for missing data, with robust (Huber-White) standard errors.

Likewise, in the AR model for objective weight there is evidence of a strong and consistent effect from one wave to the next, with AR coefficients very close to, or exceeding a value of 1.000 (sometimes described as “explosive autoregression” [Phillips 1987]):  $BMI1 \rightarrow BMI2 = 1.086$ ;  $BMI2 \rightarrow BMI3 = 1.104$ ;  $BMI3 \rightarrow BMI4 = 1.072$ ;  $BMI4 \rightarrow BMI5 = 0.936$ ). This model also explains a very large proportion of the variation in objective weight, with high R-square values for all endogenous latent variables (BMI2 0.911; BMI3 0.771; BMI4 0.826; BMI5 0.818). Interestingly, the reliability estimates suggest relatively little measurement error in BMI – in either the self-report or measured observed variables – with R-square values ranging from 0.902 to 0.926. However, this does not mean that the measurement error is negligible; when estimating a model where BMI is assumed to be a perfect indicator of the latent variable (i.e., reliability = 1.000) there is a considerable decline in model fit, suggesting that the correction for error is preferable.

Lacking any formal guidance or clear theory on how the trajectories of subjective and objective weight are interrelated over time, I proceed with testing plausible combinations of the different features one may expect to include based on the temporal ordering of effects within and across waves. The full set of plausible pathways is shown in Figure 4.2, serving as the basis for including or excluding different effects and correlations in the hypothesized models shown in Table 4.5. Specifically, in the “Correlated Only” model, I assume that the two trajectories are independent in terms of direct influences, but allow for objective and subjective weight to be correlated within waves – as would be expected given their being assessed at the same point in time. The “Within-wave Obj. on Subj. Only” model assumes that the primary information individuals draw on when assessing their subjective weight (in addition to their past response) is their objective weight or body size at that same point in time. By contrast, the “Cross-lagged Only” model assumes individuals’ subjective weight influences their later life objective weight and their current objective weight influences later life subjective weight. This is consistent with the logic that current beliefs about weight influence later life weight-related behaviors and that individuals’ beliefs

about their weight may be influenced by objective weight in the time period up to the moment they are asked. In addition to these three models, I combine various aspects of these models to account for more complex structures and intersecting relationships.





$L(obj)_j$  = Latent objective weight at Wave  $j$ ;  $j = 1, 2, 3, 4$ .  
 $L(subj)_j$  = Latent subjective weight at Wave  $j$ ;  $j = 1, 2, 3, 4$ .  
 $BMI_j$  = Measures of Body Mass Index at Wave  $j$ ;  $j = 1, 2, 3, 4$ .  
 $PW_j$  = Measures of Perceived Weight at Wave  $j$ ;  $j = 1, 2, 3, 4$ .  
 Open arrows indicate error terms.  
 Double headed arrows indicate correlated errors.

**Figure 4.2** Plausible Pathways Linking Trajectories of Objective and Subjective Weight

The CFI, TLI, and 1-RMSEA criteria suggest excellent fit for all of the models; fortunately, the BIC provides more nuance in differentiating across these nested comparisons. The correlated and cross-lagged and cross-lagged and within-wave models have the best fit, with identical large and negative BIC values (-20.331). These are known as chi-square equivalent, or just “equivalent,” models, due to the fact that the same parameters can be identified with the same elements of the covariance matrix (Raykov and Penev 1999). Equivalent models are not uncommon in cross-lagged models where the directionality of the relationship among variables is not clear; in these cases, researchers’ substantive knowledge can be used to identify the more appropriate model structure (Raykov and Penev 1999). Thus, it seems more appropriate to allow for direct within-wave effects – as one would expect individuals’ objective body size to influence their subjective weight, rather than making the weaker assumption that they are only correlated.

**Table 4.5** Comparison of Intersecting Longitudinal Models for Subjective and Objective Weight

<b>Model</b>	<b><math>\chi^2</math></b>	<b>DF</b>	<b>BIC</b>	<b>CFI</b>	<b>TLI</b>	<b>1-RMSEA</b>
Correlated Only	246.894	14	115.051	0.995	0.989	0.957
Within-wave Obj. on Subj. Only	727.650	14	595.807	0.983	0.967	0.924
Cross-lagged Only	212.091	11	108.500	0.996	0.989	0.955
Correlated and Within-wave Obj. on Subj.	116.962	12	3.954	0.997	0.993	0.966
Correlated and Cross-lagged	55.008	8	-20.331	0.999	0.998	0.980
Cross-lagged and Within-wave	55.008	8	-20.331	0.999	0.997	0.975

N=12,300.

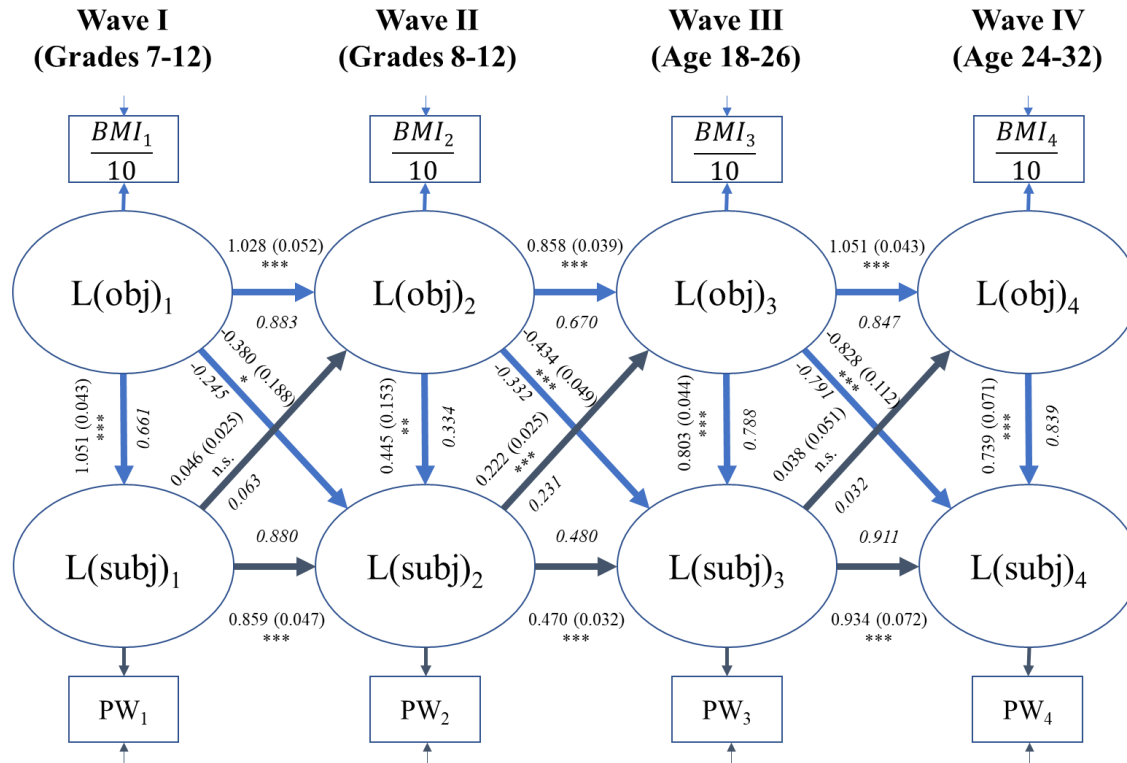
Results based on pairwise maximum likelihood estimation to account for missing data, with robust (Huber-White) standard errors.

Before interpreting the coefficients in the best fitting model of both subjective and objective weight, it is important to acknowledge recent research on the utility of random intercept cross-lagged panel models (RICLPM) to address potential issues in how the relative “stability” of longitudinal measures is accounted for in traditional cross-lagged models (Hamaker et al. 2015; Usami et al. 2019), similar to the models used in these data. Namely, Hamaker and colleagues emphasize the importance of accounting for “stable, trait-like differences” exclusively in relation to “within-unit fluctuations” by using random intercepts to account for the part of the variance in a measure attributable to this “long-run” influence (Mulder and Hamaker 2021: 1). Their research suggests RICLPMs typically have better fit, and lead to more accurate autoregressive and cross-lagged estimates that are more appropriate for causal interpretation.

The RICLPM model is not explicitly tested along with those presented above, on account of the fact that it does not model measurement error in a manner that allows for estimates of reliability; rather, the RICLPM model assumes that observed variables are perfect indicators of the underlying latent variable, with the latent time-invariant intercepts having a direct influence on these observed variables. Nevertheless, Hamaker et al.’s concerns about stability in cross-lagged models are pertinent to this analysis, and I proceed with fitting the proposed RICLPM. The resulting model has considerably worse fit relative to the more ‘traditional’ CLPM corrected for measurement error ( $BIC = 84.647$ ); there are also issues in the estimation with respect to negative variances. Thus, I feel confident that the CLPM – modified to include direct effects, rather than cross-lagged effects – is most appropriate to these data.

Figure 4.3 shows this cross-lagged model with all estimated pathways, accounting for longitudinal survey weights; both unstandardized coefficients (with standard errors) and standardized coefficients (in italics) are shown, on account of the fact that these two measures have different scales. Indeed, observed BMI was divided by 10 to bring variances among observed variables closer to one another and help with model convergence; though this makes interpreting unstandardized coefficients less clear, it does not

affect model fit. Thus, with the exception of the autoregressive coefficients, I focus on the standardized estimates as they are useful for comparing the *relative* effect of measures in cross-lagged models (Kuiper and Ryan 2018).



Model fit statistics:  $\chi^2 = 23.558$ ,  $DF = 8$ ,  $SBIC = -47.390$ ,  $CFI = 0.999$ ,  $TLI = 0.995$ ,  $1-RMSEA = 0.983$ .

BMI divided by 10 to reduce variance and help with model convergence.

L(obj)<sub>j</sub> = Latent objective weight at Wave  $j$ ;  $j = 1, 2, 3, 4$ .

L(subj)<sub>j</sub> = Latent subjective weight at Wave  $j$ ;  $j = 1, 2, 3, 4$ .

BMI<sub>j</sub> = Measures of Body Mass Index at Wave  $j$ ;  $j = 1, 2, 3, 4$ .

PW<sub>j</sub> = Measures of Perceived Weight at Wave  $j$ ;  $j = 1, 2, 3, 4$ .

Open arrows indicate error terms.

Double headed arrows indicate correlated errors.

Standardized coefficients in *italics*.

\*\*\*  $p < 0.001$ , \*\*  $p < 0.01$ , \*  $p < 0.05$ , n.s.  $p \geq 0.05$ .

**Figure 4.3** Intersecting Pathways of Objective and Subjective Weight from Adolescence to Early Adulthood

The previously described large, autoregressive coefficients for the two trajectories are largely unchanged in the full model, though there is some attenuation across all estimates. Notably, the autoregressive effect between Waves II and III for SWS is reduced to 0.470, but it is still statistically significant. There is evidence of a consistent, negative cross-lagged effect between objective weight at one wave and subjective weight in the next, such that greater objective weight in the prior wave is associated with lower perceived subjective weight in the next. This cross-lagged effect is greatest between objective weight at Wave III and subjective weight at Wave IV, with a standardized estimate of -0.791; the effect is smaller, but also negative and statistically significant in prior waves, with standardized estimates close to -0.3. By contrast, the cross-lagged effect of subjective weight at prior wave to objective weight at the subsequent wave is positive but smaller. Both unstandardized and standardized estimates from Wave I subjective weight to Wave II objective weight and Wave III subjective weight to Wave IV objective weight are not significantly different from 0. However, there is a larger and significant, positive effect from Wave II subjective weight to Wave III objective weight (0.231 standardized) – a time when most respondents finish high school and are either in college or have completed their education – such that greater perceived weight is associated with larger body size. This likely explains some of the attenuation in the autoregressive subjective weight coefficient between these two time periods. Finally, there is a consistent positive and significant within-wave effect from objective weight to subjective weight, such that having a larger body size is associated with perceiving oneself as being overweight at a given point in time. Based on the standardized coefficients, this direct effect appears greater in Waves III (0.788) and IV (0.839) compared to Waves I (0.661) and II (0.334). Interestingly, the R-squared values are generally unchanged compared to the prior autoregressive models; however, about 50% of the variance in Wave I subjective weight is explained by Wave I objective weight, given that this is the only term influencing Wave I subjective weight in the model.

Table A.11 shows the same estimates for this model in the full, unweighted sample of 12,300 Wave V adults, yielding comparable estimates. A key difference is that this model was able to converge without dividing BMI by 10, hence the larger estimates for the unstandardized cross-lagged coefficients.

Given the interest in looking at gender differences in the relationship between these trajectories – as well as how they are related to adult health – Table A.12 presents coefficient estimates from a model testing for configural invariance. The fit for this model – where the structure is the same for males and females, but parameters are free to vary – is very good, with a large and negative BIC of -117. The general patterns noted above are observed in both female and male respondents, with no apparent differences in the autoregressive, cross-lagged, or direct relationships. That said, latent variable means and intercepts differ, as might be expected based on descriptive statistics, with consistently higher estimated values for females perceiving themselves as overweight over time.

In the final stage of the analysis, Wave V physical and mental health outcomes are regressed on Wave IV subjective and objective weight, accounting for their intersecting trajectories. The overall fit of the model is excellent, with CFI, TLI, and 1-RMSEA all very close to 1 and a large and negative BIC of -1273. Once again, I primarily focus on standardized coefficients given that the scale of the variables differs across health outcomes, with Wave V BMI, SBP, DBP, and CRP all divided by 10 to ensure similar variances among all variables in the model.

There are notable differences in the association between subjective and objective weight and the different outcomes, as seen in Table 4.6. Unsurprisingly, Wave IV objective weight continues to have a large, positive association with Wave V BMI, as befitting the autoregressive trajectory, and Wave IV subjective weight continues to have a small and nonsignificant cross-lagged association with subsequent BMI. Wave IV objective weight is also positively associated with higher latent SBP (0.828) and latent DBP (0.681), though Wave IV subjective weight exhibits a significant negative association with both (-0.506 for SBP, -0.408 for DBP), on average. Likewise, Wave IV objective weight is associated with higher risk of measured hypertension or being on anti-hypertensive medicine (0.592), whereas there is a negative association with Wave IV subjective weight (-0.198). By contrast, CRP is positively associated with both Wave IV BMI (0.193) and SWS (0.164).



**Table 4.6** Coefficient Estimates for Health Outcomes Regressed on Subjective Weight (SUBJ.) and Objective Weight (OBJ.)

Parameters	Estimate	Std. Err.	P-value	Stdz. Est.
<i>Latent Variables</i>				
Systolic Blood Pressure →				
→				
Reading #1	1.000			
Reading #2	1.000			
Reading #3	0.964	0.014	0.000	
Diastolic Blood Pressure →				
→				
Reading #1	1.000			
Reading #2	1.000			
Reading #3	0.957	0.017	0.000	
Depression →				
→				
Felt Depressed	1.000			
Had Blues	0.824	0.015	0.000	
Felt Sad	0.747	0.018	0.000	
<i>Health Outcome Regressions</i>				
Wave V BMI ←				
←				
Wave IV OBJ.	0.963	0.050	0.000	0.842
Wave IV SUBJ.	-0.008	0.061	0.893	-0.006
SBP ←				
←				
Wave IV OBJ.	1.554	0.156	0.000	0.828
Wave IV SUBJ.	-1.118	0.193	0.000	-0.506
DBP ←				
←				
Wave IV OBJ.	0.957	0.114	0.000	0.681
Wave IV SUBJ.	-0.674	0.142	0.000	-0.408
Measured Hypertension/Rx ←				
←				
Wave IV OBJ.	0.788	0.113	0.000	0.592
Wave IV SUBJ.	-0.310	0.141	0.028	-0.198
CRP ←				
←				
Wave IV OBJ.	0.164	0.031	0.000	0.193
Wave IV SUBJ.	0.165	0.037	0.000	0.164
Latent Depression ←				
←				
Wave IV OBJ.	-0.072	0.038	0.062	-0.083
Wave IV SUBJ.	0.132	0.043	0.002	0.130
Depression Dx ←				
←				
Wave IV OBJ.	-0.419	0.074	0.000	-0.315
Wave IV SUBJ.	0.656	0.087	0.000	0.419
Anxiety Dx ←				
←				
Wave IV OBJ.	-0.503	0.076	0.000	-0.378
Wave IV SUBJ.	0.682	0.099	0.000	0.436
Trouble Sleeping ←				
←				
Wave IV OBJ.	-0.162	0.078	0.039	-0.090

Wave IV SUBJ.	0.340	0.096	0.000	0.161
<i>SWS and BMI Regressions</i>				
Wave IV OBJ. ←				
Wave III OBJ.	0.998	0.044	0.000	0.836
Wave III SUBJ.	0.087	0.049	0.079	0.073
Wave III OBJ. ←				
Wave II OBJ.	0.854	0.032	0.000	0.685
Wave II SUBJ.	0.193	0.024	0.000	0.207
Wave II OBJ. ←				
Wave I OBJ.	1.047	0.052	0.000	0.897
Wave I SUBJ.	0.037	0.023	0.112	0.051
Wave IV SUBJ. ←				
Wave III SUBJ.	1.008	0.059	0.000	1.000
Wave III OBJ.	-0.964	0.111	0.000	-0.950
Wave IV OBJ.	0.793	0.087	0.000	0.933
Wave III SUBJ. ←				
Wave II SUBJ.	0.482	0.023	0.000	0.515
Wave II OBJ.	-0.402	0.067	0.000	-0.320
Wave III OBJ.	0.799	0.065	0.000	0.793
Wave II SUBJ. ←				
Wave I SUBJ.	0.880	0.044	0.000	0.902
Wave I OBJ.	-0.771	0.320	0.016	-0.492
Wave II OBJ.	0.754	0.263	0.004	0.562
Wave I SUBJ. ←				
Wave I OBJ.	1.063	0.046	0.000	0.663
<i>R-Square</i>				
Wave I BMI	0.767			
Wave II BMI	0.818			
Wave III BMI	0.762			
Wave IV BMI	0.820			
Wave I PW	0.747			
Wave II PW	0.737			
Wave III PW	0.607			
Wave IV PW	0.611			
Wave II OBJ.	0.868			
Wave III OBJ.	0.707			
Wave IV OBJ.	0.812			
Wave I SUBJ.	0.439			
Wave II SUBJ.	0.923			
Wave III SUBJ.	0.904			
Wave IV SUBJ.	0.850			
Wave V BMI	0.699			
L-SBP	0.200			
SBP #1	0.762			
SBP #2	0.812			
SBP #3	0.801			

L-DBP	0.138
DBP #1	0.806
DBP #2	0.834
DBP #3	0.805
Measured Hypertension/Rx	0.182
CRP	0.120
L-Depression	0.005
Feel Depressed	0.860
Had Blues	0.597
Feel Sad	0.518
Depression Dx	0.041
Anxiety Dx	0.041
Trouble Sleeping	0.008

N=7,105.

Observed BMI, observed SBP and DBP, and observed CRP divided by 10 to reduce variance and help with model convergence.

Covariances, means and intercepts, and variances omitted for parsimony.

Results based on casewise maximum likelihood (also called FIML) estimation to account for missing data, with diagonal weighted least squares standard errors due to binary outcome variables.

Weighted estimates account for person-level longitudinal survey weights (WI-V), school-level clustering, and regional strata.

Model fit statistics:  $\chi^2 = 225.147$ ,  $DF = 169$ ,  $BIC = -1273.639$ ,  $CFI = 0.997$ ,  $TLI = 0.996$ ,  $1-RMSEA = 0.993$ .

With respect to mental health related outcomes, Wave IV objective weight is not significantly associated with latent depression, whereas there is a positive association with Wave IV subjective weight (0.130). Wave IV objective weight has a significant negative association with both diagnosed depression (-0.315) and anxiety (-0.378), compared to significant positive associations with Wave IV subjective weight (0.419 and 0.436, respectively). Finally, there a significant, but smaller negative association between BMI and trouble sleeping (-0.090) and a significant positive association between Wave IV subjective weight and trouble sleeping (0.161). The R-squared values provide important context for this model and its explanatory power across the different physical and mental health outcomes; critically, it is apparent that objective and subjective weight alone do not explain much of the variation in either physical

and mental health. Aside from Wave V BMI, objective and subjective weight only explain about 10-20% of the variation in blood pressure, hypertension, and CRP. The R-squared values are considerably lower for mental health, where over 95% of the variation is unexplained.

Prior to comparing gender differences in the associations between Wave IV subjective and objective weight and Wave V health outcomes, I provide the caveat that I could not definitively demonstrate that this model has configural invariance across both genders. Formal tests of measurement invariance encountered convergence issues, though I believe this is largely attributable to the complexity of the model rather than misspecification. Indeed, the model fit is excellent when assessed separately by gender, with CFI, TLI, and 1-RMSEA near 1 and equally large and negative BICs (-1204 for female respondents; -1160 for male respondents). Thus, coupled with evidence of configural invariance from the previous model without health outcomes, one might expect that the estimates provided in Table 4.7 are valid.

The general pattern for the associations between objective and subjective weight and physical health is very similar for both females and males. Wave IV objective weight continues to be strongly, positively associated with Wave V BMI, while Wave IV subjective weight is not. Wave IV objective weight is also positively associated with SBP and DBP and measured hypertension or use of antihypertensive medication, while these same measures are negatively associated with Wave IV subjective weight. However, it appears that the association between blood pressure and objective weight is somewhat weaker for females as compared to males (0.631 vs. 0.759 for SBP; 0.390 vs. 0.654 for DBP; 0.347 vs. 0.468 for measured hypertension or medication). Likewise, the positive association between CRP and Wave IV objective weight is greater among male respondents as compared to females (0.634 vs. 0.312), but the relationship between CRP and Wave IV subjective weight is not significant among females and negative among males (-0.352). Conversely, the associations between mental health and objective and subjective are more pronounced for female respondents as compared to males.

There is no significant relationship with latent depression for either group, but Wave IV subjective weight is significantly associated with greater diagnosed depression among females (0.271), as well as greater diagnosed anxiety (0.344). Neither measure is associated with trouble sleeping for females or males.

**Table 4.7** Coefficient Estimates for Health Outcomes Regressed on Subjective Weight Status (SWS) and Body Mass Index (BMI); Female vs. Male

Parameters	Female				Male			
	Estimate	Std. Err.	P-value	Stdz. Est.	Estimate	Std. Err.	P-value	Stdz. Est.
<i>Latent Variables</i>								
Systolic Blood Pressure →								
Reading #1	1.000				1.000			
Reading #2	1.000				1.000			
Reading #3	0.976	0.022			0.958	0.022	0.000	
Diastolic Blood Pressure →								
Reading #1	1.000				1.000			
Reading #2	1.000				1.000			
Reading #3	0.958	0.023			0.955	0.026	0.000	
Depression →								
Felt Depressed	1.000				1.000			
Had Blues	0.826	0.024	0.000		0.832	0.024	0.000	
Felt Sad	0.749	0.027	0.000		0.724	0.028	0.000	
<i>Health Outcome Regressions</i>								
Wave V BMI ←								
Wave IV OBJ.	1.043	0.082	0.000	0.929	1.010	0.110	0.000	0.870
Wave IV SUBJ.	-0.149	0.109	0.172	-0.096	-0.058	0.110	0.596	-0.052
SBP ←								
Wave IV OBJ.	1.007	0.186	0.000	0.631	1.561	0.329	0.000	0.759
Wave IV SUBJ.	-0.498	0.257	0.053	-0.225	-0.805	0.324	0.013	-0.402
DBP ←								
Wave IV OBJ.	0.476	0.132	0.000	0.390	1.028	0.273	0.000	0.654
Wave IV SUBJ.	-0.046	0.183	0.803	-0.027	-0.595	0.270	0.028	-0.389
Measured Hypertension/Rx ←								
Wave IV OBJ.	0.420	0.151	0.005	0.347	0.704	0.232	0.002	0.468
Wave IV SUBJ.	0.193	0.213	0.364	0.115	-0.070	0.241	0.771	-0.048

CRP ←								
Wave IV OBJ.	0.279	0.054	0.000	0.312	0.443	0.062	0.000	0.634
Wave IV SUBJ.	0.041	0.078	0.595	0.033	-0.239	0.062	0.000	-0.352
Latent Depression ←								
Wave IV OBJ.	-0.016	0.064	0.801	-0.020	-0.069	0.083	0.406	-0.074
Wave IV SUBJ.	0.137	0.084	0.103	0.121	0.040	0.086	0.641	0.044
Depression Dx ←								
Wave IV OBJ.	-0.158	0.100	0.115	-0.131	-0.339	0.213	0.111	-0.225
Wave IV SUBJ.	0.456	0.143	0.001	0.271	0.292	0.204	0.152	0.199
Anxiety Dx ←								
Wave IV OBJ.	-0.353	0.118	0.003	-0.291	-0.407	0.212	0.055	-0.271
Wave IV SUBJ.	0.579	0.179	0.001	0.344	0.378	0.207	0.068	0.258
Trouble Sleeping ←								
Wave IV OBJ.	-0.050	0.119	0.677	-0.030	0.083	0.149	0.577	0.042
Wave IV SUBJ.	0.302	0.167	0.071	0.133	-0.105	0.149	0.479	-0.055
<i>SWS and BMI Regressions</i>								
Wave IV OBJ. ←								
Wave III OBJ.	0.954	0.061	0.000	0.803	1.109	0.129	0.000	0.926
Wave III SUBJ.	0.125	0.079	0.112	0.091	-0.014	0.117	0.904	-0.014
Wave III OBJ. ←								
Wave II OBJ.	0.882	0.042	0.000	0.667	0.668	0.067	0.000	0.599
Wave II SUBJ.	0.263	0.033	0.000	0.248	0.256	0.042	0.000	0.304
Wave II OBJ. ←								
Wave I OBJ.	1.052	0.063	0.000	0.870	0.911	0.099	0.000	0.816
Wave I SUBJ.	0.049	0.032	0.127	0.062	0.087	0.046	0.062	0.118
Wave IV SUBJ. ←								
Wave III SUBJ.	1.019	0.092	0.000	1.025	0.868	0.213	0.000	0.817
Wave III OBJ.	-0.794	0.098	0.000	-0.927	-1.292	0.317	0.000	-1.050
Wave IV OBJ.	0.654	0.076	0.000	0.908	1.196	0.218	0.000	1.165
Wave III SUBJ. ←								
Wave II SUBJ.	0.373	0.032	0.000	0.407	0.306	0.056	0.000	0.314
Wave II OBJ.	-0.475	0.086	0.000	-0.417	-0.353	0.106	0.001	-0.273
Wave III OBJ.	0.849	0.081	0.000	0.985	1.054	0.141	0.000	0.910

Wave II SUBJ. ←								
Wave I SUBJ.	0.848	0.055	0.000	0.870	0.819	0.049	0.000	0.844
Wave I OBJ.	-0.275	0.214	0.178	-0.183	-0.225	0.227	0.322	-0.152
Wave II OBJ.	0.345	0.171	0.078	0.277	0.361	0.197	0.066	0.272
Wave I SUBJ. ←								
Wave I OBJ.	0.989	0.054	0.000	0.641	1.075	0.072	0.000	0.704

*R-Square*

Wave I BMI	0.801	0.788
Wave II BMI	0.854	0.823
Wave III BMI	0.814	0.681
Wave IV BMI	0.860	0.753
Wave I PW	0.728	0.755
Wave II PW	0.717	0.744
Wave III PW	0.568	0.684
Wave IV PW	0.565	0.710
Wave II OBJ.	0.831	0.815
Wave III OBJ.	0.722	0.713
Wave IV OBJ.	0.786	0.835
Wave I SUBJ.	0.411	0.495
Wave II SUBJ.	0.870	0.873
Wave III SUBJ.	0.955	0.891
Wave IV SUBJ.	0.909	0.909
Wave V BMI	0.706	0.680
L-SBP	0.184	0.196
SBP #1	0.724	0.778
SBP #2	0.774	0.809
SBP #3	0.792	0.775
L-DBP	0.133	0.127
DBP #1	0.791	0.803
DBP #2	0.813	0.840
DBP #3	0.792	0.798
Measured Hypertension/Rx	0.208	0.182
CRP	0.117	0.130



L-Depression	0.010	0.002
Feel Depressed	0.866	0.860
Had Blues	0.645	0.565
Feel Sad	0.536	0.480
Depression Dx	0.024	0.011
Anxiety Dx	0.016	0.016
Trouble Sleeping	0.011	0.001

N(Female)=4,152; N(Male)=2,953.

Covariances, means and intercepts, and variances omitted for parsimony.

Results based on casewise maximum likelihood (also called FIML) estimation to account for missing data, with diagonal weighted least squares standard errors due to binary outcome variables.

Weighted estimates account for person-level longitudinal survey weights (WI-V), school-level clustering, and regional strata.

Model fit statistics for Female respondents:  $\chi^2 = 203.767$ , DF = 169, BIC = -1204.230, CFI = 0.997, TLI = 0.995, 1-RMSEA = 0.993.

Model fit statistics for Male respondents:  $\chi^2 = 190.529$ , DF = 169, BIC = -1159.878, CFI = 0.998, TLI = 0.996, 1-RMSEA = 0.993.

Tables A.13 and A.14, in the Appendix, demonstrate that the estimates and patterns noted above are largely unchanged when accounting for individuals' age, race and ethnicity, and educational attainment (or parent's educational attainment in Waves I and II). A key difference is that CRP is not positively associated with either objective and subjective weight in the overall model, but instead negatively associated with Wave IV subjective weight (-0.153). Also, the association between Wave IV subjective weight and latent depression is significant in both the overall model (0.120), and among females (0.209). The unstandardized coefficient estimates associated with the covariates are generally consistent with extant knowledge of how gender, race/ethnicity, and educational attainment are associated with health outcomes. Females have lower blood pressure and hypertension, whereas non-Hispanic Black adults have elevated blood pressure and a higher likelihood of hypertension relative to their non-Hispanic White counterparts. However, females have higher CRP, on average, as well as worse mental health with respect to diagnosed depression and diagnosed anxiety. These same measures of mental health are generally lower among all race/ethnic groups relative to non-Hispanic White adults, and the same pattern for gender and race/ethnicity is observed for trouble sleeping. College-educated respondents and children with parents who are more highly educated generally have lower BMIs.

Finally, simply looking at the association between Wave IV objective and subjective weight and Wave V health outcomes does not necessarily provide a complete account of how the intersecting trajectories of objective and subjective weight are associated with adult health. Namely, the estimates between the Wave IV latent variables for weight and the various Wave V outcomes are *net* of the autoregressive, cross-lagged, and direct effects; the resulting coefficients are not indicative of how these pathways of subjective and objective weight interact in having both direct and *indirect* effects on adult health. This is not to suggest the estimates described above have no meaning – rather, they are not clearly interpretable given the complexity of negative and positive pathways preceding them, as they reflect exclusively “short-run” versus “long-run” effects (Zyphur et al. 2020). Thus, it is more instructive to consider how subjective and objective weight in Wave I are associated with Wave V health outcomes as a function of these intersecting trajectories, which can be accomplished by estimating the total effects, as in

past research using cross-lagged panel models (Kane et al. 2018; Zyphur et al. 2020). Moreover, it is interesting to compare how much of the total effect from either Wave I objective weight or subjective weight on a given Wave V outcome occurs through the “path dependent,” or autoregressive trajectory, as compared to how these measures are related to one another over time. With five waves of data linked through autoregressive, cross-lagged, and direct effects, one should not expect very large effects due to considerable attenuation over time on account of many multiplicative terms, but the general direction of the effect and relative magnitudes are of interest rather than the size (Adachi and Willoughby 2015). Both the total and path dependent effects for the different health outcomes are shown in Table 4.8, adjusting for age, gender, race and ethnicity, and educational attainment.

**Table 4.8** Total and Indirect Effects for Health Outcomes Regressed on Subjective Weight (SUBJ.) and Objective Weight (OBJ.), Adjusted for Age, Gender, Race/Ethnicity, and Education

Parameters	Estimate	Std. Err.	P-value	Stdz. Est.
Wave V BMI ←				
Wave I OBJ. Total Effect	1.017	0.066	0.000	0.524
Wave I OBJ. Path Dependent Effect	0.690	0.111	0.000	0.355
Wave I SUBJ. Total Effect	0.307	0.051	0.000	0.251
Wave I SUBJ. Path Dependent Effect	-0.013	0.022	0.548	-0.011
SBP ←				
Wave I OBJ. Total Effect	0.704	0.074	0.000	0.220
Wave I OBJ. Path Dependent Effect	0.478	0.152	0.002	0.150
Wave I SUBJ. Total Effect	0.212	0.066	0.001	0.105
Wave I SUBJ. Path Dependent Effect	-0.010	0.055	0.855	-0.005
DBP ←				
Wave I OBJ. Total Effect	0.434	0.055	0.000	0.180

Wave I OBJ. Path Dependent Effect	0.300	0.118	0.011	0.124
Wave I SUBJ. Total Effect	0.128	0.052	0.013	0.084
Wave I SUBJ. Path Dependent Effect	-0.009	0.047	0.851	-0.006
Measured Hypertension/Rx ←				
Wave I OBJ. Total Effect	0.597	0.062	0.000	0.247
Wave I OBJ. Path Dependent Effect	0.252	0.107	0.018	0.104
Wave I SUBJ. Total Effect	0.264	0.051	0.000	0.173
Wave I SUBJ. Path Dependent Effect	0.077	0.046	0.098	0.050
CRP ←				
Wave I OBJ. Total Effect	0.307	0.025	0.000	0.207
Wave I OBJ. Path Dependent Effect	0.276	0.047	0.000	0.186
Wave I SUBJ. Total Effect	0.056	0.021	0.009	0.060
Wave I SUBJ. Path Dependent Effect	-0.042	0.015	0.006	-0.044
Latent Depression ←				
Wave I OBJ. Total Effect	-0.014	0.021	0.522	-0.009
Wave I OBJ. Path Dependent Effect	-0.071	0.037	0.056	-0.048
Wave I SUBJ. Total Effect	0.029	0.016	0.066	0.031
Wave I SUBJ. Path Dependent Effect	0.034	0.017	0.047	0.037
Depression Dx ←				
Wave I OBJ. Total Effect	0.039	0.043	0.368	0.016
Wave I OBJ. Path Dependent Effect	-0.134	0.080	0.096	-0.055
Wave I SUBJ. Total Effect	0.099	0.036	0.007	0.065
Wave I SUBJ. Path Dependent Effect	0.088	0.039	0.023	0.058
Anxiety Dx ←				

Wave I OBJ. Total Effect	-0.052	0.046	0.265	-0.021
Wave I OBJ. Path Dependent Effect	-0.222	0.086	0.009	-0.091
Wave I SUBJ. Total Effect	0.086	0.041	0.034	0.056
Wave I SUBJ. Path Dependent Effect	0.104	0.041	0.010	0.068
Trouble Sleeping ←				
Wave I OBJ. Total Effect	0.016	0.028	0.564	0.008
Wave I OBJ. Path Dependent Effect	0.004	0.029	0.902	0.002
Wave I SUBJ. Total Effect	0.040	0.036	0.270	0.013
Wave I SUBJ. Path Dependent Effect	0.019	0.063	0.759	0.006

N=6,247.

"Path Dependent" effect refers to autoregressive trajectory for OBJ. and SUBJ.:  
W1 → W2 → W3 → W4.

Observed BMI, observed SBP and DBP, and observed CRP  
divided by 10 to reduce variance and help with model convergence.

Results based on casewise maximum likelihood (also called FIML) estimation to  
account for missing data among endogenous variables, with diagonal weighted least  
squares standard errors due to binary outcome variables.

Weighted estimates account for person-level longitudinal survey weights (WI-V),  
school-level clustering, and regional strata.

Model fit statistics:  $\chi^2 = 361.617$ ,  $DF = 274$ ,  $SBIC = -2033.104$ ,  $CFI = 0.994$ ,  
 $TLI = 0.991$ ,  $1-RMSEA = 0.993$ .

A one standard deviation (SD) increase in objective weight at Wave I continues to be positively associated with a 0.524 SD increase in BMI at Wave V, with approximately two-thirds of the effect attributable to path dependency in BMI. Though the path dependent effect of subjective weight on Wave V BMI is nonexistent, a one SD increase in Wave I subjective weight is associated with a 0.307 SD increase in Wave V BMI due to its intersecting association with objective weight over time. There is a similar pattern for SBP, DBP, and hypertension, such that a one SD increase in objective weight at Wave I is significantly associated with increased SBP, DBP, and hypertension at Wave V (0.220, 0.180, 0.247

SD, respectively). Approximately 70% of the total effect between Wave I objective and SBP and DBP is attributable to path dependency, compared to ~40% for hypertension or medication use. Subjective weight has no path dependent association with any of the blood pressure outcomes, but has a small positive association through objective weight (0.105, 0.084, 0.060 SD, respectively). The association between Wave I objective weight and Wave V inflammation, on the basis of CRP, is positive, such that a one SD increase in objective weight is associated with a 0.207 SD increase in CRP, with almost the entirety of the effect through BMI rather than objective weight (~90%). As before, there is no path dependent association between subjective weight and CRP, but there is a small and positive total effect (0.060 SD).

The inverse pattern is observed for the total and path dependent effects between Wave I objective and subjective weight and Wave V mental health outcomes. There is no evidence of an association between objective weight and latent depression, but a small positive association between subjective weight and latent depression (0.031 SD), which would be larger if not for the association between subjective and objective weight over time (0.037 SD). Likewise, there is no association between objective weight and diagnosed depression, but a small positive association with Wave I subjective weight (0.065 SD). Wave V anxiety diagnosis is also not associated with Wave I objective weight, though its path dependent effect has a small, negative and significant association (-0.091 SD); however, the association with Wave I subjective weight is small but positive (0.056 SD). There does not appear to be any significant association between either objective or subjective weight and trouble sleeping.

Finally, I examine gender differences in these total and path dependent effects in Table 4.9. The similarity of the model structure for both female and male respondents appears to result in similar patterns in the direct and path dependent effects compared to the overall sample, with respect to both their general direction and magnitude. Nevertheless, key deviations from the overall pattern – indicative of important gender differences – are observed as well. Generally, the association between Wave I objective weight and both SBP and DBP is greater for males compared to females (0.276 vs. 0.190 SD; 0.216 vs. 0.146 SD), especially on account of the path dependent effect. The association between CRP and Wave I

objective weight appears somewhat stronger for males compared to females, also on account of the path dependent effect. However, the total effect between CRP and Wave I subjective is positive and significant for females (0.096 SD) and not significant for males, for whom there is a significant negative path dependent effect (-0.083 SD). There is a clear gender difference in total effects for Wave I subjective weight on latent depression, wherein there is a positive total (0.074 SD) and path dependent effect (0.065 SD) compared to seemingly no relationship for male respondents. The same finding is true of diagnosed depression and anxiety, where the effect from Wave I subjective weight on these outcomes is positive and significant for female respondents (0.108 and 0.096 SD, respectively) but not their male counterparts. Finally, while there were no significant effects from Wave I objective and subjective weight on trouble sleeping in the overall sample, the stratified results show that a one SD increase in Wave I objective weight is associated with a 0.043 standard deviation increase in trouble sleeping among female respondents.

**Table 4.9** Total and Indirect Effects for Health Outcomes Regressed on Subjective Weight (SUBJ.) and Objective Weight (OBJ.), Adjusted for Age, Race/Ethnicity, and Education; Female vs. Male

Parameters	Female				Male			
	Estimate	Std. Err.	P-value	Stdz. Est.	Estimate	Std. Err.	P-value	Stdz. Est.
Wave V BMI ←								
Wave I OBJ. Total Effect	1.043	0.082	0.000	0.492	0.922	0.089	0.000	0.539
Wave I OBJ. Path Dependent Effect	0.688	0.119	0.000	0.325	0.609	0.178	0.001	0.356
Wave I SUBJ. Total Effect	0.315	0.064	0.000	0.230	0.319	0.085	0.000	0.282
Wave I SUBJ. Path Dependent Effect	-0.040	0.033	0.227	-0.029	0.003	0.029	0.917	0.003
SBP ←								
Wave I OBJ. Total Effect	0.563	0.085	0.000	0.190	0.830	0.123	0.000	0.276
Wave I OBJ. Path Dependent Effect	0.363	0.145	0.012	0.122	0.682	0.287	0.017	0.227
Wave I SUBJ. Total Effect	0.176	0.071	0.013	0.092	0.237	0.107	0.027	0.119
Wave I SUBJ. Path Dependent Effect	-0.016	0.069	0.818	-0.008	-0.055	0.068	0.421	-0.028
DBP ←								
Wave I OBJ. Total Effect	0.335	0.057	0.000	0.146	0.510	0.093	0.000	0.216
Wave I OBJ. Path Dependent Effect	0.112	0.094	0.233	0.049	0.532	0.223	0.017	0.226
Wave I SUBJ. Total Effect	0.178	0.050	0.000	0.120	0.103	0.079	0.188	0.066





Wave I OBJ. Total Effect	0.144	0.058	0.014	0.060	-0.111	0.071	0.116	-0.048
Wave I OBJ. Path Dependent Effect	-0.082	0.090	0.366	-0.034	-0.228	0.156	0.143	-0.099
Wave I SUBJ. Total Effect	0.168	0.053	0.001	0.108	0.020	0.042	0.639	0.013
Wave I SUBJ. Path Dependent Effect	0.115	0.051	0.024	0.074	0.066	0.049	0.177	0.044
Anxiety Dx ←								
Wave I OBJ. Total Effect	0.010	0.051	0.843	0.004	-0.161	0.075	0.032	-0.070
Wave I OBJ. Path Dependent Effect	-0.203	0.092	0.028	-0.084	-0.311	0.161	0.053	-0.135
Wave I SUBJ. Total Effect	0.151	0.058	0.009	0.096	0.021	0.050	0.673	0.014
Wave I SUBJ. Path Dependent Effect	0.143	0.056	0.010	0.091	0.088	0.052	0.091	0.058
Trouble Sleeping ←								
Wave I OBJ. Total Effect	0.136	0.052	0.009	0.043	-0.044	0.057	0.440	-0.015
Wave I OBJ. Path Dependent Effect	0.041	0.081	0.617	0.013	0.047	0.097	0.626	0.016
Wave I SUBJ. Total Effect	0.076	0.046	0.096	0.037	-0.044	0.033	0.183	-0.022
Wave I SUBJ. Path Dependent Effect	0.029	0.045	0.523	0.014	-0.033	0.035	0.350	-0.017

N(Female)=4,152; N(Male)=2,606.

"Path Dependent" effect refers to autoregressive trajectory for OBJ. and SUBJ.:  $W1 \rightarrow W2 \rightarrow W3 \rightarrow W4$ .

Observed BMI, observed SBP and DBP, and observed CRP divided by 10 to reduce variance and help with model convergence.

Results based on casewise maximum likelihood (also called FIML) estimation to account for missing data among endogenous variables, with diagonal weighted least squares standard errors due to binary outcome variables.

Weighted estimates account for person-level longitudinal survey weights (WI-V), school-level clustering, and regional strata.

Model fit statistics for Male respondents:  $\chi^2 = 295.971$ , DF = 268, SBIC = -1812.002, CFI = 0.996, TLI = 0.993, 1-RMSEA = 0.994.

Model fit statistics for Female respondents:  $\chi^2 = 369.979$ , DF = 268, SBIC = -1827.625, CFI = 0.990, TLI = 0.983, 1-RMSEA = 0.990.

## Discussion and Conclusion

This project seeks to broaden conceptualization and measurement in health research by emphasizing the utility of both objective and subjective measures when examining the relationship between body size and health. Researchers should be cognizant of reporting issues and biases associated with subjective measures; however, “self-reported data should not axiomatically be characterized as inferior solely because they come from respondents” (Ferraro and Farmer 1999: 313). Indeed, Goldman et al. (2004) suggest that “rather than enhance our efforts to collect so-called objective measures through physicians’ reports and biomarkers, we may need to focus on aspects of well-being that are notoriously difficult to measure, such as mental and emotional health limitations imposed by health conditions” (p.56). In obesity research, the body mass index continues to be the “gold standard” for objective weight and health (Gutin 2018; Nicholls 2013); yet much of the poor health and physiological dysregulation that we associate with obesity and body weight is not necessarily captured by this measure (Tomiyama et al. 2018). Giving priority to this “objective” measure might be unwarranted, as would dismissing individuals’ perceptions of and feelings about their body weight, in a society where social and health norms about the body and its appearance are so highly intertwined (Gutin 2021).

Indeed, much of the extant research on perceived weight has focused on its relationship with objective body size, rather than considered the extent to which it provides key insights on the psychosocial aspects of body size and weight. Sociological research on the function of the body in society (Bourdieu 1984; Fox 2012), and the stigma attached to overweight bodies as “deviant” (Cahnman 1968; Maddox et al. 1968), suggests that individuals’ subjective weight taps into the social *experiences* associated with overweight and obesity, and thus requires further scrutiny. In line with past research on the importance of subjective measures, this study considers the longitudinal measurement properties of subjective weight and then uses this knowledge to provide estimates of its relationship with key adult health outcomes while accounting for the complex and intersecting relationship with objective weight over time. As such, the primary takeaways from this study – and their substantive implications – consider

how subjective weight is both separate from and related to objective weight over time, as well as how these two trajectories both influence adult physical and mental health.

Firstly, this study provides needed context on past research describing “misperception” in the extent to which individuals’ subjective and objective weight track with one another. Namely, this lack of one-to-one correspondence is not surprising given the relatively stability of subjective weight over time, and its strong wave-to-wave predictive power, even across observations separated by multiple years. Indeed, the strong autoregressive relationship largely persists after accounting for how subjective weight intersects with objective body weight. While a true notion of stability would be more consistent with evidence of some kind of enduring latent influence (Bollen and Gutin [Forthcoming]), a “time-invariant” model including such a latent intercept did not have better fit to the Add Health data. Yet, the best-fitting autoregressive model suggests that individuals’ subjective weight is largely influenced by their prior, or pre-existing, views of their weight – as indicated by the large proportion of variance in latent subjective weight explained by this model. Based on this model, one may assume that individuals’ weight “identities” form fairly early in life and largely persist throughout the life course (Blaxter 2004; Fox and Ward 2008; Sobal and Maurer 2017; Whyte 2009), consistent with past research on relative stability in the measure and the notion that weight perceptions may have a heritable component (Wedow et al. 2016; Wedow et al. 2018). Subjective weight is clearly associated with individuals’ objective weight – as evidenced by the large direct effect of objective weight on subjective weight within waves – but this association is not the driving factor underlying the longitudinal trajectory of subjective weight.

However, the strong predictive power of the autoregressive model does not imply that the wave-to-wave relationship between subjective weight and both itself and objective weight is consistent over time; there are notable deviations in the autoregressive estimates, as well as the cross-lagged and direct relations. Although a “growth”-based model, consistent with a life course pattern, was not a good fit for the data, the autoregressive model exhibits less stability – for both women and men – in the “transitional” stage in life between Waves II and III when many respondents are leaving their homes, completing their education, and starting both careers and families (Elder et al. 2003; Shanahan 2000). The

lagged effect of subjective weight is weaker, while there is a significant *positive* cross-lagged effect from subjective weight to objective weight, suggesting that perceiving oneself as overweight is associated with future weight gain. Indeed, the more interlinked relationship between the two measures between these two waves is consistent with research showing how many of the social institutions that rise to prominence at this point in the life course are subject to different forms of weight bias and discrimination, which may affect how much influence subjective and objective weight have on one another. For instance, a large body of research documents weight discrimination and bias in educational and workplace environments, as well as in romantic relationships and marriage (Puhl and Heuer 2010; Puhl et al. 2008; Varney 2014). Individuals' *awareness* of their bodies and their weight – and the stress associated with having the wrong body or body weight – may be exacerbated at these ages, leading to the “paradoxical” relationship between believing oneself to be overweight and gaining weight observed in past research (Daly et al. 2017; Tomiyama et al. 2018; Unger et al. 2017). Of course, the available evidence in this study is only suggestive of these explanations, and the inclusion of relevant contextual variables at these ages would be required to substantiate these claims.

The significant *negative* association between objective weight and future subjective weight – implying that higher body size is associated with a lower perception of one's weight – provides additional context on the complex relationship between these two measures over time. Indeed, this finding speaks to extant literature on the “normalization” of weight over the life course, as individuals may *acclimate* to having a larger body size and weight as they age (Smith and Holm 2011). Given broader trends of population-wide weight gain over the life course (Lee et al. 2010; Mizuno et al. 2004) – especially among this cohort of adults (Gordon-Larsen et al. 2010) – it is possible that normalization is not exclusively a function of individual's weight, but also a function of their peers and those in their community (Burke and Heiland 2007; Burke and Heiland 2018; Christakis and Fowler 2007; Robinson 2017; Wedow et al. 2018). Once again, additional information would be necessary to further explore these claims. However, it is important to note that the *total effect* of objective weight on subsequent subjective weight is positive, as this direct, negative cross-lagged effect is offset by a larger indirect positive effect through prior

subjective weight and subsequent objective weight. Namely, even if individuals may “get used to” having a larger body – on the basis of individual or social acclimation, or both – this does not counteract the strong lagged influence of subjective weight, or how lagged objective weight continues to influence subsequent subjective weight.

Importantly, the high degree of stability in both subjective and objective weight challenges the premise of many public health interventions seeking to increase individuals’ “awareness” of their being overweight (Daly et al. 2017; Daly et al. 2019; Haynes et al. 2018; Robinson et al. 2017). The underlying logic is that that awareness engenders better health behaviors and lifestyles that lead to weight loss and better health; however, this approach is ignorant of evidence in this study – and others – suggesting that subjective and objective weight represent separate constructs that follow independent, lagged trajectories.

One key implication from this study is that such interventions are likely to be ineffective, as individuals’ subjective weight has minimal to no influence on their future objective weight or – in the previously described transition from Waves II to III – is actually associated with *weight gain*. Namely, a key obstacle to population-wide weight loss is not a lack of awareness but individuals’ inability to act on this knowledge should they choose to. Decades of research on obesity conclusively demonstrate that it the product of *structural* issues in the United States, wherein change requires modifications to individuals’ environments that engender organic action as a function of accessible and easily integrated everyday activities and behaviors (Novak and Brownell 2011; Novak and Brownell 2012; Schwartz and Brownell 2007), rather than as a function of individuals’ agency (Adams et al. 2016). Indeed, the misplaced focus on individuals’ perceptions of their weight is an important consideration in the harmful and counterproductive framing of obesity as a function of individuals’ lack of willpower, poor choice, and ignorance of healthful knowledge and practices (Brownell et al. 2010; Ciciurkaite and Perry 2018; Puhl and Brownell 2003; Saguy 2012; Shugart 2016) – beliefs that have been shown to *reduce* support for obesity-related public policy (Barry et al. 2009). Emphasis on misperception perpetuates the focus on the individual and their personal failures, despite broad consensus that most U.S. adults are at the whim of

macro-level social, economic, and political forces acting on their weight and health that are difficult – if not impossible – to fully disengage from at the individual level.

Indeed, acknowledgement of subjective and objective weight as separate entities speaks to the larger issue of how researchers conceptualize individuals' health as a function of both the physiological and psychosocial aspects of body weight. Substantiating past research on the subject (Daly et al. 2017; Daly et al. 2019; Haynes et al. 2018; Haynes et al. 2019; Frisco et al. 2010; Robinson et al. 2017; Tomiyama et al. 2018; Unger et al. 2017), this study demonstrates that individuals' perceptions of their weight are tied to multiple negative health outcomes independent of their body mass index. However, this study builds on past work in accounting for measurement error and the longitudinal and intersecting nature of the relationship between subjective and objective weight. Consequently, this study underscores a key distinction between these different aspects of weight and their associations with physiological and psychosocial outcomes, as well as gender differences in these associations.

Unsurprisingly, objective body size continues to be associated with many of the negative physiological health outcomes identified in past research, such as elevated blood pressure and inflammation. However, there is no evidence of a strong association between objective weight and worse mental health, as these dimensions of health appear to be more closely associated with subjective weight and how dissatisfaction with one's weight – or knowledge that it is not “right” – takes a toll on one's health. Given the gendered context for body weight and image in the United States, the negative mental health associated with subjective weight is most apparent for female respondents, both in terms of magnitude and statistical significance. However, the intersecting trajectories of objective and subjective weight throughout the life course provides evidence that subjective weight is *indirectly* associated with worse physiological health *through* its relationship with objective weight.

These results clearly show that both aspects of weight – the objective reality of having a higher body weight, and the subjective experience of perceiving oneself as overweight – are key determinants of individuals' overall health and wellbeing. However, extant perspectives on overweight and obesity as public health issues often fall into two, opposing ideological camps. Emphasis on body size as an



indicator of health, and obesity as a disease, makes body weight the direct target of interventions aimed at reducing individuals' BMIs; by contrast, emphasis on body size as a socially defined measure of "normality" champions body positivity and Health at Every Size, arguing that a focus on weight loss should not come at the expense of other dimensions of individuals' physical and mental health (Bacon and Aphramor 2011; Gutin 2021; Kraschnewski et al. 2010; Mann et al. 2007). Many obesity researchers recognize body weight as both a physiological and social source of stress; yet, strategies aimed at targeting one pathway may not account for lingering effects brought on by the other. Indeed, the fact that body size is both a physical and social trait means that an "either/or" binary towards improving individuals' physiological or psychosocial health is inappropriate, and potentially counterproductive. While this complicates the narrative of addressing body weight at both the individual and population level, intervening on both the physical and social aspects of body weight is likely to produce more lasting and comprehensive change than interventions premised on the notion that lower body weight is a guarantee to better health or that a higher body weight is not consequential to one's health.

Namely, there is truth in both perspectives, but less acknowledgement of how they reflect different sets of individual and structural solutions, especially in the case of subjective weight. On the one hand, BMI is not a definitive marker of health (Gutin 2018), and obesity is not a monolithic state of disease or impairment, such that the qualitative labels attached to objective weight can cause more harm than good (Greenhalgh 2015; Jutel 2011; Jutel 2014). Yet, it is important to recognize and respond to the fact that many individuals stand to benefit from losing weight and seeing improvement in their health. Unfortunately, physicians and researchers often have a limited view of body size as a measure or marker of health, lacking important social or psychological context for what body size means to a given individual and how that subjective meaning is implicated in current and future health. For instance, many clinicians and researchers advocate for expanding the set of physiological indicators used to assess individuals' health in relation to their body weight, thus providing greater nuance in distinguishing between real and misplaced concern about overweight and obesity as health risks (Garvey et al. 2014; Guo and Garvey 2016; Guo et al. 2014). Should individuals' psychosocial contexts and subjective

experiences be taken into consideration as well? This is largely a rhetorical question, as diagnostic protocol is governed by its own set of structural constraints and guidelines (Jutel 2014; Rosenberg 2002). Nonetheless, the findings in this study and elsewhere suggest that subjective weight is an underappreciated factor in how researchers and care providers evaluate the costs and benefits associated with weight loss focused interventions.

This is not to suggest that the subjective experience and psychosocial ramifications of body weight are entirely absent in discourse on overweight and obesity. However, the growing call for greater body positivity and body diversity in relation to health has largely been framed in the language of advocacy (Cohen et al. 2020; Cwynar-Horta 2016; Friedman et al. 2019; Lazuka et al. 2020; Webb et al. 2017), rather than acknowledged as a legitimate effort to recognize the health implications of individuals' bodies as a source of stigma that is implicated in the physiological and psychosocial consequences of obesity.

In recognizing the difficulty – if not outright futility – of sustainable, long-term weight loss for much of the population (Puhl et al. 2020), many researchers now stress the importance of intervening on the *social* mechanisms leading to worse health and wellbeing among children and adults with overweight and obesity. Puhl and colleagues have spent decades chronicling the myriad social pathways and factors in the workplace, educational settings, healthcare, interpersonal relationships, and media lead to worse treatment and fewer rewards for individuals with overweight and obesity (Pearl 2018; Pearl and Puhl 2018; Puhl and Brownell 2001; Puhl and Brownell 2003; Puhl and Heuer 2009; Puhl and Heuer 2010; Puhl et al. 2005; Puhl et al. 2020), directly impacting their socioeconomic prospects, quality of life, and health. These various forms of weight stigma, bias, and discrimination represent psychosocial mechanisms that cannot – and should not – be addressed by interventions premised on individual weight loss. Rather, they represent institutional sources of injustice and inequity that require institutional-level action, such as legislation, policies, and education or training that targets hiring and pay discrimination among employers, bullying and unfair treatment by both peers and teachers in educational settings, implicit bias and negligence among physicians, and inaccurate or defamatory news coverage, among

many other plausible interventions (Pearl 2018; Pearl et al. 2017; Puhl et al. 2020). Critically, public health messaging that avoids equating weight loss with individuals' health – or avoids implicating individuals' as being flawed due to their having overweight or obesity – is shown to be more effective in encouraging healthy behaviors and lifestyles (Pearl 2018).

### *Limitations*

Prior to concluding, I note some limitations of the study, and how addressing them can help advance research on subjective weight, weight-related stigma, and broader questions surrounding body weight and health. As mentioned, the key variable of interest – individuals' perceptions of their weight – is an imperfect measure of the much broader constructs of body image and body satisfaction that are relevant to this study (Durso and Latner 2008; Lillis et al. 2010; Sandoz et al. 2013). Though the use of perceived weight in this analysis is consistent with past work, a multi-dimensional perceptual measure would allow for a more comprehensive assessment of subjective weight and its implications for psychosocial wellbeing, as well as how it tracks with objective weight and body size over time. This may be especially important in the case of explaining differences between female and male respondents, as past work has shown that weight, in and of itself, is a less focal issue for men as compared to body composition and muscularity (Grogan 2007; Pope et al. 2000). It is understandable that a large, longitudinal data set like Add Health has limited space for additional questions; however, the inclusion of questions about weight discrimination in Waves IV and V offers an interesting opportunity to examine them as key mediators, or additional pathways, connecting subjective and objective weight and how they are associated with Wave V health outcomes. Indeed, preliminary analyses from these data suggest that individuals' experiences of discrimination on the basis of their weight are strongly associated with both objective and subjective weight, as well as a number of physical and mental health outcomes. More explicit consideration of weight-based discrimination – as well discrimination on the basis of physical appearance – is an important line of future research

There are also some limitations in the flexibility with which trajectories could be modeled in the analysis. The Add Health data are advantageous in covering different periods of the life course and

having high-quality health data; however, the number of waves is somewhat limited when assessing more complex longitudinal models, such as those including non-linear slopes (Bauldry and Bollen 2018). Moreover, the spacing between waves is inconsistent, which makes the interpretability of the autoregressive coefficients challenging (Kuiper and Ryan 2018). The comparison of models in these analyses was fairly definitive with respect to the choice of autoregressive models for both subjective and objective weight, but additional research can be done to validate these conclusions. To my knowledge, the NLSY97 is the only other comparable data set that tracks these two measures over a similar period in the life course, with fewer years separating waves. Even though the NLSY97 data are self-reported and lack as comprehensive a set of health outcomes, they can help demonstrate if the lagged effects trajectory continues to perform well with more time points and fewer years between measures. Past research suggests the autoregressive model would continue to have excellent fit, likely having higher autoregressive coefficients and greater explanatory power, but additional data would allow for better assessments of autoregressive latent trajectory models (Bollen and Gutin [Forthcoming]).

Finally, additional work should be done to examine these trajectories, their associations, and their relation to outcomes among other groups. For one, this sample is age-limited, and some of the more adverse physiological outcomes associated with both subjective and objective weight measures are yet to manifest. The Health and Retirement Study also asks respondents about their subjective weight (Wedow et al. 2018), though the older starting age of the sample precludes the ability to assess how a more lifelong trajectory of subjective weight is associated with adult health.

More importantly, this study only examined gender differences, on account of the gendered reality of body size and associated norms of what is an appropriate weight and appearance (Bordo 2004; Grogan 2007). However, these norms are by no means limited to women and men, as there is also evidence of differences in body weight norms and perceptions on the basis of race and ethnicity, as well as individuals' socioeconomic status (Akan and Grilo 1995; Bennett and Wolin 2006; Cachelin et al. 2002; Dorsey et al. 2009; Fitzgibbon et al. 2000; Gregory et al. 2008; Kronenfeld et al. 2010; Paeratakul et al. 2002; Vaughan et al. 2008; Webb et al. 2014). These are all incredibly important axes through

which to examine these issues, as are additional intersectional frameworks (Ciciurkaite and Perry 2018; Cole 2009; Himmelstein et al. 2017; Watson et al. 2019; Wildes et al. 2001). Social norms about beauty, fitness, and body size are often targeted towards specific groups. Indeed, many have noted that the “thin, fit ideal” in the United States is really a “White, female” ideal (Arciszewski et al. 2012; Greenhalgh 2015; Saguy 2012).

In turn, there is an open question about the health consequences associated with the intersection of these ideals and identities with one’s BMI and perception of weight, given that much of what is understood about body image derives from work focused on White women (Cole 2009). On the one hand, it is plausible that a social comparative framework offers some degree of *protection* from the harm associated with not adhering to these societal ideals – as individuals largely draw on “within” group comparisons that lead them to be more satisfied with their weight and avoid any ensuing psychosocial consequences. Conversely, greater distance from social norms – even those that are targeted towards a group one is not a “member” of – can be *more harmful*, especially if individuals are actively judged by others who compare them to these unattainable ideals, which in turn informs individuals’ own assessments of their weight.

These are important and compelling lines of inquiry, requiring careful examination within this SEM framework. As seen in these analyses, the relative complexity of the models makes formal tests of measurement invariance quite difficult, which proves limiting in making formal comparisons across groups. Indeed, additional consideration of invariance across race and ethnic groups failed to converge in these analyses, suggesting that examination of these differences requires a more systematic approach to model-building wherein group-specific trajectories and intersections between trajectories need to be identified. This work can be integrated with “contextual” data on weight perception – as seen in past research using Add Health data (Wedow et al. 2018) – to better understand the social origins and social contexts for the models seen in these analyses.

## *Conclusion*

Amid high and rising rates of obesity in the United States, it is reasonable for population health researchers to speculate that shifting norms about what constitutes a healthy and normal body size and weight may be implicated as a key contributing cause (Burke et al. 2010). Yet the understanding of how objective and subjective weight are related to one another throughout the population is incomplete without accounting for individuals' tendency to exhibit some degree of stability – or path dependency – in various aspects of their health and perceptions of their health over time; moreover, the relationship between objective and subjective health is likely complex and variable over the life course. Thus, one cannot expect that individuals' objective and subjective weight perfectly track with one another, such that modifying one or the other will result in both a 'healthier' weight and greater satisfaction with one's weight. Rather, subjective and objective weight need to be understood as separate constructs and longitudinal processes, requiring independent study that can help facilitate more nuanced and actionable policies and interventions.

Population health research is cognizant of the importance of subjective measures of health, and this should be no different in the case of subjective weight. Individuals' weight status is a key source of social stigma in the United States (Greenhalgh 2015; Puhh and Heuer 2010); more broadly, individuals' health status – and others' perceptions of or assumptions about individuals' health – is a key determinant of one's social standing and worth (Cockerham 2005; Dew 2012). Health and social norms are inextricably intertwined, with body size and obesity as arguably the most illustrative example. Health is something that individuals experience at both a physiological and psychosocial level yet this is rarely taken into consideration in the study of body weight. Individuals' perceptions of their weight may offer key insight on these psychosocial mechanisms, yet they are often used to gauge whether and how *wrong* individuals are about their weight. In framing this as an issue of misperception, researchers should take a moment to consider the social and cultural biases that lead to such a conclusion – especially in reinforcing the narrative of personal responsibility, knowledge, and decision-making as driving factors underlying overweight and obesity (Brownell et al. 2010; Puhl and Brownell 2003; Saguy 2012; Shugart 2016).

Instead, researchers should consider how and why this discordance occurs, and whether it is truly discordance at all. These more open-ended lines of inquiry allow for future research and theory akin to past work on self-rated health (Jylhä 2009), asking if individuals' responses are based on whether they believe that their body allows them to have a satisfying and healthy life, above and beyond the limited insight provided by height and weight.

Indeed, this study suggests that many of the pathways through which subjective weight affects individuals' health are likely a function of distorted and discriminatory social norms about "healthy," "normal," and thus "good" bodies (Dew 2012; Greenhalgh 2015; Saguy 2012). While excess weight can be and often is detrimental to one's health, it is important to consider how the negative identities and beliefs individuals form in relation to their body weight are consequential as well. The United States, as well as many other countries (Puhl et al. 2015), is a highly weight-conscious nation, with institutional and interpersonal biases against individuals who do not conform to certain expectations of an appropriate and/or desirable body and appearance (Jutel and Beutow 2007). In that sense, body weight is not unlike other axes of inequality that shape individuals' day-to-day lives based on the stereotypes and assumptions that others have based on how a person *looks* (Gutin 2021), and the social and cultural messages that amplify these beliefs. The prevailing message that individuals simply need to lose weight – and conform to these ideals – cannot and should not be the solution to mitigating weight stigma as a harmful and pervasive influence in society.

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## CHAPTER 5: CONCLUSION

Sociologists, public health scientists, and numerous other researchers engaged in the study of population health are in an unenviable position when it comes to understanding body weight and health. The work is important and necessary: body weight continues to be a key determinant of individuals' health and obesity remains a key population health challenge. Yet body weight is also key aspect of individuals' identities, influencing both how they perceive themselves and how they are perceived and treated by others in a highly weight-conscious and body-normative society like the United States (Puhl and Heuer 2010; Saguy 2012). In turn, body weight and size carry many different meanings and assumptions depending on the context in which it is being used or discussed, and the various stakeholders involved (Gutin 2021). This enormity of different ways of thinking about body weight and size – and the numerous biological, physiological, psychological, and social factors and pathways that they reflect – makes population health research particularly challenging, especially when using an imprecise measure like BMI (Gutin 2018).

This dissertation does not represent an effort to resolve this complexity by suggesting there is a single, or most valid, or definitive way of conceptualizing and using body weight in research on health. Rather, it makes a case for *taking advantage of* and *examining* the ambiguity in the relationship between body weight and health to obtain novel insights on body weight as both a physical trait and a social identity, situated at the nexus of health, morality, and normality. By synthesizing research and theory across the many different disciplines and contexts in which body weight is studied, I demonstrate that it represents a complex individual and population health issue that requires an equally complex and comprehensive approach to empirical research. Namely, this dissertation explores three key contexts in which ambiguity surrounding body weight as a health and social issue is paramount, to the extent that



body weight is a focal issue in clinical settings, epidemiological assessments of the population, and individuals' subjective experiences and day-to-day lives. These are arguably the most important contexts shaping collective understanding of weight as a health issue, reflecting different underlying beliefs about the relationship between weight and health and how it should be addressed. However, this dissertation further demonstrates that these contexts are not without their own sources of ambiguity in how body weight is interpreted and used, underscoring the complexity of this ostensibly simple and parsimonious measure.

With respect to the conceptualization and use of body weight in clinical practice, the first chapter finds that uncertainty about what body weight represents in early life is an integral part of the diagnostic process for childhood obesity. Indeed, health practitioners working with children and adolescents and their families often avoid formal diagnostic or medical language and labels out of concern for what terms like “overweight” or “obese” convey about a child’s level of unhealthiness and, in turn, how they influence patients’ perceptions of themselves and families’ perceptions of their children. Instead, I observe a consistent emphasis on framing childhood obesity in the language of prognosis – and locating a child on a trajectory of health and wellbeing – that avoids making definitive pronouncements about a child’s current condition. This prognostic framework is linked to clinicians’ underlying uncertainty about the utility and validity of existing diagnostic tools (Timmermans and Stivers 2018), especially when it comes to interpreting and reacting to patients’ BMIs.

In the context of epidemiologic research on body weight and population health, the second chapter documents considerable heterogeneity in how obesity and poor cardiometabolic health co-occur in the U.S. adult population. Namely, obesity is not a monolith when it comes to its association with poor cardiometabolic health and increased mortality risk. There is much more nuance to the broad category of obesity; many adults with obesity have good cardiometabolic health, and the majority have a cardiometabolic health profile and mortality risk that is indistinguishable from an equally large subset of adults without obesity. However, the *patterning* of these different profiles of body size and cardiometabolic health is clearly social, rather than biological: college-educated adults are much more

likely to be free of any of the risks associated with either higher body weight or cardiometabolic impairment – reaffirming the fact that the health-advantages enjoyed by higher-SES adults cannot be reduced to a single source of risk (Freese and Lutfey 2011; Link and Phelan 1995; Phelan et al. 2010).

The third chapter shows the importance of understanding how the relationship between individuals' body weight and their health is a function of both the physiological and psychosocial aspects of body weight as an objective and subjective construct, respectively. Echoing a growing body of literature on this topic (Daly et al. 2017; Daly et al. 2019; Haynes et al. 2018; Haynes et al. 2019; Frisco et al. 2010; Robinson et al. 2017; Tomiyama et al. 2018; Unger et al. 2017), this chapter demonstrates that individuals' perceptions of their weight are tied to multiple negative health outcomes over and above their body mass index. Objective body size has a clear physiological toll on adult health – as evidenced by increased blood pressure and inflammation – but there is no evidence of an association with worse mental health. Rather, increased depression and anxiety is related to individuals' subjective assessments of themselves as being overweight, and the extent to which that suggests dissatisfaction with one's body and weight. Critically, objective and subjective weight affect one another from early life into adulthood, such that subjective weight is indirectly associated with worse physiological health. In sum, these results show that the objective reality of having a higher body weight and the subjective experience of perceiving oneself as overweight are strongly associated with multiple dimensions of individuals' health and wellbeing.

This dissertation is not without a number of limitations specific to each of these chapters; there are also key questions left unanswered across these studies that serve as the basis for multiple future studies using these and other data. The interview data used in the first chapter may be subject to some degree of selection bias, as they come from a sample of childhood obesity practitioners whose professional interests coincide with the more critical and self-reflexive questions about conceptualization, definition, measurement, and diagnosis being asked in this study. Namely, their views may not be representative of the much broader population of clinicians and health professionals who encounter issues surrounding childhood obesity on a day-to-day basis; nor can these views be safely extrapolated to the

much broader issue of weight and health in other clinical settings. Representativeness is not a prerequisite of qualitative research in medical settings (Britten 1995), but it is important to acknowledge the possibility of variation in diagnosis and communication on childhood obesity across different settings and populations, as well as what determines this variation. Future work on childhood obesity as a diagnosis – and any ambiguity or uncertainty therein – should also incorporate the perspectives of patients and families on the receiving end of this diagnosis, as they represent key stakeholders in the “social diagnosis” framework (Brown et al. 2011). Practitioners and patients often reach different conclusions about the success or failure of a clinical encounter (Lutz 2019), and it is important to understand how aligned their views are in the context of childhood obesity. Though practitioners emphasize the importance of *mutual* understanding, it is unclear to what extent their patients and families experience their relationship as a partnership. For instance, directly observing clinical encounters could reveal important discrepancies in how practitioners *recall* their diagnostic language and advice as compared to what they *actually* say and do in describing patients’ weight and health. Moreover, given clear social disparities in childhood obesity, it may be especially valuable to see how patients’ backgrounds – such as their race and ethnicity or socioeconomic status – influence the degree of concordance between practitioners and patients’ experiences of a clinical visit, and how that influences the long-term trajectory of care and outcomes.

With respect to the second chapter, it is important to acknowledge that both the data and methods used to identify multimorbidity and the co-occurrence of obesity with other cardiometabolic risks represent one of *many* possible approaches to addressing the same question, as evidenced by the large body of past work in this area (Blüher 2020; Primeau et al. 2011; Stefan et al. 2008; Tomiyama et al. 2016; Wildman et al. 2008). Though I examine the sensitivity of the results to alternate specifications and choice of variables, I could not consider all possible combinations of relevant anthropometric and cardiometabolic variables and corresponding cutoffs for “high risk,” which could lead to substantively different class solutions. Likewise, latent class analysis is one of multiple options researchers have for identifying data-driven patterns, and it is important to validate these results using alternate methods.

Beyond these concerns about the robustness of the findings, further work with more recent restricted-use mortality data would allow researchers to include more refined cause-specific analyses and consider important group differences. Results from this study clearly show how the cause of death under consideration leads to different estimates of the associations between groupings of body size and cardiometabolic health and mortality risk, as well as the percent mediation in the educational gradient of mortality risk. Looking at all-cause mortality may be less appropriate than examining causes of death more closely linked to body weight and cardiometabolic dysregulation, as in past research with vital statistics (Masters et al. 2018). These additional restricted data – with a longer follow-up period for mortality – would also facilitate additional group comparisons not examined in this study. Ideally, researchers could identify group-specific latent class solutions and assess their association with mortality risk, given important intersectional differences in key metrics of population health (Bauer 2014; Bowleg 2012). Finally, the availability of rich biomarker data in longitudinal data provides opportunities to examine not only the presence of latent classes but also *transitions* from one class to another as individuals age. This latter analysis would be particularly insightful for understanding how and why the latent classes identified in this study come to be associated with varying levels of mortality risk over time.

The key variable of interest in the third chapter – individuals' perceived weight – is not a comprehensive measure of negative body image, body dissatisfaction, internalized weight bias, weight stigma, weight discrimination, or the many other hypothesized mechanisms thought to explain how and why subjective weight is associated with worse health over and above body mass index (Durso and Latner 2008; Lillis et al. 2010; Puhl and Huer 2010; Sandoz et al. 2013). At present, this study is limited by the availability of more comprehensive subjective measures of the body and weight in longitudinal data, let alone with a large sample size and well-measured physical and mental health outcomes. Thus, I cannot definitively identify the mechanisms underlying the association between subjective weight and worse health, or how it is associated with objective weight over time. Better measurement of the multidimensionality of subjective weight is critical for future work examining additional group differences in both the relationship between subjective and objective weight over time, and how both

measures are associated with adult health. This study emphasizes the importance of gender differences, but there is evidence of differences in body weight norms and perceptions based on individuals' race and ethnicity and socioeconomic status, as well as how they differ based on gender (Bennett and Wolin 2006; Cachelin et al. 2002; Ciciurkaite and Perry 2018; Cole 2009; Dorsey et al. 2009; Fitzgibbon et al. 2000; Gregory et al. 2008; Himmelstein et al. 2017; Kronenfeld et al. 2010; Paeratakul et al. 2002; Vaughan et al. 2008; Watson et al. 2019). While there does not appear to be a longitudinal dataset that allows for a full accounting of the many dimensions of subjective weight identified in psychological research, further work with Add Health may consider how individuals' desire to modify their weight or their experiences with weight discrimination can be incorporated into this structural equation modeling framework. Moreover, the stepwise modeling strategy outlined in this third chapter can serve as a guide for identifying the appropriate group-specific trajectories and intersecting relationships between subjective and objective body weight.

Limitations aside, the results from this dissertation have important substantive implications for the conceptualization and use of body weight in clinical and epidemiologic research and settings, as well as the kinds of interventions and policies that are designed to address the inverse relationship between individuals' weight and their health. For one, these results provide empirical support for the theory that acceptance, rather than avoidance, of uncertainty in diagnosis and clinical decision-making represents a key pathway by which practitioners engage with the various social and non-medical factors that affect patients' lives and health (Brown et al. 2011; Croft et al. 2015). Despite growing awareness of the social determinants of health in the medical field, clinicians have few opportunities to directly engage with their patients' health at this "social" level beyond having greater awareness and empathy for extenuating circumstances in patients' lives (Metzl and Hansen 2014). However, clinicians play a key role in affecting change at the social, rather than the biomedical, level. Reich et al. (2016) provide an actionable framework for integrating fundamental cause theory into medical care, towards the goal of implementing "fundamental interventions." While there are many reasons why viewing a patient through a *biomedical* lens is appropriate in a given situation (Reich et al. 2016), both this study and Reich et al. demonstrate

how broader consideration of patients' social circumstances is often more informative. Clinicians cannot directly intervene on these social factors as they might with other aspects of a patient's health, but they are a prominent voice in advocating for social policy with direct implications for individuals' health (Reich et al. 2016), such as legislation on housing, infrastructure, minimum wage, and many other factors that influence obesity and health more broadly. Echoing the sentiment of many participants in this study, Reich et al. (2016) also describe the plausibility of "needs-based assessments" during clinical visits, that help screen for the *social* factors that have a direct bearing on clinical care and future health. Ideally, clinicians would be part of a wide network of social workers and community health professionals, such that patients and families are connected to individuals and resources that allow them to better adhere to and act on the advice that clinicians provide during a patient visit.

Better assessment of risk is also crucial at the population level, especially when using fairly imprecise measures like BMI or obesity to study health disparities. As the second chapter shows, there is a wide range of substantive groupings of body size and cardiometabolic risks among U.S. adults, such that no single combination of body size and cardiometabolic risk fully explains the educational gradient in mortality. However, certain risk profiles are clearly more concerning than others; obesity is often one of many risk factors of concern and, in some cases, appears to present no additional risk in and of itself. This more nuanced understanding of body size, health, and overall risk raises questions about the continued emphasis on obesity as a key source of social disparities in mortality in the United States. Undoubtedly, obesity is a contributing factor, but it is important to recognize that obesity is primarily a concern when we have more direct evidence of its being the *cause* of poor health (Sharma and Campbell-Scherer 2017). One of the primary justifications for the continued reliance on BMI to define obesity in population health research is its easy of data collection and interpretation (Gutin 2018); these are valuable attributes of any measure, but they should not come at the expense of having an incomplete understanding of population health. Medical researchers have advocated for a more nuanced diagnosis of obesity that integrates other dimensions of individuals' health (Sharma and Kushner 2009). In turn, epidemiologic, population-level research can provide support for these concerns, providing the critical "evidentiary" basis needed to

reevaluate and change clinical standards and protocols (Timmermans and Berg 2003). More broadly, population researchers and policymakers should invest in data collection and design that facilitate for a more holistic understanding of individuals' health and allow for a more comprehensive evaluation of the success of a given intervention or policy beyond a narrow focus on weight loss.

Likewise, the third chapter underscores the importance of expanding population and public health understanding of body weight and obesity as a health concern to be more inclusive of the subjective and psychosocial experience of being “overweight” in a society that values thinness and equates fitness with being a “good” person (Greenhalgh 2015). Obesity researchers recognize that body weight is often both a physiological and social source of stress; however, extant approaches to intervening on the negative health outcomes associated with higher body weight often focus on body weight itself as the target of the intervention and the metric of success. Yet long-term weight loss is unsustainable for the majority of adults (Bacon and Aphramor 2011; Puhl et al. 2020); nor is weight loss the appropriate solution for the many social explanations underlying worse health and wellbeing among children and adults with overweight and obesity. Body weight is a key source of stigma in the United States, and a key factor underlying worse experiences and outcomes in the workplace, educational settings, and healthcare, among many other settings (Puhl et al. 2020). The many forms of weight bias and discrimination that individuals with overweight and obesity encounter represent psychosocial mechanisms for which weight loss is not a panacea. Rather, these are institutional sources of inequality that require institutional-level action, such as legislation, policies, and education or training that targets hiring and pay discrimination among employers, bullying and unfair treatment by both peers and teachers in educational settings, implicit bias and negligence among physicians, and various other targeted interventions (Pearl 2018; Pearl et al. 2017; Puhl et al. 2020). At a more fundamental – and much simpler – level, population researchers studying obesity should avoid the uncritical conflation of elevated risk associated with higher weight and unhealthiness as a state of being (Gutin 2021); this mentality and language is often inaccurate and counterproductive, perpetuating the notion that overweight and obesity represent flawed or deviant identities (Pearl 2018; Puhl and Heuer 2010).

Ultimately, all three projects speak to the broader issue of how researchers, policymakers, and the public at large think about body weight and its relation to health; indeed, the overarching goal of this dissertation is to encourage more critical and reflexive thinking on how social and health norms are intertwined in society, and how that structures negative beliefs and biases towards individuals on the basis of what a number suggests about their health, or what their appearance implies about their standing in society. These biases are pervasive in research and society-at-large; moreover, they are almost entirely superficial in the sense that individuals' weight or appearance provides limited insight on who they are, how they live their lives, or what their health is like.

This kind of superficiality – and its relation to broader issues of inequality and stratification – is well established in sociological research. The notion that phenotypic attributes become imbued with social meaning – and thus become health-relevant traits – is not a novel concept (Link and Phelan 2001). Directly equating body size with race is too strong a comparison, to the extent that race is tied to endemic legacies and systems of oppression (Phelan and Link 2015), but one should not ignore how BMI and race exemplify how one's phenotype affects health through non-biophysiological pathways. The issues surrounding race as an essentialized concept provide a clear illustration of how phenotypic traits are conflated with their social consequences (Frank 2007; Gutin 2019; Morning 2011), wherein race, itself, is assumed to be the innate, causal mechanism underlying poor health. Yet, decades of research prove that the relationship between race and health is attributable to race being a proxy for the many social ills inflicted upon non-White persons via interpersonal and institutional forms of discrimination and disenfranchisement (Phelan and Link 2015). Unfortunately, this message fails to resonate in a society where health is actively used to gauge individuals' social standing (Scambler 2009); the moral judgment attached to healthiness substantiates the belief that those who are unhealthy are 'bad' members of society. This gives rise to a vicious cycle by which the poor health of a marginalized group is used to justify their marginalization, likely leading to worse health in the future. A comparable process of essentializing BMI has been at work for decades, legitimizing BMI as a surrogate marker of biophysiological health, while ignoring the psychosocial implications of its being a marker of appearance and status. Once again,



tautological reasoning is partially to blame; a person becomes unhealthy upon attaining an unhealthy BMI, implying some kind of transition in their latent health.

Decades of sociological research reveal the importance of moving past such superficial biases to uncover the deeper social and institutional mechanisms at work, for which appearance or phenotype is merely a proxy. More broadly, BMI is a marker for health in the same way phenotypic attributes like race and gender are determinants of health; they gauge future risk rather than serve as measures of current health. Certainly, BMI is distinctive – and challenging – as it is not exclusively a marker of appearance and has real biophysiological consequences. Studying the relationship between body size and health is important, but there is a need to better acknowledge uncertainty in what body size represents. The conceptualization of race in health research continues to serve as a useful parallel; there are legitimate concerns about how race being used to perpetuate biogenetic explanations (Bliss 2012; Shim 2002), but recognition that race is a socially-meaningful category is vital for advancing justice and equity (Epstein 2008). Thus, rather than limit discussion to biophysiological explanations for why BMI is associated with adverse health, the inequality framework allows for a broader set of psychosocial pathways and interventions. Again, a direct parallel is inappropriate, but it is informative in showing how limited the understanding of the relationship between body weight and health may be due to these implicit biases in how the association is described and examined.

Conceptualizing body weight in terms of inequality, and advocating for weight neutrality, is more than just a rhetorical device. A weight neutral approach to research recognizes the importance of examining a wide variety of plausible mechanisms and explanations in the association between body weight and health. At present, the physiological and psychosocial mechanisms are often explored *independent* of one another; there is a critical gap in scholarship on how these two perspectives can be integrated to further collective knowledge of body weight and health, and improve body weight-related policy – especially when it is apparent that current efforts to address obesity and improve quality of life among adults with obesity are not effective. This dissertation shows that addressing all plausible explanations for how and why body weight is associated with poor health is not feasible in the context of

a single study. However, acknowledging these alternate mechanisms – and accepting that the relationship is not straightforward and unambiguous – is key to more open-minded, unbiased, and innovative research that speaks to the diversity of health and lived experiences represented by body weight.

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## APPENDIX 1: SEMI-STRUCTURED INTERVIEW GUIDE

*Indented questions represent PROMPTS for previous non-indented questions.*

### **Introduction:**

“My name is Iliya Gutin, and I am a PhD student in sociology at UNC-Chapel Hill. I am interested in how obesity is defined and measured as well as how it is diagnosed and treated in clinical settings. The goal of this interview is to gain insight from obesity experts, such as yourself, to help inform how obesity is used and discussed in health research and practice.”

### **Background information:**

“Could you tell me your formal title?”

“What are your medical/research specialization and/or areas of interest?”

“How many years of experience do you have?”

“What is the proportion of time you are engaged in clinical practice?”

“How did you initially develop an interest in obesity, and/or childhood obesity in particular?”

“Did you receive any specialized training in obesity-related medicine and research?”

“Could you briefly describe this training?”

### **Defining and measuring obesity:**

“How do you define obesity?”

“What are some of the characteristics/features that come to mind?”

“Does this definition vary based on context?” For example:

“Among fellow clinicians?”

“With patients and their families?”

“Do you take a child’s age/gender/race into consideration?”

“Outside of a clinical context entirely?”

“Have you observed changes in the definition and understanding of obesity over time?”

“Has the way you define obesity changed throughout your career?”



“What has motivated these changes?”

“Do you consider obesity to be a disease?”

If YES: “What about obesity makes it a disease?”

If NO: “Why not, or what other category does it fall into?”

“Do you believe a majority of health professionals consider obesity to be a disease?”

“Do you believe it is important or necessary to call obesity a disease?”

“How does this differ compared to other medical terms and classifications?”

“Does this influence how you interact with patients?”

“Do you think patients view obesity as a ‘disease’?”

**Diagnosing obesity:**

“How do you measure obesity in a clinical setting?”

“Relatedly, how do you arrive at a diagnosis of obesity, especially among children?”

“How do you think about weight as a measure of obesity, and health in general?”

“Do you believe that children and adults can be “healthy at any size”?”

“Do you ever use this concept in your practice?”

“Given more time and resources, would you use different diagnostic criteria?”

“How do you communicate this diagnosis to your patients and their families?”

“What are the types of reactions you usually encounter?”

“Do you ever question the decision to make a diagnosis of obesity?”

“Have you ever felt it was ‘wrong’ to diagnose obesity in a patient?”

“Have you ever ignored a patient’s BMI/weight status?”

**Treating obesity:**

“How do you discuss ‘treatment’ options for weight and/or obesity?”

“Is this usually general advice, or does this vary from patient to patient?”

“What counts as a successful outcome?”

“What are the kinds of outcomes or changes in health are you looking for?”

“How important is weight/BMI?”

“How do you perceive your role as a doctor in helping to reduce obesity?”

“What are the major obstacles to your success?”

“What are the major obstacles to your patients’ success?”

“Where does obesity usually fit in among patients’ health concerns?”

**Other (*time permitting, or incorporated throughout*):**

“Do you think most medical practitioners need obesity-specific training?”

“How do you view the role of personal responsibility in weight and health?”

“What do you think is the single most important factor in obesity prevention efforts?”

“How would compare the BMI to other measures of health you use?”

**APPENDIX 2: CHAPTER 3 SUPPLEMENTAL TABLES**

**Table A.1** Conditional Item Probabilities for 4-Class Solution

	"Ideal" (29%)	"Mixed" Health w/ Obesity (25%)	"Mixed" Health w/o Obesity (34%)	"High Risk" (12%)
<b>Body Size and History</b>				
<i>Obesity</i>	0.60%	77.2%	3.90%	83.5%
<i>Ever obese</i>	4.20%	100%	11.2%	98.6%
<i>Obesity 10 years ago</i>	0.00%	42.4%	0.00%	61.2%
<i>Obesity at age 25</i>	0.30%	19.10%	0.00%	22.4%
<i>Waist-to-hip</i>	30.3%	85.9%	93.2%	97.6%
<i>Waist-to-height</i>	35.2%	99.4%	92.1%	99.7%
<b>Cardiovascular</b>				
<i>Pulse</i>	0.70%	1.10%	1.20%	3.30%
<i>Hypertension (M)</i>	30.1%	63.1%	68.2%	79.9%
<i>Hypertension (Dx)</i>	7.30%	33.6%	31.5%	66.9%
<b>Dyslipidemia</b>				
<i>High Chol. (M)</i>	33.0%	54.0%	69.7%	55.1%
<i>High Chol. (Dx)</i>	11.1%	27.9%	38.8%	50.8%
<i>High Trigly.</i>	5.70%	40.7%	49.8%	65.9%
<i>High Apob</i>	9.40%	41.1%	61.1%	42.6%
<i>Low HDL</i>	6.30%	27.1%	26.3%	41.0%
<b>Hyperglycemia</b>				
<i>High HbA1c</i>	4.10%	10.6%	20.4%	79.0%
<i>High Glucose</i>	19.5%	36.0%	50.8%	85.6%
<i>High Insulin</i>	0.50%	5.40%	30.9%	38.9%
<i>Diabetes (Dx)</i>	0.70%	0.40%	5.20%	45.0%
<b>Other</b>				
<i>High CRP</i>	12.8%	45.7%	30.9%	65.0%
<i>High Alb.-to-Creat.</i>	0.20%	0.40%	1.00%	6.20%

*Notes:*

National Health and Nutrition Examination Survey, 1988-2014, Ages 30-74.

Estimates account for NCHS-provided survey weights.

**Table A.2** Adjusted All-cause Mortality Risk Across Latent Classes

	<i>Odds Ratio</i>	<i>95% CI</i>	
<b>5-Class Solution (ref. "Ideal")</b>			
"Fat but Fit"	1.25	1.05	1.49
Mixed Health w/ Obesity	1.37	1.19	1.58
Mixed Health w/o Obesity	1.23	1.09	1.39
High Risk	2.04	1.77	2.35
<b>Education (ref. BA or higher)</b>			
<i>Less than HS</i>	1.46	1.23	1.73
<i>HS or equal</i>	1.36	1.18	1.56
<i>Some college</i>	1.38	1.16	1.64
<b>Survey Cycle (ref. 1988-1994)</b>			
<i>1999-2000</i>	0.56	0.49	0.63
<i>2001-2002</i>	0.40	0.36	0.45
<i>2003-2004</i>	0.31	0.27	0.36
<i>2005-2006</i>	0.30	0.25	0.36
<i>2007-2008</i>	0.23	0.20	0.28
<i>2009-2010</i>	0.23	0.18	0.29
<i>2011-2012</i>	0.26	0.19	0.34
<i>2013-2014</i>	0.62	0.54	0.70
<b>Age</b>	1.05	1.02	1.08
<b>Age-squared</b>	1.00	1.00	1.00
<b>Female</b>	0.79	0.73	0.84
<b>Race/ethnicity (ref. NH White)</b>			
<i>NH Black</i>	1.13	1.03	1.24
<i>MX-American</i>	1.12	1.00	1.25
<i>Other</i>	0.87	0.74	1.03
<b>Foreign-born</b>	0.89	0.78	1.02
<b>Income-to-needs ratio (ref. 0-0.99)</b>			
<i>1.00-1.99</i>	1.85	1.62	2.10
<i>2.00-3.99</i>	1.47	1.29	1.68
<i>4.00+</i>	1.11	0.98	1.25
<b>Health insurance</b>	0.88	0.77	1.00
<b>Smoking status (ref. Never)</b>			
<i>Former</i>	1.28	1.16	1.41
<i>Current</i>	2.09	1.93	2.26
<b>Sample size</b>		40,095	
<b>Number of deaths</b>		7,106	

Notes:

National Health and Nutrition Examination Survey, 1988-2014,  
Ages 30-75.

Deaths restricted to ages 30-85.

Estimates account for NCHS-provided survey weights.

Estimates based on multiple imputation to account for missing data.

**Table A.3** Fully-adjusted Cause-specific Mortality Risk Across Latent Classes

	Underlying Diabetes			Underlying Hypertension			Heart Disease			Heart Disease, Diabetes, or Cancer		
	<i>Odds Ratio</i>	<i>95% CI</i>		<i>Odds Ratio</i>	<i>95% CI</i>		<i>Odds Ratio</i>	<i>95% CI</i>		<i>Odds Ratio</i>	<i>95% CI</i>	
<b>5-Class Solution</b> ( <i>ref. "Ideal"</i> )												
"Fat but Fit"	1.28	0.53	3.10	1.48	0.85	2.59	1.03	0.66	1.60	1.09	0.88	1.34
Mixed Health w/ Obesity	4.45	2.18	9.08	2.84	1.57	5.15	2.02	1.42	2.90	1.37	1.15	1.62
Mixed Health w/o Obesity	2.83	1.42	5.62	1.83	1.09	3.09	1.60	1.13	2.28	1.21	1.05	1.40
High Risk	20.5	10.2	41.2	5.41	3.05	9.60	3.45	2.23	5.32	2.06	1.68	2.53
<b>Sample size</b>	40,095			40,095			40,095			40,095		
<b>Number of deaths</b>	1,017			1,014			1,395			3,502		

*Notes:*

National Health and Nutrition Examination Survey, 1988-2014, Ages 30-75.

Deaths restricted to ages 30-85.

Estimates account for NCHS-provided survey weights.

Estimates based on multiple imputation to account for missing data.

Adjusted for educational attainment, age, age-squared, survey year, gender, race/ethnicity, nativity, income-to-needs ratio, health insurance, and smoking status.



2007-2008	6.60%	4.37%	8.83%
2009-2010	8.58%	7.02%	10.1%
2011-2012	7.56%	5.20%	9.91%
2013-2014	5.17%	3.98%	6.35%
<b>Age</b>	-0.29%	-0.33%	-0.24%
<b>Female</b>	3.24%	2.29%	4.18%
<b>Race/ethnicity (ref. NH White)</b>			
<i>NH Black</i>	4.74%	3.50%	5.98%
<i>MX-American</i>	2.93%	1.21%	4.65%
<i>Other</i>	2.04%	-0.24%	4.33%
<b>Foreign-born</b>	-6.97%	-8.91%	-5.03%
<b>Income-to-needs ratio (ref. 0-0.99)</b>			
1.00-1.99	2.77%	1.04%	4.51%
2.00-3.99	2.31%	0.51%	4.10%
4.00+	1.50%	0.21%	2.80%
<b>Health insurance</b>	-0.74%	-2.34%	0.87%
<b>Smoking status (ref. Never)</b>			
<i>Former</i>	0.07%	-1.20%	1.35%
<i>Current</i>	-3.52%	-4.80%	-2.24%

Mixed Health w/ Obesity

95% C.I.

<b>Education (ref. BA or higher)</b>			
<i>Less than HS</i>	4.29%	2.11%	6.48%
<i>HS or equal</i>	4.57%	2.79%	6.35%
<i>Some college</i>	4.16%	2.32%	5.99%
<b>Survey Cycle (ref. 1988-1994)</b>			
1999-2000	1.73%	-0.21%	3.68%
2001-2002	1.60%	-0.61%	3.81%
2003-2004	5.62%	4.10%	7.15%
2005-2006	3.53%	1.98%	5.08%
2007-2008	2.81%	0.95%	4.68%
2009-2010	1.09%	-0.76%	2.94%
2011-2012	3.62%	2.13%	5.12%
2013-2014	2.14%	0.59%	3.68%
<b>Age</b>	0.14%	0.11%	0.17%
<b>Female</b>	-2.79%	-3.86%	-1.72%
<b>Race/ethnicity (ref. NH White)</b>			
<i>NH Black</i>	-2.64%	-3.63%	-1.65%
<i>MX-American</i>	1.85%	0.27%	3.44%
<i>Other</i>	-3.26%	-5.31%	-1.20%
<b>Foreign-born</b>	-3.45%	-5.41%	-1.50%
<b>Income-to-needs ratio (ref. 0-0.99)</b>			
1.00-1.99	2.10%	0.20%	4.00%



2.00-3.99	2.11%	0.23%	4.00%
4.00+	1.41%	-0.03%	2.85%
<b>Health insurance</b>	-0.90%	-2.64%	0.84%
<b>Smoking status (ref. Never)</b>			
Former	0.69%	-0.71%	2.10%
Current	-2.39%	-3.71%	-1.07%

Mixed Health w/o Obesity

95% C.I.

<b>Education (ref. BA or higher)</b>			
Less than HS	1.77%	-0.99%	4.54%
HS or equal	0.87%	-1.63%	3.37%
Some college	-0.29%	-2.66%	2.09%
<b>Survey Cycle (ref. 1988-1994)</b>			
1999-2000	-6.46%	-8.81%	-4.11%
2001-2002	-6.00%	-9.47%	-2.54%
2003-2004	-5.95%	-8.48%	-3.41%
2005-2006	-5.69%	-8.18%	-3.20%
2007-2008	-7.54%	-9.78%	-5.31%
2009-2010	-7.79%	-10.7%	-4.93%
2011-2012	-10.7%	-13.1%	-8.29%
2013-2014	-5.86%	-7.73%	-3.99%
<b>Age</b>	0.93%	0.86%	1.00%
<b>Female</b>	-12.6%	-13.9%	-11.3%
<b>Race/ethnicity (ref. NH White)</b>			
NH Black	-6.58%	-8.11%	-5.05%
MX-American	-0.45%	-2.40%	1.51%
Other	2.89%	-0.21%	5.98%
<b>Foreign-born</b>	8.55%	6.37%	10.74%
<b>Income-to-needs ratio (ref. 0-0.99)</b>			
1.00-1.99	-3.72%	-6.58%	-0.86%
2.00-3.99	-4.97%	-7.51%	-2.44%
4.00+	-2.09%	-3.86%	-0.31%
<b>Health insurance</b>	0.12%	-2.35%	2.58%
<b>Smoking status (ref. Never)</b>			
Former	-1.80%	-3.30%	-0.30%
Current	4.39%	2.49%	6.29%

High Risk

95% C.I.

<b>Education (ref. BA or higher)</b>			
Less than HS	6.71%	5.27%	8.15%
HS or equal	4.64%	3.34%	5.93%
Some college	3.04%	2.11%	3.96%

<b>Survey Cycle (ref. 1988-1994)</b>			
1999-2000	1.58%	0.13%	3.03%
2001-2002	2.38%	0.88%	3.87%
2003-2004	3.85%	2.35%	5.35%
2005-2006	5.68%	3.79%	7.57%
2007-2008	5.81%	4.29%	7.32%
2009-2010	6.65%	4.56%	8.74%
2011-2012	7.76%	6.53%	9.00%
2013-2014	1.00%	0.44%	2.43%
<b>Age</b>	0.35%	0.32%	0.38%
<b>Female</b>	-0.97%	1.67%	-0.27%
<b>Race/ethnicity (ref. NH White)</b>			
NH Black	6.38%	5.26%	7.50%
MX-American	5.21%	3.73%	6.70%
Other	2.48%	0.65%	4.32%
<b>Foreign-born</b>	-3.95%	5.39%	-2.52%
<b>Income-to-needs ratio (ref. 0-0.99)</b>			
1.00-1.99	4.75%	3.32%	6.18%
2.00-3.99	3.76%	2.63%	4.89%
4.00+	1.48%	0.32%	2.63%
<b>Health insurance</b>	2.38%	1.04%	3.73%
<b>Smoking status (ref. Never)</b>			
Former	1.37%	0.40%	2.33%
Current	-2.31%	-3.33%	-1.28%

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*Notes:*

National Health and Nutrition Examination Survey,  
1988-2014, Ages 30-74.  
Estimates account for NCHS-provided survey weights.  
Estimates based on multiple imputation to account for missing data.

**Table A.5** Average Marginal Effects for Educational Attainment Across Latent Classes

		"Ideal"				
		Unadjusted		Adjusted		
		95% C.I.		95% C.I.		
<b>Education (ref. BA or higher)</b>						
<i>Less than HS</i>	-20.4%	-22.9%	-17.9%	-15.9%	-18.5%	-13.4%
<i>HS or equal</i>	-12.7%	-14.8%	-10.6%	-12.5%	-14.5%	-10.4%
<i>Some college</i>	-11.6%	-14.6%	-8.67%	-11.1%	-13.8%	-8.37%
<b>Mixed Health w/ Obesity</b>						
		Unadjusted		Adjusted		
		95% C.I.		95% C.I.		
<b>Education (ref. BA or higher)</b>						
<i>Less than HS</i>	3.90%	1.66%	6.15%	5.85%	3.02%	8.68%
<i>HS or equal</i>	5.33%	3.24%	7.42%	5.69%	3.44%	7.95%
<i>Some college</i>	8.68%	6.45%	10.9%	6.95%	4.72%	9.17%
<b>Mixed Health w/o Obesity</b>						
		Unadjusted		Adjusted		
		95% C.I.		95% C.I.		
<b>Education (ref. BA or higher)</b>						
<i>Less than HS</i>	5.13%	2.59%	7.67%	2.28%	-0.39%	4.96%
<i>HS or equal</i>	1.44%	-0.90%	3.78%	1.31%	-1.07%	3.69%
<i>Some college</i>	-1.98%	-4.48%	0.52%	-0.06%	-2.42%	2.30%
<b>High Risk</b>						
		Unadjusted		Adjusted		
		95% C.I.		95% C.I.		
<b>Education (ref. BA or higher)</b>						
<i>Less than HS</i>	11.4%	9.93%	12.8%	7.82%	6.05%	9.59%
<i>HS or equal</i>	5.94%	4.47%	7.40%	5.46%	3.86%	7.05%
<i>Some college</i>	4.95%	3.86%	6.03%	4.19%	3.00%	5.39%

*Notes:*

National Health and Nutrition Examination Survey, 1988-2014, Ages 30-75.

Estimates account for NCHS-provided survey weights.

Estimates based on multiple imputation to account for missing data.

Adjusted for educational attainment, age, survey year, gender, race/ethnicity, nativity, income-to-needs ratio, health insurance, and smoking status.

**Table A.6** Average Marginal Effects for All Covariates

		"Ideal"	
		95% C.I.	
<b>Education (ref. BA or higher)</b>			
<i>Less than HS</i>	15.9%	-18.5%	-13.4%
<i>HS or equal</i>	12.5%	-14.5%	-10.4%
<i>Some college</i>	11.1%	-13.8%	-8.37%
<b>Survey Cycle (ref. 1988-1994)</b>			
<i>1999-2000</i>	2.53%	-5.03%	-0.03%
<i>2001-2002</i>	6.03%	-8.62%	-3.45%
<i>2003-2004</i>	8.26%	-10.9%	-5.59%
<i>2005-2006</i>	7.60%	-9.95%	-5.24%
<i>2007-2008</i>	7.73%	-9.92%	-5.55%
<i>2009-2010</i>	8.24%	-10.6%	-5.86%
<i>2011-2012</i>	-7.50%	-10.5%	-4.52%
<i>2013-2014</i>	2.51%	-5.68%	0.66%
<b>Age</b>	1.17%	-1.26%	-1.09%
<b>Female</b>	13.8%	12.4%	15.2%
<b>Race/ethnicity (ref. NH White)</b>			
<i>NH Black</i>	1.58%	-3.13%	-0.03%
<i>MX-American</i>	8.93%	-10.8%	-7.01%
<i>Other</i>	3.94%	-6.43%	-1.45%
<b>Foreign-born</b>	5.76%	3.83%	7.69%
<b>Income-to-needs ratio (ref. 0-0.99)</b>			
<i>1.00-1.99</i>	-6.11%	-8.79%	-3.43%
<i>2.00-3.99</i>	2.99%	-5.29%	-0.68%
<i>4.00+</i>	2.35%	-4.01%	-0.69%
<b>Health insurance</b>	0.95%	-3.52%	1.62%
<b>Smoking status (ref. Never)</b>			
<i>Former</i>	0.11%	-1.94%	1.72%
<i>Current</i>	3.78%	1.86%	5.70%
		Mixed Health w/ Obesity	
		95% C.I.	
<b>Education (ref. BA or higher)</b>			
<i>Less than HS</i>	5.85%	3.02%	8.68%
<i>HS or equal</i>	5.69%	3.44%	7.95%
<i>Some college</i>	6.95%	4.72%	9.17%
<b>Survey Cycle (ref. 1988-1994)</b>			
<i>1999-2000</i>	8.32%	5.66%	11.0%
<i>2001-2002</i>	11.1%	8.19%	14.0%
<i>2003-2004</i>	10.8%	7.70%	13.9%
<i>2005-2006</i>	7.37%	5.30%	9.44%

2007-2008	9.09%	6.83%	11.3%
2009-2010	9.86%	7.48%	12.2%
2011-2012	10.5%	7.74%	13.3%
2013-2014	8.81%	6.60%	11.0%
<b>Age</b>	-0.17%	-0.22%	-0.12%
<b>Female</b>	-0.23%	-1.43%	0.97%
<b>Race/ethnicity (ref. NH White)</b>			
<i>NH Black</i>	2.00%	0.46%	3.53%
<i>MX-American</i>	3.05%	0.61%	5.49%
<i>Other</i>	-1.95%	-4.77%	0.87%
<b>Foreign-born</b>	-9.99%	-12.5%	-7.47%
<b>Income-to-needs ratio (ref. 0-0.99)</b>			
<i>1.00-1.99</i>	4.61%	2.44%	6.77%
<i>2.00-3.99</i>	4.21%	2.10%	6.32%
<i>4.00+</i>	2.62%	0.93%	4.31%
<b>Health insurance</b>	-1.97%	-4.23%	0.29%
<b>Smoking status (ref. Never)</b>			
<i>Former</i>	0.14%	-1.58%	1.87%
<i>Current</i>	-5.41%	-6.92%	-3.90%

Mixed Health w/o Obesity

95% C.I.

<b>Education (ref. BA or higher)</b>			
<i>Less than HS</i>	2.28%	-0.39%	4.96%
<i>HS or equal</i>	1.31%	-1.07%	3.69%
<i>Some college</i>	-0.06%	-2.42%	2.30%
<b>Survey Cycle (ref. 1988-1994)</b>			
<i>1999-2000</i>	-6.25%	-8.74%	-3.75%
<i>2001-2002</i>	-6.16%	-9.49%	-2.82%
<i>2003-2004</i>	-6.02%	-8.47%	-3.57%
<i>2005-2006</i>	-5.56%	-7.85%	-3.27%
<i>2007-2008</i>	-7.13%	-9.34%	-4.92%
<i>2009-2010</i>	-8.09%	-10.69%	-5.50%
<i>2011-2012</i>	-10.6%	-12.7%	-8.39%
<i>2013-2014</i>	-6.22%	-8.14%	-4.30%
<b>Age</b>	0.92%	0.85%	0.99%
<b>Female</b>	-12.0%	-13.4%	-10.7%
<b>Race/ethnicity (ref. NH White)</b>			
<i>NH Black</i>	-6.80%	-8.28%	-5.31%
<i>MX-American</i>	-0.83%	-2.92%	1.25%
<i>Other</i>	2.33%	-0.88%	5.53%
<b>Foreign-born</b>	8.91%	6.66%	11.2%
<b>Income-to-needs ratio (ref. 0-0.99)</b>			
<i>1.00-1.99</i>	-3.79%	-6.55%	-1.04%

2.00-3.99	-5.20%	-7.69%	-2.70%
4.00+	-2.22%	-4.01%	-0.43%
<b>Health insurance</b>	0.86%	-1.51%	3.23%
<b>Smoking status (ref. Never)</b>			
Former	-1.88%	-3.41%	-0.35%
Current	4.43%	2.48%	6.37%
		High Risk	
		95% C.I.	
<b>Education (ref. BA or higher)</b>			
Less than HS	7.82%	6.05%	9.59%
HS or equal	5.46%	3.86%	7.05%
Some college	4.19%	3.00%	5.39%
<b>Survey Cycle (ref. 1988-1994)</b>			
1999-2000	0.46%	-1.40%	2.32%
2001-2002	1.11%	-0.61%	2.83%
2003-2004	3.45%	1.59%	5.32%
2005-2006	5.78%	3.71%	7.86%
2007-2008	5.78%	4.18%	7.37%
2009-2010	6.47%	4.03%	8.90%
2011-2012	7.53%	5.94%	9.12%
2013-2014	-0.08%	-1.77%	1.62%
<b>Age</b>	0.42%	0.38%	0.46%
<b>Female</b>	-1.55%	-2.37%	-0.73%
<b>Race/ethnicity (ref. NH White)</b>			
NH Black	6.38%	5.12%	7.63%
MX-American	6.72%	5.02%	8.41%
Other	3.56%	1.35%	5.77%
<b>Foreign-born</b>	-4.68%	-6.27%	-3.08%
<b>Income-to-needs ratio (ref. 0-0.99)</b>			
1.00-1.99	5.30%	3.60%	6.99%
2.00-3.99	3.97%	2.56%	5.39%
4.00+	1.96%	0.60%	3.31%
<b>Health insurance</b>	2.06%	0.59%	3.52%
<b>Smoking status (ref. Never)</b>			
Former	1.84%	0.65%	3.04%
Current	-2.79%	-3.80%	-1.79%

*Notes:*

National Health and Nutrition Examination Survey,  
1988-2014, Ages 30-74.  
Estimates account for NCHS-provided survey weights.  
Estimates based on multiple imputation to account for  
missing data.

**Table A.7** Percent Contribution of Latent Classes to Educational Disparities in All-Cause Mortality

	Percent Mediated	
	<i>Unadjusted</i>	<i>Adjusted</i>
<b>Education (ref. BA or higher)</b>		
<i>Less than HS (ref. "Ideal")</i>	17.6	15.3
Mixed Health w/ Obesity	3.13	3.94
Mixed Health w/o Obesity	3.99	1.74
"High Risk"	10.5	9.63
<i>HS or equal (ref. "Ideal")</i>	16.0	14.0
Mixed Health w/ Obesity	4.95	4.87
Mixed Health w/o Obesity	1.79	1.43
"High Risk"	9.28	7.66
<i>Some college (ref. "Ideal")</i>	21.1	11.3
Mixed Health w/ Obesity	11.4	6.02
Mixed Health w/o Obesity	-0.84	0.93
"High Risk"	10.5	4.33

*Notes:*

National Health and Nutrition Examination Survey, 1988-2014, Ages 30-75.

Deaths restricted to ages 30-85.

Estimates account for NCHS-provided survey weights.

Adjusted for age, age-squared, survey year, gender, race/ethnicity, nativity, income-to-needs ratio, health insurance, and smoking status.

**Table A.8** Percent Contribution of Latent Classes to Educational Disparities in Cause-specific Mortality

	Percent Mediated			
	<i>Underlying Diabetes</i>	<i>Underlying Hypertension</i>	<i>Heart Disease</i>	<i>Heart Disease, Diabetes, or Cancer</i>
<b>Education (ref. BA or higher)</b>				
<i>Less than HS (ref. "Ideal")</i>	37.2	28.2	25.6	17.5
Mixed Health w/ Obesity	8.65	8.49	6.91	3.87
Mixed Health w/o Obesity	5.81	3.93	3.88	2.25
"High Risk"	22.7	15.8	14.8	11.4
<i>HS or equal (ref. "Ideal")</i>	32.8	22.1	27.3	14.8
Mixed Health w/ Obesity	10.46	8.84	9.91	4.52
Mixed Health w/o Obesity	4.67	2.73	3.70	1.75
"High Risk"	17.66	10.6	13.7	8.57
<i>Some college (ref. "Ideal")</i>	44.4	46.5	18.1	11.2
Mixed Health w/ Obesity	22.1	27.2	9.92	5.41
Mixed Health w/o Obesity	5.18	4.40	1.94	1.10
"High Risk"	17.1	14.9	6.27	4.70

*Notes:*

National Health and Nutrition Examination Survey, 1988-2014, Ages 30-75.

Deaths restricted to ages 30-85.

Estimates account for NCHS-provided survey weights.

Adjusted for age, age-squared, survey year, gender, race/ethnicity, nativity, income-to-needs ratio, health insurance, and smoking status.



**APPENDIX 3: CHAPTER 4 SUPPLEMENTAL TABLES**

**Table A.9** Descriptive Statistics for Perceived Weight, Body Mass Index, and Sociodemographic Covariates and Health Outcomes

	<i>Overall</i>		<i>Female</i>		<i>Male</i>	
	Mean/Prop.	Std. Dev.	Mean/Prop.	Std. Dev.	Mean/Prop.	Std. Dev.
<i>Perceived Weight</i>						
Wave I	3.190	0.797	3.334	0.782	3.002	0.776
Wave II	3.196	0.760	3.330	0.750	3.019	0.737
Wave III	3.354	0.800	3.495	0.784	3.162	0.782
Wave IV	3.649	0.831	3.788	0.818	3.461	0.811
<i>Body Mass Index (BMI)</i>						
Wave I	22.536	4.471	22.317	4.419	22.816	4.521
Wave II	23.058	5.074	22.981	5.200	23.157	4.904
Wave III	26.678	6.411	26.677	6.959	26.678	5.583
Wave IV	29.175	7.607	29.224	8.208	29.109	6.705
<i>Sociodemographic</i>						
Age at Wave I	15.580	1.738	15.511	1.738	15.671	1.733
Female	0.567	0.496	-	-	-	-
Race/Ethnicity						
NH-White	0.600	0.490	0.584	0.493	0.622	0.485
NH-Black	0.202	0.401	0.225	0.417	0.173	0.378
Hispanic	0.123	0.329	0.124	0.330	0.122	0.327
NH-Other	0.074	0.263	0.068	0.251	0.083	0.276
College-educated Parent	0.347	0.476	0.340	0.473	0.356	0.479
Wave III In-College +	0.517	0.500	0.537	0.499	0.490	0.500
Wave IV B.A. Degree +	0.358	0.480	0.388	0.487	0.319	0.466
Wave V B.A. Degree +	0.407	0.491	0.445	0.497	0.357	0.480
<i>Wave V Outcomes</i>						

BMI	30.703	7.848	30.873	8.444	30.440	6.814
Syst. BP (1)	124.345	16.302	120.417	15.631	130.215	15.501
Syst. BP (2)	122.923	15.622	119.140	14.880	128.568	14.985
Syst. BP (3)	122.200	15.287	118.526	14.636	127.685	14.579
Diast. BP (1)	80.185	11.467	78.021	11.149	83.419	11.170
Diast. BP (2)	79.662	11.149	77.468	10.805	82.935	10.850
Diast. BP (3)	79.261	11.024	77.042	10.692	82.573	10.676
Measured						
Hypertension/Rx	0.308	0.462	0.249	0.432	0.396	0.489
C-reactive Protein	4.026	6.133	4.789	6.879	2.888	4.580
Felt Depressed Freq.	1.391	0.680	1.425	0.704	1.346	0.647
Felt Blues Freq.	1.358	0.691	1.380	0.704	1.330	0.673
Felt Sad Freq.	1.575	0.669	1.634	0.686	1.497	0.637
Depression Dx	0.246	0.430	0.301	0.459	0.173	0.378
Anxiety Dx	0.220	0.415	0.274	0.446	0.149	0.356
Sleep Trouble Freq.	1.722	1.329	1.853	1.330	1.551	1.309

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N(Overall)=12,300; N(Female)=6,974, N(Male)=5,326.

Results based on casewise maximum likelihood (also called FIML) estimation to account for missing data, with robust (Huber-White) standard errors.

**Table A.10** Comparison of Longitudinal Models for Subjective Weight Across Different Specifications

Very Under/Slightly Under/About Right vs. Slightly Over vs. Very Over (Continuous)							
Model	$\chi^2$	DF	BIC	CFI	TLI	1-RMSEA	Notes
Autoregressive	1.045	1	-8.372	1.000	1.000	0.998	None
Fixed Effects	416.035	3	387.783	0.940	0.879	0.843	Negative variances
Linear Growth	217.934	3	189.682	0.971	0.943	0.892	Negative variances
Freed Loading Growth	22.278	1	12.861	0.995	0.969	0.920	Negative variances
ALT-Fixed Effects	26.044	1	16.627	0.998	0.986	0.946	Regressions constrained to be equal
Under vs. About Right vs. Over (Continuous)							
Model	$\chi^2$	DF	BIC	CFI	TLI	1-RMSEA	Notes
Autoregressive	0.403	1	-9.014	1.000	1.000	1.000	None
Fixed Effects	713.982	3	685.730	0.888	0.776	0.805	Negative variances
Linear Growth	100.892	3	72.640	0.987	0.973	0.933	Negative variances
Freed Loading Growth	27.570	1	18.153	0.994	0.963	0.921	Negative variances
ALT-Fixed Effects	28.314	1	18.897	0.997	0.984	0.945	Regressions constrained to be equal
Under/About Right vs. Over							
Model	$\chi^2$	DF	BIC	CFI	TLI	1-RMSEA	Notes
Autoregressive	0.730	1	-8.687	1.000	1.000	1.000	None
Fixed Effects	619.866	3	591.614	0.901	0.802	0.820	None
Linear Growth	149.622	3	121.370	0.979	0.958	0.917	Negative variances

Freed Loading Growth	22.913	1	13.496	0.995	0.968	0.927	<i>Negative variances</i>
ALT-Fixed Effects	51.185	1	41.768	0.996	0.976	0.938	<i>Regressions constrained to be equal</i>
Very Under vs. Slightly Under vs. About Right vs. Slightly Over vs. Very Over (Ordinal)							
<b>Model</b>	<b><math>\chi^2</math></b>	<b>DF</b>	<b>BIC</b>	<b>CFI</b>	<b>TLI</b>	<b>1-RMSEA</b>	<b>Notes</b>
Autoregressive	2.907	1	-6.510	1.000	1.000	0.988	<i>None</i>
Fixed Effects	951.528	3	923.276	0.985	0.969	0.840	<i>Negative variances</i>
Linear Growth	34.129	3	5.877	0.999	0.999	0.971	<i>Negative variances</i>
Freed Loading Growth	2.323	1	-7.094	1.000	1.000	0.990	<i>None</i>
ALT-Fixed Effects	0.093	1	-9.324	1.000	1.000	1.000	<i>Regressions constrained to be equal</i>
Very Under/Slightly Under/About Right vs. Slightly Over vs. Very Over (Ordinal)							
<b>Model</b>	<b><math>\chi^2</math></b>	<b>DF</b>	<b>BIC</b>	<b>CFI</b>	<b>TLI</b>	<b>1-RMSEA</b>	<b>Notes</b>
Autoregressive	0.099	1	-9.318	1.000	1.000	1.000	<i>None</i>
Fixed Effects	481.692	3	453.440	0.990	0.981	0.886	<i>Negative variances</i>
Linear Growth	178.083	3	149.831	0.996	0.993	0.931	<i>Negative variances</i>
Freed Loading Growth	24.084	1	14.667	1.000	0.997	0.957	<i>Negative variances</i>
ALT-Fixed Effects	13.442	1	4.025	1.000	0.998	0.968	<i>Regressions constrained to be equal</i>
Under vs. About Right vs. Over (Ordinal)							
<b>Model</b>	<b><math>\chi^2</math></b>	<b>DF</b>	<b>BIC</b>	<b>CFI</b>	<b>TLI</b>	<b>1-RMSEA</b>	<b>Notes</b>
Autoregressive	0.149	1	-9.268	1.000	1.000	1.000	<i>None</i>
Fixed Effects	639.834	3	611.582	0.985	0.970	0.869	<i>Negative variances</i>

Linear Growth	161.096	3	132.844	0.996	0.993	0.935	<i>Negative variances</i>
Freed Loading Growth	43.544	1	34.127	0.999	0.994	0.941	<i>Negative variances</i>
ALT-Fixed Effects	23.893	1	14.476	0.999	0.997	0.957	<i>Regressions constrained to be equal</i>

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N=12,300.

Results based on casewise maximum likelihood (also called FIML) estimation to account for missing data, with robust (Huber-White) standard errors for continuous models and diagonal weighted least squares standard errors for ordinal models.

Variance on first and last two observed measures of perceived weight constrained to be equal, respectively.

First two thresholds for categorical specifications fixed at 1 and 2, respectively.

**Table A.11** Unweighted Coefficient Estimates for Best-fitting Longitudinal Model of Subjective Weight (SUBJ.) and Objective Weight (OBJ.)

<b>Parameters</b>	<b>Estimate</b>	<b>Std. Err.</b>	<b>P-value</b>
<i>Regressions</i>			
Wave IV OBJ. ←			
Wave III OBJ.	1.117	0.029	0.000
Wave III SUBJ.	-0.671	0.236	0.004
Wave III OBJ. ←			
Wave II OBJ.	1.064	0.021	0.000
Wave II SUBJ.	0.412	0.136	0.002
Wave II OBJ. ←			
Wave I OBJ.	1.073	0.024	0.000
Wave I SUBJ.	0.093	0.122	0.448
Wave IV SUBJ. ←			
Wave III SUBJ.	0.898	0.042	0.000
Wave III OBJ.	-0.089	0.008	0.000
Wave IV OBJ.	0.084	0.006	0.000
Wave III SUBJ. ←			
Wave II SUBJ.	0.543	0.024	0.000
Wave II OBJ.	-0.068	0.005	0.000
Wave III OBJ.	0.096	0.003	0.000
Wave II SUBJ. ←			
Wave I SUBJ.	0.798	0.032	0.000
Wave I OBJ.	-0.072	0.014	0.000
Wave II OBJ.	0.082	0.012	0.000
Wave I SUBJ. ←			
Wave I OBJ.	0.114	0.002	0.000
<i>Intercepts</i>			
Wave I OBJ.	22.614	0.041	0.000
Wave II OBJ.	-1.337	0.241	0.000
Wave III OBJ.	0.764	0.267	0.000
Wave IV OBJ.	1.450	0.303	0.000
Wave I SUBJ.	0.616	0.050	0.000
Wave II SUBJ.	0.391	0.044	0.000
Wave III SUBJ.	0.606	0.039	0.000
Wave IV SUBJ.	0.572	0.055	0.000
<i>Variances</i>			
Wave I BMI	1.852	0.173	0.000
Wave II BMI	1.852	0.173	0.000
Wave III BMI	3.641	0.367	0.000
Wave IV BMI	3.641	0.367	0.000
Wave I PW	0.160	0.008	0.000
Wave II PW	0.160	0.008	0.000

Wave III PW	0.209	0.010	0.000
Wave IV PW	0.209	0.010	0.000
Wave I OBJ.	18.623	0.458	0.000
Wave II OBJ.	2.270	0.312	0.000
Wave III OBJ.	8.965	0.349	0.000
Wave IV OBJ.	11.216	0.758	0.000
Wave I SUBJ.	0.233	0.011	0.000
Wave II SUBJ.	0.038	0.011	0.001
Wave III SUBJ.	0.079	0.007	0.000
Wave IV SUBJ.	0.065	0.014	0.000

*R-Square*

Wave I BMI	0.910
Wave II BMI	0.929
Wave III BMI	0.913
Wave IV BMI	0.937
Wave I PW	0.747
Wave II PW	0.723
Wave III PW	0.673
Wave IV PW	0.696
Wave II OBJ.	0.906
Wave III OBJ.	0.767
Wave IV OBJ.	0.793
Wave I SUBJ.	0.508
Wave II SUBJ.	0.909
Wave III SUBJ.	0.816
Wave IV SUBJ.	0.863

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N=12,300.

Results based on casewise maximum likelihood (also called FIML) estimation to account for missing data, with robust (Huber-White) standard errors.

**Table A.12** Coefficient Estimates for Best-fitting Longitudinal Model of Subjective Weight (SUBJ.) and Objective Weight (OBJ.); Female vs. Male

Parameters	Female				Male			
	Estimate	Std. Err.	P-value	Stdz. Est.	Estimate	Std. Err.	P-value	Stdz. Est.
<i>Regressions</i>								
Wave IV OBJ. ←								
Wave III OBJ.	0.985	0.072	0.000	0.834	1.099	0.133	0.000	0.918
Wave III SUBJ.	0.075	0.081	0.359	0.056	-0.063	0.122	0.604	-0.057
Wave III OBJ. ←								
Wave II OBJ.	0.868	0.054	0.000	0.648	0.769	0.065	0.000	0.641
Wave II SUBJ.	0.303	0.041	0.000	0.277	0.219	0.040	0.000	0.248
Wave II OBJ. ←								
Wave I OBJ.	1.080	0.081	0.000	0.894	0.967	0.088	0.000	0.862
Wave I SUBJ.	0.030	0.040	0.454	0.037	0.070	0.044	0.115	0.097
Wave IV SUBJ. ←								
Wave III SUBJ.	0.926	0.137	0.000	0.889	1.047	0.181	0.000	1.014
Wave III OBJ.	-0.753	0.147	0.000	-0.815	-1.054	0.293	0.000	-0.937
Wave IV OBJ.	0.671	0.078	0.000	0.857	0.854	0.157	0.000	0.909
Wave III SUBJ. ←								
Wave II SUBJ.	0.368	0.044	0.000	0.379	0.317	0.051	0.000	0.329
Wave II OBJ.	-0.460	0.070	0.000	-0.386	-0.303	0.084	0.000	-0.232
Wave III OBJ.	0.819	0.058	0.000	0.922	0.936	0.075	0.000	0.860
Wave II SUBJ. ←								
Wave I SUBJ.	0.863	0.086	0.000	0.885	0.791	0.075	0.000	0.813
Wave I OBJ.	-0.381	0.312	0.222	-0.257	-0.304	0.312	0.330	-0.199
Wave II OBJ.	0.437	0.220	0.047	0.356	0.451	0.273	0.099	0.332
Wave I SUBJ. ←								
Wave I OBJ.	0.993	0.066	0.000	0.653	1.113	0.067	0.000	0.710



*Means*

Wave I OBJ.	2.250	0.016	0.000	2.258	0.015	0.000
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*Intercepts*

Wave II OBJ.	-0.213	0.077	0.006	-0.082	0.102	0.423
Wave III OBJ.	-0.370	0.076	0.000	0.216	0.065	0.001
Wave IV OBJ.	0.038	0.132	0.771	0.173	0.097	0.073
Wave I SUBJ.	1.120	0.154	0.000	0.494	0.154	0.001
Wave II SUBJ.	0.323	0.110	0.009	0.285	0.110	0.010
Wave III SUBJ.	1.179	0.077	0.000	0.401	0.077	0.000
Wave IV SUBJ.	0.575	0.141	0.009	0.489	0.141	0.001

*Variances*

Wave I OBJ.	0.191	0.014	0.000	0.192	0.016	0.000
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*Residual Variances*

Wave I BMI	0.049	0.008	0.000	0.061	0.008	0.000
Wave II BMI	0.049	0.008	0.000	0.061	0.008	0.000
Wave III BMI	0.106	0.011	0.000	0.110	0.009	0.000
Wave IV BMI	0.106	0.011	0.000	0.110	0.009	0.000
Wave I PW	0.175	0.018	0.000	0.140	0.022	0.000
Wave II PW	0.175	0.018	0.000	0.140	0.022	0.020
Wave III PW	0.219	0.021	0.000	0.204	0.019	0.000
Wave IV PW	0.219	0.021	0.000	0.204	0.019	0.622
Wave II OBJ.	0.043	0.016	0.007	0.031	0.014	0.000
Wave III OBJ.	0.131	0.010	0.000	0.103	0.009	0.000
Wave IV OBJ.	0.154	0.018	0.000	0.125	0.022	0.000
Wave I SUBJ.	0.253	0.030	0.000	0.234	0.027	0.000
Wave II SUBJ.	0.041	0.032	0.200	0.071	0.031	0.001
Wave III SUBJ.	0.059	0.014	0.000	0.055	0.012	0.000
Wave IV SUBJ.	0.048	0.035	0.168	0.017	0.034	0.000

*R-Square*

Wave I BMI	0.796			0.759		
Wave II BMI	0.851			0.798		

Wave III BMI	0.825	0.760
Wave IV BMI	0.868	0.820
Wave I PW	0.716	0.771
Wave II PW	0.705	0.761
Wave III PW	0.643	0.669
Wave IV PW	0.661	0.683
Wave II OBJ.	0.845	0.872
Wave III OBJ.	0.737	0.703
Wave IV OBJ.	0.779	0.750
Wave I SUBJ.	0.427	0.504
Wave II SUBJ.	0.903	0.841
Wave III SUBJ.	0.850	0.866
Wave IV SUBJ.	0.888	0.962

N=7,105.

Results based on casewise maximum likelihood (also called FIML) estimation to account for missing data, with robust (Huber-White) standard errors.

Model fit statistics:  $\chi^2 = 24.508$ , DF = 16, BIC = -117.389, CFI = 0.999, TLI = 0.997, 1-RMSEA = 0.988.

**Table A.13** Coefficient Estimates for Health Outcomes Regressed on Subjective Weight (SUBJ.) and Objective Weight (OBJ.), Adjusted for Age, Gender, Race/Ethnicity, and Education

Parameters	Estimate	Std. Err.	P-value	Stdz. Est.
<i>Health Outcome Regressions</i>				
Wave V BMI ←				
Wave IV OBJ.	0.945	0.068	0.000	0.835
Wave IV SUBJ.	-0.047	0.078	0.546	-0.038
Age	-0.020	0.014	0.164	
Female	0.057	0.048	0.239	
NH-Black	0.115	0.057	0.043	
Hispanic	0.095	0.079	0.227	
NH-Other	0.035	0.104	0.734	
Wave V BA+	-0.185	0.076	0.015	
SBP ←				
Wave IV OBJ.	0.656	0.162	0.000	0.352
Wave IV SUBJ.	-0.035	0.190	0.855	-0.017
Age	0.024	0.021	0.242	
Female	-0.926	0.102	0.000	
NH-Black	0.420	0.093	0.000	
Hispanic	-0.196	0.145	0.175	
NH-Other	-0.030	0.170	0.859	
Wave V BA+	-0.172	0.123	0.161	
DBP ←				
Wave IV OBJ.	0.411	0.137	0.003	0.291
Wave IV SUBJ.	-0.031	0.163	0.851	-0.020
Age	0.035	0.016	0.034	
Female	-0.525	0.085	0.000	
NH-Black	0.281	0.073	0.000	
Hispanic	-0.239	0.122	0.049	
NH-Other	0.162	0.150	0.281	
Wave V BA+	-0.167	0.097	0.086	
Measured Hypertension/Rx ←				
Wave IV OBJ.	0.345	0.130	0.008	0.835
Wave IV SUBJ.	0.268	0.157	0.088	-0.038
Age	0.018	0.019	0.364	
Female	-0.541	0.093	0.000	
NH-Black	0.304	0.093	0.001	
Hispanic	-0.357	0.156	0.022	
NH-Other	0.219	0.215	0.309	
Wave V BA+	-0.156	0.143	0.278	
CRP ←				

Wave IV OBJ.	0.378	0.040	0.000	0.438
Wave IV SUBJ.	-0.145	0.048	0.003	-0.153
Age	-0.028	0.010	0.003	
Female	0.286	0.042	0.000	
NH-Black	0.048	0.043	0.262	
Hispanic	-0.025	0.057	0.667	
NH-Other	0.138	0.068	0.042	
Wave V BA+	-0.143	0.079	0.069	
Latent Depression ←				
Wave IV OBJ.	-0.097	0.051	0.059	-0.112
Wave IV SUBJ.	0.120	0.058	0.040	0.126
Age	-0.005	0.007	0.487	
Female	0.038	0.038	0.324	
NH-Black	0.036	0.037	0.328	
Hispanic	-0.069	0.053	0.192	
NH-Other	-0.109	0.047	0.020	
Wave V BA+	-0.036	0.049	0.462	
Depression Dx ←				
Wave IV OBJ.	-0.183	0.109	0.092	-0.130
Wave IV SUBJ.	0.307	0.129	0.017	0.200
Age	-0.016	0.018	0.359	
Female	0.354	0.082	0.000	
NH-Black	-0.321	0.082	0.000	
Hispanic	-0.347	0.095	0.000	
NH-Other	-0.438	0.130	0.001	
Wave V BA+	0.037	0.091	0.681	
Anxiety Dx ←				
Wave IV OBJ.	-0.304	0.111	0.006	-0.215
Wave IV SUBJ.	0.364	0.134	0.007	0.235
Age	-0.023	0.019	0.226	
Female	0.323	0.068	0.000	
NH-Black	-0.426	0.086	0.000	
Hispanic	-0.351	0.118	0.003	
NH-Other	-0.564	0.154	0.000	
Wave V BA+	-0.165	0.092	0.072	
Trouble Sleeping ←				
Wave IV OBJ.	0.027	0.086	0.758	0.015
Wave IV SUBJ.	0.012	0.101	0.902	0.006
Age	0.009	0.018	0.616	
Female	0.370	0.060	0.000	
NH-Black	-0.194	0.071	0.007	
Hispanic	-0.288	0.119	0.015	
NH-Other	-0.150	0.158	0.343	
Wave V BA+	0.156	0.108	0.151	

*SWS and BMI Regressions*

Wave IV OBJ. ←

Wave III OBJ.	1.051	0.064	0.000	0.868
Wave III SUBJ.	0.037	0.071	0.606	0.032
Age	-0.019	0.008	0.014	
Female	0.018	0.039	0.638	
NH-Black	0.046	0.037	0.214	
Hispanic	0.037	0.036	0.300	
NH-Other	-0.088	0.068	0.197	
Wave IV BA+	-0.039	0.059	0.510	

Wave III OBJ. ←

Wave II OBJ.	0.750	0.039	0.000	0.619
Wave II SUBJ.	0.265	0.027	0.000	0.290
Age	0.006	0.007	0.412	
Female	-0.091	0.026	0.000	
NH-Black	0.070	0.025	0.005	
Hispanic	0.047	0.031	0.133	
NH-Other	0.005	0.035	0.886	
Wave III In College +	-0.026	0.029	0.379	

Wave II OBJ. ←

Wave I OBJ.	0.925	0.069	0.000	0.791
Wave I SUBJ.	0.091	0.033	0.006	0.123
Age	-0.001	0.006	0.885	
Female	-0.025	0.019	0.179	
NH-Black	0.025	0.016	0.132	
Hispanic	-0.017	0.023	0.458	
NH-Other	-0.049	0.044	0.270	
Parent BA+	-0.049	0.026	0.061	

Wave I OBJ. ←

Age	0.029	0.006	0.000
Female	-0.013	0.020	0.496
NH-Black	0.090	0.028	0.001
Hispanic	0.063	0.039	0.106
NH-Other	0.057	0.051	0.264
Parent BA+	-0.058	0.025	0.021

Wave IV SUBJ. ←

Wave III SUBJ.	0.922	0.087	0.000	0.886
Wave III OBJ.	-0.896	0.129	0.000	-0.811
Wave IV OBJ.	0.799	0.077	0.000	0.876
Age	0.006	0.009	0.486	
Female	-0.021	0.049	0.667	
NH-Black	-0.052	0.047	0.272	
Hispanic	-0.085	0.047	0.070	
NH-Other	0.022	0.058	0.701	
Wave IV BA+	-0.096	0.047	0.040	

Wave III SUBJ. ←				
Wave II SUBJ.	0.344	0.028	0.000	0.355
Wave II OBJ.	-0.406	0.068	0.000	-0.316
Wave III OBJ.	0.937	0.070	0.000	0.882
Age	0.005	0.008	0.527	
Female	0.267	0.029	0.000	
NH-Black	-0.122	0.037	0.001	
Hispanic	0.066	0.041	0.107	
NH-Other	0.026	0.048	0.585	
Wave III In College +	0.013	0.026	0.626	
Wave II SUBJ. ←				
Wave I SUBJ.	0.902	0.040	0.000	0.921
Wave I OBJ.	-0.262	0.123	0.033	-0.169
Wave II OBJ.	0.286	0.095	0.003	0.216
Age	0.017	0.007	0.017	
Female	0.036	0.025	0.147	
NH-Black	0.021	0.035	0.548	
Hispanic	0.002	0.037	0.947	
NH-Other	0.135	0.047	0.004	
Parent BA+	0.007	0.027	0.791	
Wave I SUBJ. ←				
Wave I OBJ.	1.068	0.059	0.000	0.674
Age	-0.043	0.007	0.000	
Female	0.371	0.028	0.000	
NH-Black	-0.172	0.033	0.000	
Hispanic	-0.055	0.045	0.224	
NH-Other	-0.222	0.060	0.000	
Parent BA+	0.005	0.027	0.861	

N=6,247.

Observed BMI, observed SBP and DBP, and observed CRP divided by 10 to reduce variance and help with model convergence.

Latent variable regressions, covariances, means and intercepts, variances, and R-squared values omitted for parsimony.

Results based on casewise maximum likelihood (also called FIML) estimation to account for missing data among endogenous variables, with diagonal weighted least squares standard errors due to binary outcome variables.

Weighted estimates account for person-level longitudinal survey weights (WI-V), school-level clustering, and regional strata.

Model fit statistics:  $\chi^2 = 361.617$ , DF = 274, SBIC = -2033.104, CFI = 0.994, TLI = 0.991, 1-RMSEA = 0.993.

**Table A.14** Coefficient Estimates for Health Outcomes Regressed on Subjective Weight (SUBJ.) and Objective Weight (OBJ.), Adjusted for Age, Race/Ethnicity, and Education; Female vs. Male

Parameters	Female				Male			
	Estimate	Std. Err.	P-value	Stdz. Est.	Estimate	Std. Err.	P-value	Stdz. Est.
<i>Health Outcome Regressions</i>								
Wave V BMI ←								
Wave IV OBJ.	0.968	0.085	0.000	0.875	0.907	0.116	0.000	0.799
Wave IV SUBJ.	-0.135	0.113	0.231	-0.093	0.013	0.121	0.917	0.011
Age	-0.022	0.019	0.239		-0.021	0.018	0.236	
NH-Black	0.145	0.066	0.028		0.065	0.078	0.405	
Hispanic	0.073	0.108	0.497		0.113	0.128	0.378	
NH-Other	0.103	0.218	0.635		-0.014	0.103	0.889	
Wave V BA+	-0.321	0.095	0.001		0.001	0.143	0.997	
SBP ←								
Wave IV OBJ.	0.511	0.177	0.004	0.329	1.016	0.274	0.000	0.508
Wave IV SUBJ.	-0.054	0.234	0.819	-0.026	-0.231	0.276	0.404	-0.117
Age	0.004	0.021	0.852		0.044	0.036	0.224	
NH-Black	0.452	0.111	0.000		0.384	0.156	0.014	
Hispanic	-0.251	0.201	0.212		-0.188	0.232	0.417	
NH-Other	-0.127	0.293	0.664		0.036	0.236	0.879	
Wave V BA+	-0.192	0.154	0.211		-0.145	0.235	0.538	
DBP ←								
Wave IV OBJ.	0.158	0.127	0.213	0.132	0.792	0.233	0.001	0.506
Wave IV SUBJ.	0.210	0.170	0.215	0.135	-0.346	0.230	0.133	-0.224
Age	0.018	0.019	0.331		0.047	0.026	0.077	
NH-Black	0.372	0.083	0.000		0.169	0.113	0.135	
Hispanic	-0.348	0.154	0.023		-0.161	0.201	0.424	
NH-Other	-0.074	0.235	0.754		0.380	0.221	0.086	
Wave V BA+	-0.192	0.110	0.079		-0.117	0.187	0.531	
Measured Hypertension/Rx ←								





Wave IV OBJ.	-0.286	0.127	0.024	-0.225	-0.464	0.212	0.028	-0.303
Wave IV SUBJ.	0.487	0.184	0.008	0.293	0.369	0.212	0.081	0.244
Age	-0.052	0.025	0.037		0.006	0.027	0.827	
NH-Black	-0.451	0.101	0.000		-0.413	0.142	0.004	
Hispanic	-0.343	0.121	0.005		-0.358	0.191	0.062	
NH-Other	-0.529	0.207	0.011		-0.563	0.209	0.007	
Wave V BA+	-0.220	0.104	0.034		-0.077	0.171	0.651	
Trouble Sleeping ←								
Wave IV OBJ.	0.057	0.113	0.613	0.035	0.071	0.143	0.622	0.036
Wave IV SUBJ.	0.097	0.149	0.517	0.045	-0.139	0.147	0.344	-0.072
Age	0.010	0.025	0.679		0.005	0.024	0.845	
NH-Black	-0.305	0.103	0.003		-0.137	0.098	0.163	
Hispanic	-0.177	0.142	0.214		-0.402	0.158	0.011	
NH-Other	-0.026	0.188	0.891		-0.261	0.222	0.240	
Wave V BA+	-0.007	0.144	0.964		0.375	0.162	0.020	
<i>SWS and BMI Regressions</i>								
Wave IV OBJ. ←								
Wave III OBJ.	0.903	0.076	0.000	0.744	1.319	0.144	0.000	1.091
Wave III SUBJ.	0.185	0.095	0.052	0.137	-0.194	0.128	0.128	-0.186
Age	0.001	0.010	0.918		-0.047	0.012	0.000	
NH-Black	0.121	0.042	0.004		-0.009	0.057	0.869	
Hispanic	0.029	0.051	0.577		0.068	0.069	0.323	
NH-Other	-0.110	0.098	0.261		-0.085	0.078	0.278	
Wave IV BA+	-0.079	0.061	0.197		0.004	0.137	0.979	
Wave III OBJ. ←								
Wave II OBJ.	0.814	0.048	0.000	0.625	0.622	0.075	0.000	0.561
Wave II SUBJ.	0.290	0.036	0.000	0.280	0.285	0.048	0.000	0.337
Age	-0.011	0.011	0.288		0.025	0.009	0.003	
NH-Black	0.067	0.041	0.102		0.044	0.039	0.259	
Hispanic	0.026	0.045	0.565		0.058	0.051	0.252	
NH-Other	-0.006	0.058	0.923		0.042	0.051	0.414	
Wave III In College +	-0.018	0.039	0.645		-0.055	0.039	0.155	

Wave II OBJ. ←								
Wave I OBJ.	0.968	0.080	0.000	0.799	0.819	0.108	0.000	0.729
Wave I SUBJ.	0.078	0.038	0.038	0.100	0.144	0.054	0.008	0.193
Age	-0.010	0.007	0.173		0.012	0.011	0.250	
NH-Black	0.053	0.029	0.063		-0.004	0.029	0.883	
Hispanic	-0.018	0.029	0.550		-0.015	0.039	0.693	
NH-Other	-0.112	0.071	0.113		0.041	0.057	0.473	
Parent BA+	-0.015	0.035	0.666		-0.070	0.038	0.066	
Wave I OBJ. ←								
Age	0.022	0.009	0.013		0.035	0.008	0.000	
NH-Black	0.223	0.030	0.000		-0.042	0.046	0.365	
Hispanic	0.095	0.046	0.039		0.031	0.054	0.562	
NH-Other	0.028	0.063	0.652		0.092	0.060	0.123	
Parent BA+	-0.081	0.027	0.003		-0.025	0.033	0.452	
Wave IV SUBJ. ←								
Wave III SUBJ.	0.958	0.098	0.000	0.925	1.027	0.228	0.000	0.968
Wave III OBJ.	-0.758	0.106	0.000	-0.816	-1.369	0.444	0.002	-1.118
Wave IV OBJ.	0.644	0.080	0.000	0.841	1.107	0.247	0.000	1.094
Age	0.007	0.013	0.597		0.034	0.021	0.099	
NH-Black	0.065	0.056	0.242		-0.141	0.086	0.101	
Hispanic	0.039	0.065	0.544		-0.150	0.110	0.171	
NH-Other	0.038	0.065	0.560		0.031	0.088	0.727	
Wave IV BA+	0.133	0.054	0.014		0.004	0.134	0.975	
Wave III SUBJ. ←								
Wave II SUBJ.	0.341	0.034	0.000	0.366	0.269	0.055	0.000	0.275
Wave II OBJ.	-0.422	0.087	0.000	-0.360	-0.336	0.097	0.001	-0.263
Wave III OBJ.	0.853	0.084	0.000	0.949	1.083	0.117	0.000	0.939
Age	0.015	0.011	0.191		-0.013	0.011	0.229	
NH-Black	-0.137	0.045	0.002		-0.076	0.058	0.189	
Hispanic	0.034	0.058	0.561		0.097	0.080	0.224	
NH-Other	0.006	0.059	0.922		0.007	0.068	0.922	
Wave III In College +	0.037	0.034	0.274		0.040	0.039	0.302	

Wave II SUBJ. ←								
Wave I SUBJ.	0.900	0.057	0.000	0.916	0.862	0.069	0.000	0.886
Wave I OBJ.	-0.210	0.142	0.138	-0.138	-0.157	0.167	0.348	-0.107
Wave II OBJ.	0.228	0.108	0.035	0.182	0.237	0.146	0.104	0.181
Age	0.015	0.009	0.097		0.014	0.011	0.206	
NH-Black	0.037	0.041	0.366		0.011	0.053	0.841	
Hispanic	-0.011	0.050	0.824		0.012	0.049	0.799	
NH-Other	0.012	0.062	0.848		0.244	0.072	0.001	
Parent BA+	0.002	0.040	0.968		-0.007	0.040	0.865	
Wave I SUBJ. ←								
Wave I OBJ.	1.028	0.063	0.000	0.664	1.094	0.085	0.000	0.724
Age	-0.001	0.011	0.957		-0.081	0.010	0.000	
NH-Black	-0.258	0.043	0.000		-0.086	0.051	0.092	
Hispanic	-0.045	0.053	0.395		-0.055	0.059	0.348	
NH-Other	-0.180	0.087	0.039		-0.297	0.080	0.000	
Parent BA+	-0.016	0.042	0.702		0.011	0.039	0.768	

N(Female)=4,152; N(Male)=2,606.

Observed BMI, observed SBP and DBP, and observed CRP divided by 10 to reduce variance and help with model convergence.

Latent variable regressions, covariances, intercepts, variances, and R-squared values omitted for parsimony.

Results based on casewise maximum likelihood (also called FIML) estimation to account for missing data among endogenous variables, with diagonal weighted least squares standard errors due to binary outcome variables.

Weighted estimates account for person-level longitudinal survey weights (WI-V), school-level clustering, and regional strata.

Model fit statistics for Male respondents:  $\chi^2 = 295.971$ , DF = 268, SBIC = -1812.002, CFI = 0.996, TLI = 0.993, 1-RMSEA = 0.994.

Model fit statistics for Female respondents:  $\chi^2 = 369.979$ , DF = 268, SBIC = -1827.625, CFI = 0.990, TLI = 0.983, 1-RMSEA = 0.990.