

# Childhood income dynamics and adult adiposity

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**Abstract**

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Conventional wisdom—reflected in literature reviews as well as newspaper and magazine stories—holds that poor children are at risk for developing obesity and that there is a negative gradient between income and body mass. This wisdom is reflected in programs and interventions targeting the nutrition and exercise habits of poor children. In fact, there appears to be far more literature discussing mechanisms for these relationships, from relative food prices to access to parks to epigenetic programming, than literature supporting a causal effect of childhood economic resources on later body mass.

This project set out to investigate what it is about income in childhood that is related to later excess body fat. I investigated different poverty definitions (demonstrating a methodology for empirically deriving optimal poverty thresholds), the marginal effect of more income within small ranges of income (in semi-parametric models), and instability of income net of permanent childhood income. I analyzed these putative causal factors via models for moving beyond crude measures whereby a single indicator of economic circumstances at some unspecified point in childhood stands in for all childhood experiences. The aim was to isolate what aspect of childhood income at what point in childhood accounts for the apparent effect of relative economic deprivation on body mass. Fixed effects models would show that the total effect of childhood economic conditions was robust to unobserved heterogeneity.

Where I find effects consistent with the conventional wisdom, they tend to be small.



Testing five poverty definitions across five models of childhood poverty dynamics, the largest single effect is for spending all of one's childhood below either 185% or 200% of the federal poverty level, versus never being poor under these definitions, accounting for a 2.4% higher body mass index (BMI) in adulthood. For a baseline BMI of 20 (30), this implies a new BMI of 20.5 (30.7). The best fitting model suggests that spending all of early childhood (the birth year through the year the child turns 5) and all of middle childhood (6 to 11) but none of adolescence below 185% of the usual poverty line produces a BMI around 3.8% higher, moving from 20 (30) to 20.8 (31.1). Modeling the marginal effect of all childhood income from 0 to 750% of the poverty line suggests a reduction in BMI of 3 points over this range for white women, and nearly 3 points for African American females over the same income range in middle childhood. Moving over the entire possible range of income instability in early childhood accounts for a BMI around 3.8% higher.

These results are not consistent, however: Higher income (up to 750% of poverty) is associated with *higher* BMIs for African American males and generally little difference for white males and Hispanics, and fixed effects models suggest only protective effects—*lower* adult BMI—of more of childhood spent in poverty or experiencing income declines in childhood. Furthermore, the fixed effects models suggest that, controlling for income instability and other factors, higher childhood permanent income is associated with *higher* adult BMI, the opposite of the conventional wisdom.

People react to given economic conditions in different ways, depending upon family and cultural norms, the availability of consumption-smoothing resources such as wealth or family support, community resilience factors, etc. Individual differences in stress perception and coping skills will further moderate the health effects of what appear to be otherwise equivalently stressful situations. While much attention has been paid to the obesity risk associated with low income, and some to the risk attendant to income instability, this analysis suggests any long-term effects of childhood income dynamics are small at best. This does not necessarily conflict with research that shows within-childhood effects of income or poverty on obesity risk, or research that provides causal evidence of the adiposity contri-



butions of income dynamics within adulthood. It does suggest that there is a disconnect, that moderators occurring after childhood will nearly as often make the well off large and the poor slender as what the conventional wisdom might dictate.



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## DEDICATION

To my dear wife, Duana: That'll do.

## Chapter 1

**SOCIOECONOMIC EXPERIENCES AND ADIPOSITY: EVIDENCE, MODELS, AND MECHANISMS**

Chances are you have read or watched or heard something about food, nutrition, exercise, or obesity very recently. The topic of obesity may be unique in the extent to which so many academics, advocates, and advisors have something to say. And so we have a cacophony of conventional wisdom and favored explanations, a flood of diet tips and exhortations to exercise more. Yet obesity prevalence in the United States and other wealthy nations and the attendant health risks of excess adiposity (body fat) continue to increase.

The multiple voices illustrate the multiple potential causes and numerous lenses with which we can examine obesity. Once one steps back from the core drivers of obesity, energy intake and energy expenditure, no single cause will account for a large portion of the variance in obesity within a population, and no single cure will suffice. Some consensus is building on basic risk and protective factors for obesity. One concern is how obesity, like other health outcomes, appears to be related to social disadvantage. This conventional wisdom was expressed in a recent article in *The Atlantic*: “In fact, obesity has become a marker of sorts for lower socioeconomic status. . . . Untangling correlation and causation is difficult, and many of the causes overlap. But obesity researchers increasingly believe that material disadvantages best explain the spread of obesity among poor people” (Ambinder, 2010, p. 76). Yet one indicator of disadvantage, the prevalence of poverty, has changed little while obesity has attained what many call epidemic status.

In this dissertation, I draw largely but not exclusively on literature from the fields of economics, sociology, and public health. My main aim is to transcend associations that rely on correlational data and simple measures. More detailed investigation is beginning to tease out the complexities of the relationship between socioeconomic status (SES) and

body mass. In the sections that follow, I attempt to provide some disentangling, usually by addressing first the cross-sectional data and then research that attempts to provide stronger evidence for causation (e.g., via accounting for selection and/or using longitudinal data to establish time precedence). The complexity of SES includes different experiences and effects of income (or education or wealth) by race or ethnicity and gender.

There is a growing awareness that obesity is both a consequence of and a contributor to poverty. Obesity may thus contribute to perpetuating poverty among adults and children. I briefly address both directions of this relationship within adults. I then review evidence of the effects of SES in childhood on later adiposity. The conventional wisdom about income and obesity is reflected in numerous articles and policy statements regarding the likely overweight adulthood facing today's poor children. I show, however, that little research exists to support a causal role of childhood income experiences and later body mass.

Understanding the nature of the relationship between obesity and earlier socioeconomic experiences will contribute to our knowledge about transmission of health and poverty and aid targeting of intervention. Investigating and refining the operational definitions of putative causes—beginning to differentiate what it is about poverty or income experiences that seems to result in obesity—is key to addressing this form of social disadvantage. In this chapter, I build an argument for studying the relationship between childhood experiences of income and poverty and obesity.

The results of this project undermine the conventional wisdom and question the modern focus on low income children being at risk for the poor health attendant to obesity. The conventional wisdom is overly simplistic: Poverty puts children at risk of higher body mass, and higher income lowers body mass. Put to the test in a longitudinal framework, in which I capture dynamics of childhood income experiences, I find support for the relatively small number of authors who have pointed out the variation and nuance in these relationships. A simple indicator of ever being in poverty during childhood is not predictive of later adiposity. Poverty accumulation or experiences within developmental periods are, if anything,

protective, resulting in lower adult body mass. The effects of income during childhood are strikingly inconsistent across racial, ethnic, and gender groups and across developmental periods, which more income sometimes associated with higher, not lower, adiposity. Finally, I find the relationship between instability of economic experiences and adult adiposity, net of permanent childhood income, appears to be different than other conclusions about this relationship within childhood or within adulthood. The lack of relationship between income, poverty, or instability in childhood and later body mass does not necessarily conflict with other research showing significant relationships within childhood or within adulthood. It does, however, imply any causal effect of economic experiences is relatively short-run or easily moderated, and targeting low-income children may not be efficient use of limited policy resources.

### ***1.1 Socioeconomic status and obesity***

Although there is little doubt that there are genetic influences on obesity, obesity is essentially about caloric intake exceeding expenditure over time. As for causing obesity itself, “the genes or gene variants that would support [an obesity genotype] hypothesis have not been identified” (Caprio et al., 2008, p. 2214), and obesity dynamics have simply occurred too quickly for changes in genetics to be a cause (Bleich, Cutler, Murray, & Adams, 2008), leaving us with a broad and varied literature about potential influences on the proximal causes of diet and exercise. Excess body fat represents a marker of important health behaviors which are independently associated with health outcomes; a risk factor for cardiovascular disease, diabetes, and some types of cancers (Danaei et al., 2009; Visscher & Seidell, 2001); and a contributor to poor social, psychological, and economic outcomes (e.g., Cawley, 2004; Cutler, Glaeser, & Shapiro, 2003; Daniels, 2006).

Obesity is but one example of health inequalities that exist in the US and throughout the developed world, and this study is aligned with efforts to reduce socioeconomic and racial/ethnic disparities in health. The focus here is on the former, but I will return to the latter momentarily. Obesity patterns tend to mirror general demographic patterns of health disparities. Those with lower levels of income and education have higher risk of

obesity (Averett, 2012; Caprio et al., 2008; Cutler et al., 2003; Kant & Graubard, 2007; Storey, Forshee, Weaver, & Sansalone, 2003). While these gradients in obesity by either income or education, the two most common indicators of socioeconomic status (SES), are commonly cited, further analysis lends further nuance.

The relationship between income and BMI<sup>1</sup> or obesity<sup>2</sup> is not monotonic. For example, restricting the Continuing Survey of Food Intake by Individuals (CSFII) to those with incomes less than 130% of poverty found a positive relation between income and the probability of obesity and BMI for women and no relationship for men, net of other demographics, homeowner status, food stamp participation, and other covariates (Chen, Yen, & Eastwood, 2005). Self-report data from 2005 show that the obesity prevalence was higher among those 18-64 in households with income between \$10,000 and \$15,000 than among adults from the lowest income households. Obesity rates declined monotonically with income category for women but not for men, for whom obesity prevalence was highest in households with incomes between \$50,000 and \$75,000 (Schmeiser, 2009). Combined data from all waves of the National Health and Nutrition Examination Surveys (NHANES) illustrate the gradient, as seen in Figure 1.1—the probability of being obese significantly declines with income categorized in relation to the poverty level (i.e. below poverty, 100%–199%, 200%–299%, 300%–399%, and 400% and above)—but most of the decrease occurs above the middle category (2 to 3 times the poverty level; Kant & Graubard, 2007).

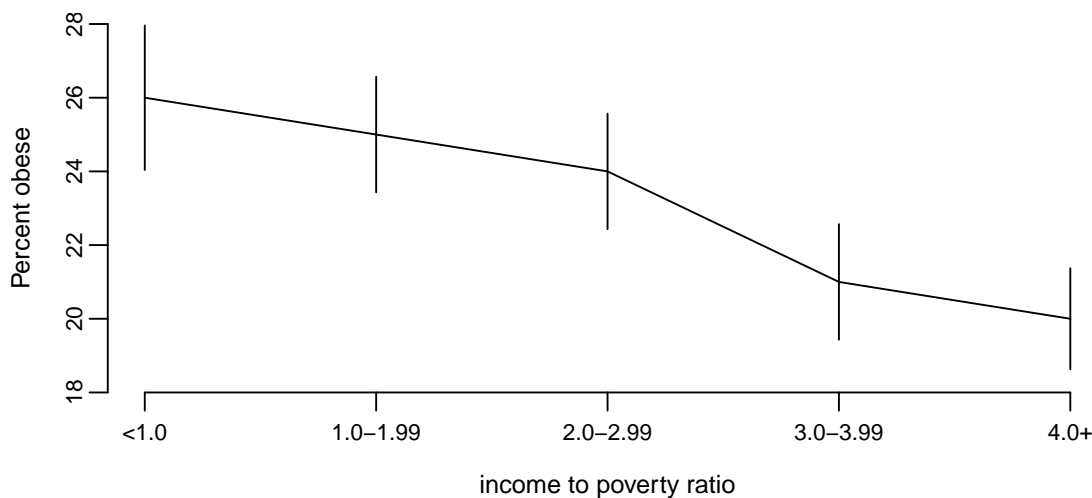
In their more detailed analysis, Komlos and Brabec (2010) found that BMI increases with income below 100% of the federal poverty line for black females and then decreases, and increases up to 250% and 300% of poverty for white and black men, respectively. Only among white women is a clear, monotonic relationship evident between income (at least up to 600% of poverty) and BMI. The income effect may be different among Hispanics

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<sup>1</sup>The most common measure of adiposity (accumulation of fatty tissue) is the Body Mass Index: BMI = weight in kilograms ÷ (height in meters)<sup>2</sup>

<sup>2</sup>Basic adiposity categories include normal weight (BMI < 25), overweight (BMI ≥ 25), and obese (BMI ≥ 30). The latter can be subdivided into moderately obese (Grade 1, BMI < 35), severely obese (Grade 2, BMI < 40), and profoundly or morbidly obese (Grade 3, BMI ≥ 40).

Figure 1.1: Predicted obesity prevalence (with 95% confidence intervals) among adults 25 to 74 by poverty-income ratio group, NHANES 1971 through 2002 from Kant and Graubard (2007; Table 4)



as well. Analysis of child longitudinal data, for example, found that baseline BMI among Hispanic children of native-born parents had a similar but up-shifted pattern as that of non-Hispanic white children, increasing with household income, but the baseline BMI of Hispanic children of immigrants did not differ by income. BMI growth of Hispanic children did not differ with income, while that of white children displayed the familiar negative income gradient (Balistreri & Van Hook, 2009).

There are various policy, empirical, and methodological arguments for considering racial/ethnic and gender differences in examining obesity, differences that would be masked—and might reduce overall effect estimates—if only the total population is considered. From the grassroots to the Federal government, one prominent policy area concerns health disparities, culminating in the government’s Healthy People goals.

Health disparities are systematically linked with social disadvantage, and may reflect social disadvantage, although a causal link does not need to be demonstrated. Whether or not a causal link exists, health disparities adversely affect

groups who are already disadvantaged socially, putting them at further disadvantage with respect to their health, thereby making it potentially more difficult to overcome social disadvantage. This reinforcement or compounding of social disadvantage is what makes health disparities relevant to social justice even when knowledge of their causation is lacking (Braveman et al., 2011, p. S151).

Adiposity is one example of such health disparities. Government statistics frequently present prevalence of obesity and overweight<sup>1</sup> by gender and ethnicity (e.g., Ogden, Lamb, Carroll, & Flegal, 2010). Women are less likely to be overweight than men but more likely to be obese (Cook & Daponte, 2008). Prevalence and trends in obesity exhibit “significant variation by racial and ethnic groups” (Flegal, Carroll, Ogden, & Curtin, 2010, p. 240). For example, Flegal and colleagues’ analysis of NHANES 2007-2008 found adult (age 20 and above) age-adjusted prevalences of obesity ranging from 31.9% for non-Hispanic white men to 49.6% among non-Hispanic African American women. The gender gap was 1.1 percentage points for whites, 8.7 points for Hispanics, and 12.3 points for blacks. Blacks were also significantly more likely to be in the categories of severely obese and morbidly obese than whites or Hispanics: 14.2% of black women were diagnosed with Grade 3 obesity (morbidly obese, BMI  $\geq$  40) versus 7.0% of Hispanic women and 6.4% of white women. These disparities as well as overall levels of obesity are targets of the Healthy People objectives.

Various methodological concerns require accounting for gender and race/ethnicity in a study of how economic background affects later health outcomes. First, a given level of income or poverty status does not have the same meaning in terms of deprivation and disadvantage for all groups. At a given level of income, wealth and debt levels vary by race/ethnicity (Caprio et al., 2008; LaVeist, 2005). An income-based indicator of “poverty” gives an incomplete accounting of an individual’s context-specific experience of material disadvantage. Among the poor, the poverty gap—the amount by which family income is below the relevant poverty threshold—is larger for nonwhites (Meyer & Wallace, 2009). On a monthly basis, blacks and Hispanics are more likely than whites to be poor in multiple months of a year or all months over a period, and have longer spells of poverty (Jantti, 2009).

Card and Blank (2008) found that after the initiation of a poverty spell (when incomes were comparable) family income for black female-headed households increased more slowly and to a lower level than white female-headed households. Thus, incorporating race/ethnicity could be seen as a way to account for differences in measurement error from a yearly poverty indicator or count of family income.

It is important to separate consideration of SES and race/ethnicity in predicting health and other outcomes, although they are often confounded (LaVeist, 2005). Each is independently associated with “multiple environmental and social risk factors... over a prolonged period of time,” contributing to the development of chronic conditions (Suglia, Duarte, Chambers, & Boynton-Jarrett, 2012, p. e1174). Poor outcomes may be more likely in the presence of multiple sources of disadvantage and/or long-lasting disadvantage (Braveman et al., 2011). Income may increase, but the social disadvantage conferred due to minority status has persisted for generations. Access to services and other resources and physiological stress levels all vary by race/ethnicity within a given level of income or education (Caprio et al., 2008). Demographic markers reflect important differences in experiences, history and background, and connection to institutions as well as possible differences in physiology. “The behaviors leading to health outcomes, not just health outcomes per se, are influenced by biological or socioenvironmental factors” (Huang, Drewnowski, Kumanyika, & Glass, 2009, p. 2). For example, research has shown that mostly African American neighborhoods have less access to chain supermarkets, which generally have a larger variety of food items, higher quality produce, and lower prices than non-chain stores, than mostly white neighborhoods of the same average income (Walker, Keane, & Burke, 2010). These different experiences may result in different relationships between an income-based measure of disadvantage and an outcome such as adiposity. An increase from \$20,000 to \$22,000 in income for a family of 4 might result in purchasing more nutritious food, or might be diverted to paying off debts in a low wealth family, put towards tuition in a low education family, or used to simply purchase higher quantities of less nutritious food where healthier food is not available or not culturally sanctioned. In addition to this differential effect of changes in income, different groups may have a different baseline of the health outcome in question—they may already

have had different health or obesity status when income was \$20,000. While this review has mostly concentrated on differences in the relationship between economic resources and adiposity by race/ethnicity, “research indicates the poverty–obesity relationship varies by sex” (Lee, Harris, & Gordon-Larsen, 2009, p. 507).

Energy intake and expenditure are embedded in cultural contexts regarding the meaning of food and importance attached to certain foods, norms regarding physical activity, and body image ideals, as well as a larger social environment of food prices, availability of energy-dense foods, and access to exercise (Caprio et al., 2008; Huang et al., 2009). Differences in experiences of stress, which (as we will see) is thought to be a driver of individual obesity, remain between ethnic groups at the same income level (Caprio et al., 2008). Similarly, as illustrated in the above research (Balistreri & Van Hook, 2009; Chen et al., 2005; Komlos & Brabec, 2010; Schmeiser, 2009) and explored further below, the effects of putative causes and effects of obesity appear to differ by ethnicity and gender. Thus, incorporating not only income but other descriptors that may capture how that income is experienced allows for more precise estimates by fitting different intercepts and different slopes.

Finally, there are arguments to considering gender and race/ethnicity in the measurement of adiposity itself. The same obesity prevalence or a specific value or range of BMI does not have identical meaning across all groups. As a measure of adiposity, BMI does not differentiate between fat and fat-free mass, so that an additional 10 pounds of muscle will increase BMI the same as an additional 10 pounds of fat. Differences in BMI therefore do not necessarily imply differences in body fat (Flegal et al., 2010) and BMI tends to produce varying degrees of misclassification of obesity status when judged against a more direct measure of adiposity (Burkhauser & Cawley, 2008). Meanwhile, there appear to be different patterns of fat distribution and metabolism by ethnicity and thus differences in health consequences at the same BMI or same category of BMI, although these differences are much smaller than those between adiposity categories (Caprio et al., 2008). Finally, while there are racial/ethnic and gender dimensions to BMI and obesity, it should be noted “that the overwhelming majority of demographic groups [have] experienced gains in their

average BMI” (Koch & Wilson, 2013, p. 18).

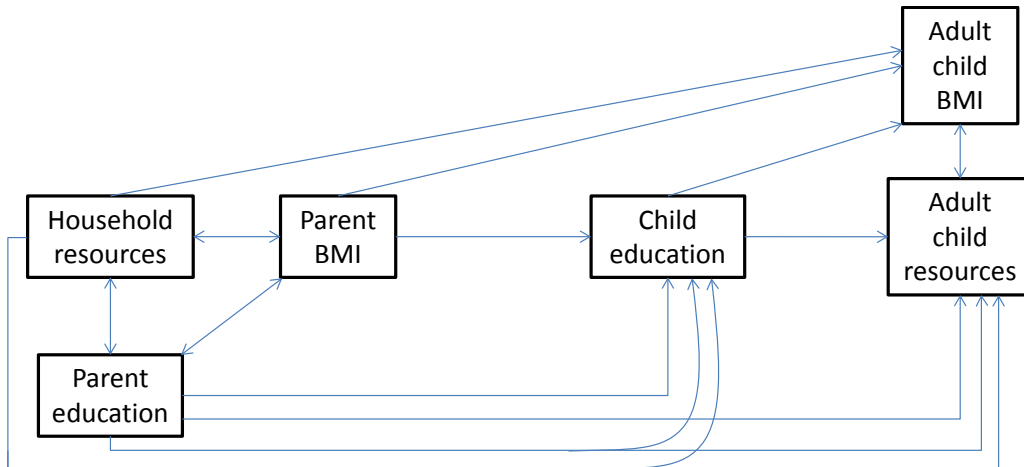
As with the oft-assumed income gradient, more detailed analysis questions the relationship between education and obesity. Evidence indicates the gradient may not be consistent across ethnic groups (e.g., Balistreri & Van Hook, 2009). Controlling for income and trichotomizing education (less than high school, high school, any postsecondary), Komlos and Brabec (2010) found no relationship between education and BMI across all national surveys combined (NHANES and its predecessor, the National Health Examination Survey). This result may illustrate the recent disappearance of a clear relationship in earlier NHANES surveys: By NHANES III (1988-1994) there was no difference in obesity levels between those with a high school degree and those who had not completed high school, and in NHANES 1999-2002 the proportion of respondents with less than a high school degree characterized as obese was the same as the prevalence among those with more than a high school degree (both of which had lower prevalence than those with high school only; Kant & Graubard, 2007). Meanwhile, obesity has increased for all education groups (Cutler et al., 2003).

Focusing on income and material resources, the relationship in Figure 1.1 could be due to income causing obesity, obesity causing income, or other factors predisposing people to both poverty and obesity. This proposal places this conundrum in a life course perspective, summarized in Figure 1.2. In the coming sections, I will summarize literature addressing the direction of causality between material resources and adiposity. I will build up the model in Figure 1.2 in sections, paying more attention to the core of what I aim to study: how family of origin material resources affect adult child adiposity.

## **1.2 Obesity causes SES**

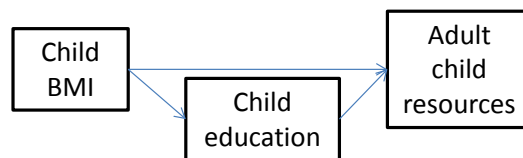
The effects of adiposity on one’s own material resources might be summarized as in Figure 1.3. The effects of education on future earnings and wealth are fairly well understood. I focus instead on the effects of weight status on education and future resources. Obesity is associated with mental (Daniels, 2006; Offer, Pechey, & Ulijaszek, 2010) and physical health conditions (e.g., Danaei et al., 2009; Daniels, 2006; Vandeloo, Bruckers, & Janssens,

Figure 1.2: Full model of intergenerational obesity



2007; Visscher & Seidell, 2001) that may directly impact human capital development and wages. Among the physical health conditions associated with obesity are diabetes, cardiovascular disease, cancer, musculoskeletal and mobility issues, shortness of breath, and sleep apnea, any one of which would impact educational attainment and work productivity and attendance. In addition, these conditions affect available material resources via increased medical copays or premiums. One estimate (Sturm, 2002, as cited in Daniels, 2006) found that the medical expenses of obese individuals are 36% higher than those of the non-obese.

Figure 1.3: Within person effects of obesity



Obese children and adolescents face the same health risks as obese adults, including cardiovascular, metabolic, respiratory, and psychological conditions (Caprio et al., 2008; Daniels, 2006). Early and prolonged exposure to these risks increases the chances of de-

veloping disease and disability. Children may be uniquely at risk for poor and life-long psychosocial outcomes, as obesity may result in “social isolation, poor school performance, and poor self-image” (Caprio et al., 2008, p. 2212). A number of studies have documented correlations between overweight or obesity and poor achievement in school. Overweight children are more likely to be documented as having impaired school functioning and behavior problems, are more likely to be in remedial or special education classes, and have lower math and reading standardized test scores. These differences are correlational, however, and may be due to other factors that may contribute to the excess adiposity, including poverty, poor nutrition, low parental education, or parental depression. Overweight status may cause impaired school performance through health-related absenteeism, sleep apnea, asthma, or depression, anxiety, and low self-esteem (Story, Kaphingst, & French, 2006), or the poor school performance may result in overeating due to stress and/or depression.

Similarly, Cawley (2004) noted that the basic relationship between weight and wages in adults could be due to (1) excess weight affecting wages via effects on health, productivity, or discrimination, (2) lower wages resulting in the excess adiposity, perhaps via diet mechanisms, or (3) unobserved characteristics that influence both weight and wages. Studies using longitudinal data—the Panel Study of Income Dynamics (PSID) or some flavor of the National Longitudinal Survey of Youth (NLSY)—that have attempted to account for such selection effects support the conclusion that obesity causes wages and family income, at least among women and particularly among white women (Cawley, 2004; Conley & Glauber, 2007; Gortmaker, Must, Perrin, Sobol, & Dietz, 1993). Focusing on black men, Conley and Glauber found a wage penalty, while Cawley’s individual fixed effects model actually found a positive association between BMI and wages. Cawley’s fixed effects and instrumental variables models each found a significant negative effect for white women. Gortmaker and colleagues concluded “that overweight during adolescence and young adulthood has important social and economic consequences that are more severe for women than for men and greater than those associated with a variety of other chronic conditions during adolescence” (p. 1011).

### **1.3 *The effect of one's own material resources on obesity***

As noted, many studies have noted an income or SES gradient to obesity, particularly female obesity. Many have noted that household wealth may be a better measure of SES than income. Similarly, a study using the PSID found that an individual's inflation-adjusted net worth was inversely related to risk of obesity, smoking, and hypertension, net of income (Hajat et al., 2010). Wealth may smooth consumption in the face of income instability (Dynan, Elmendorf, & Sichel, 2007) although wealth is highly correlated with income. As noted, the common finding of an educational gradient tends to disappear when controlling for factors such as income (Komlos & Brabec, 2010), and other analyses have concluded that the recent increase in overweight is not concentrated among the less educated (Cutler et al., 2003; Ruhm, 2012).

Relatively few studies move beyond cross-sectional associations and thus “have been unable to account for the potential endogeneity and reverse causality between income and weight and obesity prevalence” (Schmeiser, 2009, p. 1278). Excess adiposity develops over time, as calories in exceed calories out and the unexpended energy is stored, partly as glycogen and protein but mostly as fatty tissue (Schoeller, 2008), so past SES experiences are more important than current experiences. Analysis of the Alameda County Study (Baltrus, Everson-Rose, Lynch, Raghunathan, & Kaplan, 2007) separated the effect of SES background (measured in 1965) on later BMI level and BMI growth in a hierarchical model, and found significant effects of own education for all but older men, parental occupation and own education for younger women, and own household income for older men on BMI growth. Given that the subjects were ages 17 to 40 at baseline, however, it is possible that BMI growth was affected by their baseline BMI, which also affected education and income. Distributional analyses of obesity dynamics find that much of the growth in BMI is at the upper end of the distribution (Bleich et al., 2008; e.g., Cutler et al., 2003; Flegal, 2005; Flegal & Troiano, 2000; Komlos, Breitfelder, & Sunder, 2009) and numerous studies find that weight gain is correlated with starting weight (Brown, Williams, Ford, Ball, & Dobson, 2005; Gius, 2011; Gordon-Larsen et al., 2009; Ruel, Reither, Robert, & Lantz, 2010). Schmeiser took

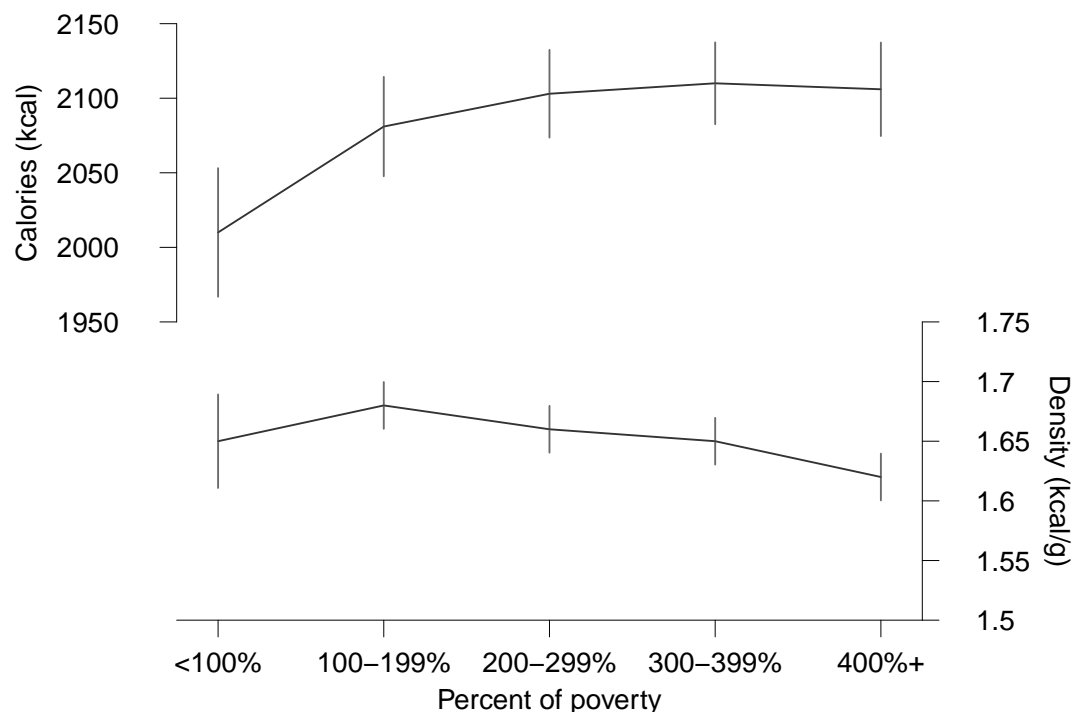
advantage of exogenous variation in state and federal Earned Income Tax Credit generosity, which theoretically induced changes in labor supply behavior, in a fixed effect instrumental variables analysis of low income adults in NLSY79. He found that an additional \$1000 of family income is associated with an additional .14 to .31 BMI units for women, with no effect for men in the sample. The effect of income was larger at the upper end of the BMI distribution, and resulted in an increased propensity to be obese. In contrast, Smith and colleagues (Smith, Stoddard, & Barnes, 2009) found, net of probability of unemployment, receipt of inheritance money is associated with weight loss. The latter finding was for men (in NLSY) at all income levels and relied on an exogenous but transitory income, while the former was restricted to low income adults and relied on long-term income effects. Thus, the causal evidence for income leading to excess adiposity within adults is limited.

There has been much discussion of what might be the mechanism connecting income-related factors to obesity. Why are the poor more likely to be obese? Price is likely one mechanism. Economic forces have contributed to the widespread availability and low cost of energy-dense foods (Cutler et al., 2003; Finkelstein & Strombotne, 2010). In the US and other industrialized countries in which fats and sugars have seen real price decreases while fish, fruits, and vegetables have increased in price (Cawley, 2006; Finkelstein & Strombotne, 2010), this means that a low cost diet often means an energy dense diet and, paradoxically, higher calorie intake (Drewnowski, 2007, 2009). Increases in obesity prevalence in the last 30 years have paralleled large increases in consumption of added sugars in foods and beverages (Marriott, Cole, & Lee, 2009; Tappy & Le, 2010). Time costs of both food preparation (Cutler et al., 2003; Finkelstein & Strombotne, 2010) and physical activity (Finkelstein & Strombotne, 2010; Lakdawalla & Philipson, 2009) are important as well. Preparing healthy meals, shopping for lower calorie options, or going for a walk or to the gym all require time. “It is also possible, and quite likely, that many rational, utility-maximizing individuals will engage in behaviors that are obesity promoting simply because—in today’s obesity-promoting environment—it is just too costly (in economic terms) to weigh less” (Finkelstein & Strombotne, p. 1522S).

If it is indeed costly in terms of time to eat and exercise well, then economic theory might predict that higher earners, with their higher opportunity cost of time (i.e. wages), would be less likely to engage in time intensive activities and more likely to select sedentariness and high calorie convenience foods (Smith et al., 2009). The fact that the opposite is the case—higher earners are more likely to engage in leisure time physical activity (Brownson, Boehmer, & Luke, 2005; Meltzer & Jena, 2010), spend more time eating (Hamrick, Andrews, Guthrie, Hopkins, & McClelland, 2011), and have better nutritional practices (Drewnowski, 2007; Kant & Graubard, 2007)—suggests that there may be another explanation, informational, economic, psychological, or cultural, for why the poor are more likely to be obese.

It may be that income dictates diet quality (i.e. energy density) rather than caloric intake. Higher earners may be selecting lower density convenience foods, such as an apple instead of a bag of chips. The poor tend to purchase low-cost, high-density food (Drewnowski, 2007, 2009). In addition to high levels of fats and/or sugars, such foods tend to be high in salt and other preservatives, extending storage life. Kant and Graubard's combined NHANES results for calories and energy density are shown in Figure 1.4. While the authors found significant trends over their categories of income for each, caloric intake and obesity prevalence (see Figure 1.1 on page 5) actually move in the opposite direction with changes in income category: The wealthy take in more calories but are less likely to be obese. Most of the increase for calories occurs moving from those below the federal poverty level (FPL) to those with incomes from one to two times poverty. Reported energy density decreases the farther the respondent is above the poverty line. Other research indicates that income is related to meat quality (i.e. fat content), purchasing of seafood, and selection and amount of fruits and vegetables (Drewnowski, 2007). On the other hand, smoking is clearly associated with less weight gain (e.g., Brown et al., 2005; Cawley, 2006; Smith et al., 2009) and, like obesity, smoking prevalence exhibits a negative SES gradient (e.g., Evans & Kutcher, 2011; Hajat, Kaufman, Rose, Siddiqi, & Thomas, 2010; Power et al., 2005; Senese, Almeida, Fath, Smith, & Loucks, 2009). As smoking declines among the poor, obesity may thus increase despite efforts to improve diet and exercise in poor communities.

Figure 1.4: Reported caloric intake and energy density (with 95% confidence intervals) from all NHANES data (through 2002), from Kant and Graubard (2007; Table 4)



While Americans spend less time preparing food than in the past, we also spend more time eating while doing something else, which decreases monitoring of intake (Wansink, 2004; Zick & Stevens, 2010). Zick and Stevens found wages made little difference in so-called secondary eating time. This would imply differential effects on caloric intake only if the meals not being monitored are higher in calories. Furthermore, the obese actually spend less time per day in primary and/or secondary eating, but have a higher proportion of all eating time that is undertaken while doing something else, and spend more time in secondary drinking (Hamrick et al., 2011). Evidence suggests the obese are more easily distracted from monitoring intake and “may be particularly susceptible to the environmental factors that spark overeating and that undermine their attempts at restraint” (Wansink, p. 459). These factors include those that structure the effort needed to initiate eating (e.g., how readily at hand are snack foods), external social cues regarding portion sizes and seconds, and the salience, variety, packaging and portion sizes of the food itself. Thus, if this predisposition

to poorer monitoring and distracted eating is present among some of the poor, who are more likely to purchase foods of high energy density, the result will be overeating of calorie-rich foods.

If weight status, at least developmentally, is ultimately all about calories in versus calories out, the higher intake by those with higher incomes, who are less likely to be obese, implies more energy expenditure by the wealthy. Indeed, as higher paying jobs, such as professional services, information, and finance, are generally sedentary, while lower paying jobs, such as construction, manufacturing, and janitorial positions, are considered to require light to moderate physical activity on the average (Church et al., 2011), it would appear the wealthy are more likely to pursue physical activity outside of work hours. Many have cited differential access to physical activity opportunities as a potential driver of obesity (Caprio et al., 2008; Cawley, 2006; Drewnowski, 2007; French, Story, & Jeffery, 2001; Gordon-Larsen, Nelson, Page, & Popkin, 2006; Popkin, 2009; Sallis & Glanz, 2006), although such studies usually suffer from selection bias issues (Cawley, 2006; Eid, Overman, Puga, & Turner, 2008). So-called built environment concerns about new developments lacking sidewalks and parks are further undermined by a lack of correlation between child physical activity and either availability of such features or safety (Cawley, 2006). Regardless of causation, it is fairly clear that lower income children and adults have less access to physical activity facilities that might discourage weight gain. On the other hand, as French and colleagues point out, “it is interesting that the ‘boom’ in health club memberships occurred during 1982–1990, a time when striking increases in weight gain in the population were observed” (p. 323) even among the wealthy (Cook & Daponte, 2008).

The large body of literature connecting income and other SES factors with adult weight is consistent with a larger literature on the effects of socioeconomic disadvantage and health outcomes. Kawachi, Adler, and Dow (2010) reviewed the issues, weaknesses, and results of research on material and symbolic (psychosocial) resources and health outcomes. They highlight three basic models, which focus on the effects of

1. absolute income, which should show diminishing returns above a certain threshold of

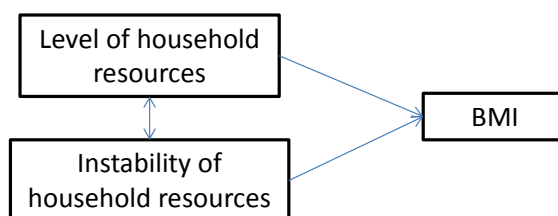
income

2. relative income, where health is a function of the income gap with a reference group, which in the relative deprivation variant includes people with higher incomes, and
3. relative rank, focusing on the stresses or resources that accrue to place in the social hierarchy.

The absolute income explanation is usually identified with the importance of material resources, while the relativist explanations are identified with psychosocial resources—but, as the authors point out, it would be difficult to hold material resources constant while changing psychosocial resources, or vice versa.

The relative stability in the official US poverty rate, which until 2010 had only been above 15% once since 1983, in 1993, and has not been below 11.3% since 1974 (US Bureau of the Census, 2011), hides considerable turnover (Jantti, 2009; Sandoval, Rank, & Hirschl, 2009). If poverty contributes to obesity, large portions of the population are at risk of falling into poverty and being exposed to the mechanisms connecting poverty to obesity. (Alternately, it may be that a higher poverty threshold is more predictive of obesity—that in terms of its effect on obesity, “poverty” should be defined as closer to 300% of the FPL.) Income volatility or bouncing in and out of (official) poverty may be as much if not more of a driver of obesity than constant exposure to poverty or one’s (permanent or average) level of income, as in Figure 1.5. A focus on instability appears to be a departure from prior models of the effects of SES on health and health behaviors.

Figure 1.5: Separate effects of level and instability of resources



In a cross-national analysis, Offer and colleagues sought to explain why English-speaking countries form a cluster of high obesity. Economic security (from Lars Osberg's Index of Economic Well-Being, comprised of security from unemployment, illness, single-parent poverty, and old age poverty) was "the strongest predictor of obesity prevalence" even when the US (a stark outlier) was eliminated and even net of fast food prices and economic inequality (Offer et al., 2010, p. 302). This analysis appears to span the absolute income, relative income, or relative rank models above, relying on potential explanations including absolute resources (financial insecurity), relative resources (inequality), and rank (subordinate status), although the overarching argument is a psychosocial one (stress). Relying on "evidence that overeating may be a personal response to chronic life stress" (p. 297), they support their hypothesis that it is the "unregulated market liberalism" of these countries that separates them (in terms of obesity) from other wealthy nations because of levels of life stress associated with the increased economic insecurity arising in such systems. Inequality (measured either as a composite of poverty intensity and the GINI coefficient or by the GINI coefficient alone) was largely a nonsignificant predictor of obesity prevalence when controlling for economic security, although it did become significant when food costs were included in the model. This might be seen as evidence against a relative income explanation. Breaking down their insecurity measure, they examined the independent effects of security against old-age poverty, security from single-parent poverty, and security from unemployment, where each index was a composite of the probability of falling into the appropriate category and the extent of relevant social protections, plus a "security from ill health" index that captures private medical expenses as a proportion of disposable income, proxying private medical cost risk. They find that the latter has the strongest effect: "The Osberg security index focuses on conditions of dependence, and expectations of social support. It is a powerful variable, but almost all the work is done by a single component, namely the uncertainty arising from having to pay for health care out of personal income" (Offer et al., 2010, p. 304). Economic insecurity is conducive to obesity due to psychological stress over material resources.

This is essentially the model underlying the analysis of Smith and colleagues (2009), who specifically investigated individual-level economic security in males net of income levels. They defined perceived economic security, “roughly speaking, as the risk of catastrophic income loss faced by an individual or household” (p. 1) and operationally as probability of job loss or poverty, number of large income declines year over year, and an income volatility measure. Insecurity—particularly the subjective experience of the probability of a loss and resulting (relative) famine—represents a stressor that may result in weight gain in adults, the argument essentially connecting economic circumstances to psychological and neuroendocrine systems connected to appetite, dietary choices, metabolism, and weight gain. The analysis found (in their instrumental variables models) a significant predictive relationship for three of the four insecurity measures (except probability of being under the poverty line): “(1) a 1 [percentage point] increase in the probability of becoming unemployed causes an increase in body weight of over half a pound, (2) for each additional year in which real income drops by at least 50%, body weight increases by 5 pounds, [and] (3) a decrease in  $R^2$  (a measure of income stability) by 0.1 units corresponds to an increase in body weight of 2.4 pounds” (p. 12). These investigations of income insecurity do address one of the unanswered questions laid out by Kawachi and colleagues (2010):

Distinguishing the effects of temporary income (income shocks) from the effects of permanent income. Income shocks may be associated with more harmful behaviors in the short term (e.g., cigarettes and booze become more affordable), whereas increases in permanent income may improve a person’s prospects for the future and lead to increased incentives to invest in their longevity (p. 65).

Financial stress may also be defined as being in debt or having other current financial difficulties. Research shows, for example, that being in credit card debt (a simple dichotomous question regarding current debt) is predictive of being overweight or obese for men and women, while women with trouble paying bills are more likely to at least overweight and more likely to be obese (interestingly, men with bill trouble were less likely to be overweight; Averett & Smith, 2012). There is thus some evidence that higher levels of economic insecurity during adulthood are associated with obesity.

The proposed causal effect of income instability is consistent with a broad literature relating stress to obesity (e.g., Caprio et al., 2008; Greenfield & Marks, 2009; Kivimaki et al., 2006; Kuo et al., 2008; Lee, 2011; Nguyen-Rodriguez, Unger, & Spruijt-Metz, 2009; O'Connor, Jones, Conner, McMillan, & Ferguson, 2008). Chronic stress chronically activates stress-related neurochemical pathways, enhancing the reward inherent in food (Huang et al., 2009). The concepts of “comfort food” and “self-medication” via food intake refer to using food as a coping strategy (e.g., Nguyen-Rodriguez et al., 2009; Offer et al., 2010; Olson, Bove, & Miller, 2007; Smith et al., 2009). The relationship between stress and weight gain appears to be moderated by tendency towards such emotional eating (O'Connor et al., 2008), perhaps in the perceived absence of other stress reduction techniques. Indeed, research shows that it is people already in the upper part of the BMI distribution who tend to gain weight with stress, while the thin tend to lose weight (Kivimaki et al., 2006), and animal models show that stress induces weight gain only in the presence of high calorie foods (Kuo et al., 2008). Note, again, that obesity itself may directly or indirectly cause stress, which may contribute to further overeating or under-exercising.

Numerous authors have written about the evolutionary predisposition of humans to eat substantially when food is available, storing energy for times of famine (e.g., Ambinder, 2010; Pollan, 2006; Popkin, 2009; Ulijaszek, 2007, 2008). “The reason humans and other animals evolved the ability to store body fat is presumably because it was necessary to survive periodic food shortages. The evidence for this is surprisingly strong. It has been demonstrated again and again, for instance, that animals in natural environments face very real periodic starvation risk, and that such risk is a strong predictor of fattening behavior” (Smith et al., 2009, p. 1). The effect in humans is not yet clear. While many but not all cross-sectional studies of food insecurity or insufficiency find a relationship with obesity (Dinour, Bergen, & Yeh, 2007; Larson & Story, 2011), a two-year longitudinal study of food insecurity (using the USDA definition) on adiposity found no effect of baseline food insecurity on weight gain (Jones & Frongillo, 2007), which may be due to weakness of this retrospective, self-report measure. A relationship between food availability and food intake has been found, however, in response to the monthly cycle of food stamps benefit

distributions (as reviewed in Dinour et al., 2007; Rosenkranz & Dzewaltowski, 2008; Smith et al., 2009; Zagorsky & Smith, 2009). Lower income families may frequently face diet changes and constriction “in the week or two before the next check arrives” (Engler-Stringer, 2010, p. 222). “Studies indicate that bingeing often follows periods of food deprivation... and that binge eating is positively associated with obesity because it can alter metabolism in ways that promote fat accumulation” (Zagorsky & Smith, p. 248). Thus, whether due to a propensity for comfort seeking in food (emotional eating) or as an evolutionary response to perceived risk of famine, humans may respond to economic stress by overeating.

There appears to thus be support for economic models of two income effects (Smith et al., 2009), as in Figure 1.5:

1. The usual income effect of the time cost of food and exercise: An increase in income results in increased weight
2. A resource stress effect: An increase in the subjective probability of income (and thus nutrition) loss results in increased weight.

Smith and colleagues argue that this instability/risk effect may explain apparent self-control or inconsistent preferences problems frequently referenced in economic models of obesity (e.g., Brownell et al., 2009; Conley & Glauber, 2007; Cutler et al., 2003; Finkelstein & Strombotne, 2010; Oswald & Powdthavee, 2007), because current decisions are actually rational in the face of a belief in a foreshortened future.

#### ***1.4 Family of origin material resources and child weight***

If recent economic stress can cause obesity in an adult, what about earlier stress? Numerous cross-sectional studies have related family poverty or wealth to child obesity (Emerson, 2009; Ogden et al., 2010; Phipps, Burton, Osberg, & Lethbridge, 2006; Sawaya, Martins, Grillo, & Florencio 2004; Singh, Siahpush, & Kogan, 2010; Storey et al., 2003; Strauss & Pollack, 2001). For example, by quartile of income-to-poverty line ratio, the obesity rate among young children in America declines from 17% among the lowest quartile to 16%,

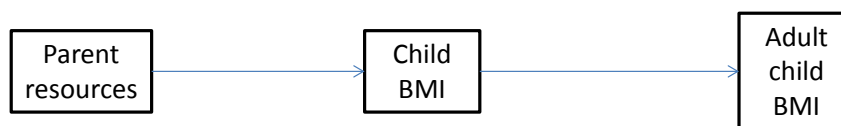
14%, and finally 11% among the richest quartile, and “obese children in the poorer families are heavier than obese children in wealthier families” (Anderson, Butcher, & Schanzenbach, 2007, p. 10).

Cohort studies often find a connection between childhood SES and later obesity (Baltrus et al., 2007; Evans & Kutcher, 2011; Laitinen, Power, & Jarvelin, 2001; Langenberg, Hardy, Kuh, Brunner, & Wadsworth, 2003; Lee et al., 2009; Power et al., 2005; Scharoun-Lee et al., 2011; Senese et al., 2009; Wells, Evans, Beavis, & Ong, 2010), although less consistent effects have been found in recent years (Senese et al., 2009), which may be related to evidence of a decline in the cross-sectional SES gradient in obesity (Kant & Graubard, 2007; Senese et al., 2009). NHANES data reveal, however, that the income gradient among children 2-11 has become more, not less, apparent in recent surveys (Anderson et al., 2007). Attention to the developmental impacts of economic stress on later body mass are consistent with a broader literature connecting economic background and later health (e.g., Hayward & Gorman, 2004; Huang et al., 2009; Magnuson & Votruba-Drzal, 2009; Seeman, Epel, Gruenewald, Karlamangla, & McEwen, 2010).

Many studies of a relationship between childhood SES and later adiposity concentrate on socioeconomic position (SEP), usually defined based on the father’s occupation (sometimes incorporating education, maternal characteristics, or other non-income conditions). For example, using father’s occupation at birth (in Finland in 1966), Laitinen and colleagues (2001) showed that BMI at birth was higher with higher SEP (reflecting lower likelihood of low birth weight), but this had reversed by age 1, and by age 31, BMI as well as BMI-defined obesity and waist-to-hip ratio were each inversely related to SEP at birth. Analysis of the Alameda County Study (Baltrus et al., 2007) showed that women aged 17-30 at baseline (in 1965) with low childhood SEP (based on reported paternal occupation or education) exhibited higher weight gain (an extra .13 kg/year) over the ensuing 35 years than women from high SEP. No effect was found for older women or men. Overall, the effect of childhood SEP on later adiposity appears to be stronger for women than for men, at least in studies since 1998 (Power et al., 2005; Senese et al., 2009).

Much of the work on SEP has been in Europe and elsewhere, and relatively few studies of the effects of childhood family circumstances have moved beyond “crude measures of socioeconomic status... typically measured only once at some nonspecific point before age 18” (Ziol-Guest, Duncan, & Kalil, 2009, p. 528). A study of youth interviewed at ages 9, 13, and 17 in upstate New York found that proportion of time spent in poverty up to age 9 (Wells et al., 2010) or income at age 9 (Evans & Kutcher, 2011) were predictive of BMI growth or BMI at age 17. Lee and colleagues (Lee et al., 2009) used Add Health data to examine longitudinal associations between poverty and young adult obesity. Their measure of poverty (any public assistance receipt during childhood or Wave I income below FPL) was associated in bivariate models with obesity at Wave III and with obesity changes from Wave II to Wave III in females but not males. The effect on obesity changes for females was eliminated when parent education, family structure, neighborhood poverty, and a proxy for perceptions of income adequacy at baseline (trouble paying bills) were added to the model. A study of poor children in the PSID (Ziol-Guest et al., 2009) found that higher parental income within low income levels during the prenatal and birth years was predictive of lower risk of adult obesity and lower adult BMI, but not family income from ages 1 to 5 or 6 to 15. In contrast, Akee and colleagues (Akee, Simeonova, Copeland, Angold, & Costello, 2013) found that unconditional cash transfers (from tribal casino royalties) resulted in higher BMI among less advantaged Native American youth. This seemingly anachronistic finding is similar to the causal effect of exogenous income among lower income women noted above (Schmeiser, 2009). Thus, there appears to be mixed evidence for the relations in Figure 1.6, the relations that underlie concerns about obesity and poor children.

Figure 1.6: Within person effects of childhood resources on obesity



Childhood SES tends to remain a significant predictor of later adiposity even when later SES is added (Langenberg et al., 2003; Power et al., 2005; Senese et al., 2009). An analysis of Add Health specifically examined indicators of both adolescent (family) and young adult (own) SES using Latent Class Analysis (LCA) to identify five groups of distinct SES progression. The group with low SES in both adolescence and young adulthood (Persistent Disadvantage) had the highest risk of obesity for white and Hispanic women, while the group with low early SES and fairly high SES in young adulthood (largely through early entry into manual labor, resulting in high income and high rates of home ownership, thus inspiring the name Disadvantaged Fast Starters) had the highest risk for men and black women and second highest for white women (Scharoun-Lee et al., 2011). That is, groups that experienced relative poverty in adolescence but later had higher incomes had among the highest risks of obesity, but all groups not in the highest SES category in adolescence and early adulthood had elevated obesity risk.

While research to establish the relationships in Figure 1.6 is ongoing, much writing has theorized or established potential mechanisms for the developmental effects of resource dynamics on adult child BMI. Family economic conditions may result in child adiposity via preference for packaged food or food away from home due to maternal working hours or parental monitoring of diet and physical activity. Maternal full-time employment, versus non-full-time, is associated with childhood obesity (Liu, Hsiao, Matsumoto, & Chou, 2009). This may explain why two of the groups in the Add Health LCA study above with low to medium SES in adolescence and higher SES in adulthood, the Material Advantage group (who usually had two working parents in adolescence) and the Educational Advantage group (who usually lived with a single mother), had significantly higher risk of obesity than the reference Highest Overall Advantage group among men and white women (Scharoun-Lee et al., 2011). In a longitudinal study in Australia, maternal working hours had non-linear effects on child weight status that were small but statistically significant: Children of mothers who worked part time were less likely to be overweight in the future, in part because they watched less television, than children of mothers who worked full time or not at all, net of maternal weight, household income, and other factors (Brown, Broom, Nicholson,

& Bittman, 2010). Similarly, maternal nonstandard work schedules have been found to be associated with obesity at ages 13 and 14 (Miller & Han, 2008). Lee and colleagues found evidence for the effect of parental monitoring on later obesity (Lee et al., 2009). This may further explain why Powell (2009) did not find a significant effect of changes in parental or youth income on adolescent BMI net of family structure, maternal full-time or part-time work, and various food environment indicators, including fast food prices.

The connection between SES and obesity may be due to eating patterns chosen or biological changes as a result of poverty. The biomedical literature discusses the “programming” effects of over- or under-nutrition in utero and in the early years of childhood, which are thought to operate via epigenetic modifications, metabolic or endocrine changes, insulin resistance or glucose tolerance, and so on (Fall, 2011; Lawlor & Chaturvedi, 2006; Li, Goran, Kaur, Nollen, & Ahluwalia, 2007; Martin-Gronert & Ozanne, 2010; Muhlhausler & Ong, 2011; Tarry-Adkins & Ozanne, 2011). Studies have shown, for example, that adults exposed in utero or in early childhood to famine conditions are more likely to be obese or be diagnosed with the closely related metabolic syndrome (at least three of elevated triglyceride levels, elevated glucose levels, lower HDL cholesterol levels, high waist circumference, or high blood pressure; Fall, 2011; Olson et al., 2007; Yanping et al., 2011). Li and colleagues (Li et al., 2007) separated children (of mothers in the NLSY79) into trajectories of normal weight, early onset overweight, and late onset overweight (via latent growth mixture modeling) and found that these groups differed significantly on net family income the year the child was born, but these differences disappeared net of maternal pre-pregnancy BMI, maternal weight gain during pregnancy, birth weight, and breastfeeding extent. Olson and colleagues (Olson et al., 2007) have speculated that the oft-found relationship between food insecurity and obesity is due to “the experience of poverty-associated food insecurity in early childhood and the eating practices and attitudes resulting from food insecurity,” including emotional eating (p. 199). Their qualitative data revealed ways in which childhood food deprivation might affect future diet choices: Some of their mothers developed an emotional attachment to having food available for their current families, resulting in stocking up; for some, “the early experiences resulted in the removing of economical and nutritious foods

from women's own and their families' diets" (p. 203); and for some, the emotion attached to enjoying food when they could developed into current emotional eating habits. These effects can result in obesity via the Costco effect—the effect that food salience and large package size have on consumption (French et al., 2001; Wansink, 2004)—of stocking up, rejecting the economical but still somewhat nutritious choices of our parents in favor of today's energy-dense cheap foods, and turning to food for comfort. Furthermore, the poor mothers in Olson and colleagues' investigation poignantly illustrated the binge-promoting effects of periodic bounty:

Immense excitement and pleasure sometimes accompanied the influx of food into these food-insecure households following the arrival of food stamps, a paycheck, or a gift of food. Both adults and children appeared to increase their intake of food once it became available again, increasing their consumption of preferred foods in particular, which had been especially missed. . . . These findings indicate that periods of overeating by both children and adults sometimes accompanied the influx of food into food-insecure households (p. 204).

“Even if many people exit poverty each year, they do not exit very far from the poverty line and are at substantial risk of reentry” (Jantti, 2009, p. 196). This may explain why little decline in obesity is seen until more than 300% of the poverty line in Figure 1.1 (page 5): Large portions of those between 100% and 300% may have been poor in the past—and at risk of falling back into poverty—and subject to the mechanisms connecting poverty to obesity. The stress of low income may arise not from predictably low access to resources but from unpredictability, from having to worry a little more than last year or last month about feeding one's family. A fortunate week or month may promote stocking up or gorging on favored foods (Olson et al., 2007). A downward income shock might impinge upon sleep duration, which has been associated with appetite dysregulation (Knutson & van Cauter, 2008), or inspire trying a cheap, high-sugar and/or -fat convenience food that becomes a viable solution to the problem of how to feed yourself or your family on a tight budget (Jastran, Bisogni, Sobal, Blake, & Devine, 2009). Indeed, in reference to the effects of poverty and income on obesity, there may be two basic kinds of formerly

poor people: Those who adapt diets and/or energy expenditure patterns associated with poverty and maintain them even with increased income (and in the case of diet, may even purchase higher quantities of energy-dense foods, further increasing their weight), and those who respond more to whatever mechanisms connect higher income with lower weight (e.g., purchasing more nutrition-dense foods, leisure time exercise, etc.). A mixture of these two subpopulations, for whom we would expect adiposity to move in opposite directions, would explain the relatively flat gradient in obesity from below poverty up to 300% of poverty in Figure 1.1.

Olson and colleagues connect their results to the concept of materialism. Allen and Wilson (2005) argued that this personality trait, usually discussed in regards to less necessary goods and services, should apply to food as well—that there exists a materialist subset of society who are “likely navigate their physical and socio-cultural environments through food” (p. 315). Their research showed that materialism is related to endorsing food security as a life goal and to reported childhood experience of food insecurity. In turn, materialism was related to having more food stored in the home and to obesity itself. Their lab experiment used a previously validated manipulation of survival security and found that experimental group members who felt insecure after the manipulation reported higher materialism. High materialism members of the experimental group more strongly endorsed food security as a goal and emotional eating.

Other potential mechanisms connecting low SES during childhood to later obesity include aspects of the home environment and parenting practices, the immediate social and community environment, and broader structural and economic forces that have differential effects on the poor. Investigations into health disparities often cite class-biased health care during formative years as a mechanism connecting low childhood SES and later health outcomes (Poulton & Caspi, 2005). Less advantaged school districts are more likely to turn to snack and sugar-sweetened beverage sales to augment limited school budgets (Anderson & Butcher, 2006; French et al., 2001; Johnson, Bruemmer, Lund, Evens, & Mar, 2009). The effects of food prices and the relative cost of energy-dense foods were discussed above.

These forces may interact with the time poverty faced by single parents and/or those trying to balance child care and multiple jobs (and other tasks). Food availability, marketing exposure, and social pressures may interact with parenting practices such as using food as a reward, which is more common among less educated and lower income mothers (Rosenkranz & Dziewaltowski, 2008). Similarly, food practices are a product of multiple forces including culture, availability, and food preparation skills (Engler-Stringer, 2010; Rosenkranz & Dziewaltowski, 2008).

Note that deprivation may affect physical activity. This may be via neighborhood effects (Caprio et al., 2008; Chang, Hillier, & Mehta, 2009; Drewnowski, 2007; French et al., 2001; Gibson, 2011; Gordon-Larsen et al., 2006; Sallis & Glanz, 2006), although there is some evidence that the effects of density, walkable neighborhoods, transit access, and mixed land use are protective only among advantaged groups (Lovasi, Neckerman, Quinn, Weiss, & Rundle, 2009) and that apparent neighborhood effects may be due to selection bias (Cawley, 2006; Eid et al., 2008; Ewing, Brownson, & Berrigan, 2006). It may also be that economic stress diminishes the likelihood of choosing to exercise, and that choosing not to pursue physical activity becomes a habit. Parents may model this behavior for their children or be unwilling to promote and monitor physical activity.

This effect on likelihood of pursuing exercise may combine with such potential mechanisms as the well-established “link between the experience of food deprivation and overeating when food becomes available” (Olson et al., 2007, p. 205), diet choices made in the face of constraints on time and other resources and maintained even with greater household income, and materialism and a tendency to hoard food more than someone who has not experienced deprivation. Finally, as noted above, there are proposed biological mechanisms to explain why the human body might respond to stress by overeating and storing fat as “a form of precautionary savings” (Smith et al., 2009, p. 3) against future risk. Combined with the reward inherent in eating and the ready availability of highly palatable energy-dense foods, this behavioral response may not be readily extinguished with the passing of risk-free time, and parents may pass this tendency on to their children even in the absence of further

actual stress.

These proposed mechanisms may explain the evidence linking economic deprivation and instability within adulthood to adult adiposity and childhood and parental experiences of deprivation and instability to a child's later body mass. A poor adult is more likely to become obese, and more likely to produce an obese child. While persistence of obesity into adulthood is high, I know of no evidence supporting the idea that this persistence is higher among low income or SES children.

The proposed relationships between economic conditions and the development of adiposity might imply that poverty or instability would increase the correlation between parent and child BMI. Contemporaneous NHANES data, however, reveal no differences in elasticity of child's BMI with respect to mother's BMI for more or less advantaged groups, and only marginal evidence of a higher elasticity of caloric intake between mother and child among the poor (Anderson et al., 2007). In contrast Classen (2010) concludes that "there exists some evidence of stronger intergenerational BMI persistence in higher SES families" (p. 36) using NLSY data.

As a society, we are concerned about how poor economic conditions may affect child development. Frequent highlighting of a relationship between income and obesity reflect this concern. An article in an issue of *The Future of Children* dedicated to childhood obesity summarized the existing literature as "low-income children are at excess risk of obesity" (Kumanyika & Grier, 2006, p. 190). Similar reviews have appeared in popular magazines such as *The Atlantic* (Ambinder, 2010) and *The Economist* (Howard, 2012). In 2011, the Centers for Disease Control and Prevention (CDC) announced the \$25 million Childhood Obesity Demonstration Project aimed at low-income children (Walker, 2011). At the beginning of 2013, the UK health minister claimed that it is now easy to pick out which children in the schoolyard are poor "because their diet...means they tend to be fat." In response to what was seen as incriminating poor parents, a British child poverty advocacy group claimed that "piles and piles of evidence...make it absolutely clear that

the real reason why our obesity problem is going to get bigger in the years ahead is because our child poverty problem is going to get much bigger'” (Booth, 2013).

When reviews of the obesity literature, public officials, and newspaper and magazine stories alike cite a relationship between poverty or lower socioeconomic status and obesity, we can say such a link has become conventional wisdom. That wisdom holds that there is an income gradient to excess body fat, and is consistent with evidence of economic disparities in other health behaviors and outcomes. The reality, of course, is much more complex. This complexity may reflect timing and extent of poverty, familial and cultural traditions around coping with stressful situations and food, genetic factors, access to social capital and services, and so on.

Beyond whether childhood SES affects adult adiposity, a key question revolves around how much exposure and when is necessary. The answers to these questions will offer much insight as to potential mechanisms. Many discussions of the SES-obesity relationship, or the broader relationship between SES and health, do not move beyond the simple consideration of any experience of deprivation, no matter when (although this is still inherently developmental in that prior experiences are included). The developmental health disparities literature often relies on two main models of how childhood SES might affect future health.

The first is a *timing* or developmental period model, which focuses on the effects of SES-related factors during sensitive or critical periods during the child's development (Cohen, Janicki-Deverts, Chen, & Matthews, 2010). Similarly, the developmental literature highlights the importance of the timing of childhood experiences and conditions, including critical periods for interventions (e.g., Miller, Maguire, & Macdonald, 2011; Morris, Duncan, & Clark-Kauffman, 2005) as well as the timing of insults such as child maltreatment (e.g., Jaffee & Maikovich-Fong, 2011). In this model, the effects of exposures during these critical periods are thought to irreversible, and thus independent of the effects of later, particularly adult, SES. Conroy and colleagues also call such a conceptualization the “latency” model, “highlighting that the health effects of these critical periods may not recognized until much

later” (Conroy, Sandel, & Zuckerman, 2010, p. 155). This implies a model in which any experience of low income during the critical period—no matter how low or for how long—creates the mechanisms that result in later adiposity. The fetal origins or fetal programming hypothesis (Muhlhausler & Ong, 2011) is a variant of the critical periods model, focusing on the prenatal through immediate post-partum periods. The PSID study comparing the effects of family income during the prenatal and birth years, ages 1 to 5, and ages 6 to 15 (Ziol-Guest et al., 2009), is an example of the developmental timing approach which found support for the fetal programming hypothesis. By not controlling for the mother’s prior experiences, however, this conclusion may have been an artifact of correlation between the mother’s prior experience and that in the prenatal and birth years. If so, the effects may have been less physiological than psychological—not so much about epigenetic programming and endocrine development itself but more about the mother’s nutrition and exercise habits developed in reaction to economic experiences just before the child’s birth.

In contrast, another main approach is to ignore timing and focus on the duration and extent, or dosage, of deprivation. This conceptualization is called the *accumulation* (Cohen et al., 2010) or cumulative effects model (Conroy et al., 2010). This model is also known as the “pathways” model due to the cumulative nature of multiple exposures in affecting health, often creating a correlated if not causal chain of events, as when maternal smoking results in low birth weight results in neurochemical changes that promote affinity for sugary foods (in the context of high availability). The unifying concept of allostatic load, which relates chronicity of stress to dysregulation among various physiological systems, is another manifestation of hypotheses focused on the cumulative effects of exposures on later health functioning (Seeman et al., 2010). The work by Wells and colleagues (Wells et al., 2010) tested the accumulation model by including a variable that measured the proportion of time (in 6-month intervals) between birth and age 9 that the subject lived in poverty. Analyses such as that by Poulton and colleagues (Poulton et al., 2002)) that average an SES measure over multiple childhood observations might be considered to fit with the pathways model, as this variable should give a lower value (depending on scaling) to subjects with more and/or lower SES observations over time. Their analysis of the Dunedin Multidisciplinary

Health and Development Study in New Zealand, for example, assessed SEP (ranging from 6 for unskilled laborers to 1 for professionals) of their cohort every other year from ages 3 through age 15 and averaged the results, so a child with 6 observations in the middle of the SEP range and 1 in the unskilled category would score lower (less disadvantaged SEP) than a child with 3 observations in the unskilled category and 4 in the middle, thus describing more exposure to the consequences of low SEP. Of course, outside of parental separation, such variation among the 6 possible SEP categories over the life span seems somewhat limited. Positive research findings using such models imply that more dosage (low SES) during childhood results in higher adult adiposity, while follow-up investigation might uncover a threshold effect.

Seemingly related lines of investigation involve cataloguing the effects of so-called cumulative risk exposure (Evans & Kim, 2010), cumulative social risk (e.g., Suglia et al., 2012) or adverse childhood experiences (e.g., Anda & Brown, 2010; Nurius, Logan-Greene, & Green, in press). While such efforts reference the accumulated effects of exposures explicitly or implicitly related to SES that likely occurred over different time points, most investigations ascribing to such conceptualizations actually do not include measures of chronicity (an exception being Wells et al., 2010). That is, while such research enlightens the pathways by which SES might influence alter functioning, they do so by crude dichotomous measures of whether a defined set of stressors (such as domestic violence, maternal depression, or food insecurity) ever occurred during childhood, thus lumping together single incidents that might have all occurred in a single year with multiple incidents over multiple years.

A third, less common model of childhood SES dynamics and future health functioning examines the impact of *changes* in SES. Analyses here tend to focus on two time points and test the upward mobility hypothesis—changing from low to high SES results in improved health outcomes—and the downward mobility hypothesis—changing from high to low SES results in poor health outcomes (Cohen et al., 2010). Such analyses appear to most commonly compare childhood SES with young adult SES (e.g., Langenberg et al., 2003; Poulton et al., 2002). A finding of higher BMI for people who experienced down-

ward mobility than those who remained at low SES, for example, implies a very different set of mechanisms—relating to change in economic circumstances—than finding no such difference.

These three models of the nature and timing of SES dynamics during childhood in affecting adult health are likely complementary rather than competing theories (Conroy et al., 2010) that ask different questions and explore different potential mechanisms, and thus support different modifiable risk factors. The upward versus downward mobility hypotheses, for example, attempt to shed light on whether later conditions might be protective or whether earlier conditions tend to dominate the production of health. Testing a measure of SES during the pre- and perinatal year will assess the effect of the mother’s diet, smoking and alcohol use, exposure to pollutants, and other stressors, while a measure of SES during adolescence will test potential mechanisms such as direct exposures to violence, fast food, and parental modeling of health behaviors. Comparing the effects of one year of childhood poverty versus three or five allows for testing of mechanisms relying on long-term exposure versus those that might operate with any exposure. “It is possible that there are cumulative effects of early SES exposure across childhood and adolescence (accumulation model), but that exposures at specific developmental periods are more impactful than those at others (timing model)... The upward mobility model may be viewed as a special case of accumulation with later rises in SES averaged together with initial low SES to result in a smaller dose of socioeconomic disadvantage relative to that experienced by those with consistently low SES” (Cohen et al., 2010, p. 50).

Many of the exposures thought to link SES to health outcomes, from relationship and housing instability to inconsistent parenting to poor household maintenance to neighborhood crime, inherently involve change and subsequent psychological and physiological stress. The allostatic load conceptualization, for example, embodies how extended and repeated stress responses harm physiological systems. “The premise is that while relatively short-term fluctuations in levels of physiologic activity are necessary for the body to respond successfully to stimuli (e.g., the fight or flight response in the face of various types of danger),

excessive fluctuations either in terms of the extent, duration or frequency (e.g., responding to perceived ‘danger’ everywhere) can result in wear and tear on the body’s regulatory systems” (Seeman et al., 2010, p. 226). Yet most studies of SES-related factors and health outcomes have focused on states rather than changes in SES. Even the upward/downward mobility models compare the state of SES at time one versus time two. “Less discussed (or tested) is the possibility that multiple changes in SES (whether upward or downward), particularly if they are drastic differences, throughout the life course may negatively impact future health outcomes by creating unstable and unpredictable environments” (Cohen et al., 2010, pp. 47–48). One can imagine that a childhood at a constant level of poverty might be relatively less stressful and have fewer negative long-term effects than one characterized by wide fluctuations in the supply of nutrition, housing quality, attentive and authoritative parenting, etc. Research findings supporting the negative effects of childhood SES status might actually be measuring the effects of attendant instability. We know, for example, that lower income families experience higher volatility of income over time (Gundersen & Ziliak, 2003). Differentiating between the two will shed light on potential mechanisms and interventions. If biases in health care availability and provision, or class-based differences in parental modeling and monitoring of diet and exercise, are the main drivers connecting childhood economic resources to adult adiposity, then we would expect a measure of family income or poverty, particularly one measuring a large portion of childhood, to be a significant predictor of adult BMI but not a measure of instability of income or poverty. In contrast, if both a poverty indicator (status) and a measure of volatility of income (instability) are significant predictors, this would support mechanisms related to family stress.

To summarize, there appear to be five models of measuring when and how SES in childhood is related to adult BMI (or other health outcomes). The most frequently used model is a crude measure of whether the adult was ever poor during childhood (or, alternately, average income or SEP, although it could be argued these weakly measure intensity of exposure over childhood). This model, of course, tells us little about the dynamics of SES exposures other than to establish time precedence. The next most frequently used method is to divide childhood into different developmental periods (timing), followed by measuring

the intensity and duration of exposures (accumulation). The least used prior model appears to be to measure changes in poverty or SES, most often used with only two time points and dichotomized SES to compare four groups (upwardly mobile, downwardly mobile, constantly low, and constantly high). The fifth model, hinted at by Cohen and colleagues (2010), is a hybrid of the timing and accumulation models, testing the effects of intensity of exposure within different developmental periods. Hypothetically, each of these models could be used with economic instability rather than status, as summarized in Table 1.1. These models allow exploration of the volatility of resources during childhood, which may affect future obesity via more of a latency model (e.g., via pre- or perinatal exposures or via setting of eating and exercise habits in adolescence), more of an accumulation model (e.g., where repeated stress promotes neurochemical changes that promote excess intake), or a model in which excessive instability and unpredictability during certain periods has a stronger effect.

### ***1.5 The case for studying the effect of resource dynamics in childhood on later adult adiposity***

To summarize, the above discussion implies future parents are more likely to become obese if they face economic insecurity or if diet and exercise patterns adopted during times of poverty are maintained even if income increases. These reactions to deprivation and insecurity may be passed on to the children, particularly if insecurity is experienced while the children grow to adulthood. Thus, the intra-personal mechanisms described above may come to affect children alongside the direct effects of childhood deprivation. There are numerous potential mechanisms to explain why living at risk of poverty might contribute to obesity. The effect of SES on adiposity bears more complex investigation.

- The income–BMI relationship is curvilinear. The probability of being obese among those just above the FPL is likely no different than that of those below poverty. The SES–obesity relationship appears to vary by gender and ethnicity.
- Viewing poverty or income instability as a form of stress fits with a broad literature connecting stress to obesity at the individual level, which may operate through

- metabolic changes
  - emotional eating
  - a desire to stock up on food when possible
  - and/or bingeing on favored foods when available.
- 
- The effects of these mechanisms are magnified by exposure to energy-dense foods.
  - Poorer groups are more likely to live in environments characterized by relative over-exposure to energy-dense foods and with less access to physical activities.
  - Evidence relates childhood SES to later obesity, but much of this research has ignored the timing of the poverty or economic resources. The child development literature highlights the importance of this timing.
  - Preliminary evidence links economic instability, net of level of resources, to obesity.

There appear to be ample explanations for why income instability or bouncing in and out of poverty during childhood—or the parent’s experience before the birth—might cause future obesity. I have not been able to locate many quantitative studies that examine the effects on later adiposity of dynamics over time of parental resources. This project thus appears to be an important addition to the literature on developmental health effects and obesity risk due to childhood income dynamics. What follows are three sets of analyses and discussions that combine the above literature on SES and obesity and models of how SES affects health to examine various aspects of childhood income dynamics as risk factors for later BMI. In Part 1 (Chapter 3), I address poverty in childhood, addressing both the threshold effects noted above as well as the dynamics of childhood experiences. In Part 2 (Chapter 4), I step back to examine income in a more flexible fashion, exploring differential effects of more income at various points in the income spectrum. Finally, in Part 3 (Chapter 5), I address childhood and pre-birth income instability and adult BMI.

Table 1.1: Summary of models examining childhood SES dynamics and adult health, with extension to SES instability, showing examples of constructs measured

Model	SES status indicator	SES instability indicator
Dichotomous	Ever in poverty	Any large income decrease
	Average income	More than 2 transitions into poverty
	Average SEP	
Timing	Average income in different developmental periods	Any large income decreases in period
	Ever in poverty during different periods	Any transition into poverty during period
Accumulation	Years spent in poverty	Number of large income decreases
	Total poverty gap (sum of yearly amounts family income was under poverty level)	Number of poverty transitions
		Variance of income
Change	SES in early/middle childhood vs. SES in adolescence	Change in poverty transitions from first half of childhood to second half
	Income trend over 3 time points/periods	Income variance trend over 2 or 3 periods
Hybrid model	Proportion of developmental period spent in poverty	Large income decreases in period
	Poverty gap in period	Poverty transitions in period Income variance in period

## Chapter 2

**OVERALL METHODOLOGY**

In this section, I will lay out the general methodology for the entire project, including data acquisition, selection of covariates, and other transcendent methodological issues. More specific methodology notes appear in the relevant chapters.

**2.1 *Data and measurement issues***

The National Longitudinal Survey of Youth—1979 follows a representative cohort aged 14–22 in 1979 annually through 1994 and biennially to date. This “main youth” survey is the source of data about mothers and household economic conditions. The NLSY79 Children and Young Adults (CYA) study, started 1986 (biennial since 1988), follows children of mothers in NLSY79, allowing for linking mothers and offspring, many of whom are now adults. The NLSY is commonly used to examine human capital, income, and related phenomena. As it also captured height and weight of both mothers and children in most survey waves, these phenomena can be related to obesity-related outcomes. For example, Classen (2010) used the linked studies to identify 2560 mothers of 4748 children for whom data were available for ages 16 to 24. Another study used the 2000 wave to find 6417 children born to 2934 mothers to analyze maternal work status and child BMI (Liu, Hsiao, Matsumoto, & Chou, 2009). Major tasks of data processing included renaming variables from the NLSY-supplied numeric codes, converting time-varying data from both the mother (family income, family size, etc.) and the child (height and weight, whether he or she was living with the mother at least part time during the year, and pregnancy status) from calendar year (e.g., in 2004) to year relative to the child’s birth year (e.g., the year the child turned 16), and calculating variables.

After extensive cleaning of height and weight data up to and including the 2010 wave of the CYA, there are 6361 adults at least 19 years of age with a good BMI. This cleaning involved first dealing with NLSY coding, in which height is sometimes entered as inches and sometimes as feet and inches and both height and weight are sometimes entered with two implied decimal places but no decimal point. Unusual growth or shrinkage over time (specifically, height changes of 6 inches or more) were flagged and investigated, which often uncovered data entry errors, some of which appeared to be due to missing inches (e.g., the young adult went from 5-6 to 5-0 and back to 5-6). To maximize availability of adult BMIs, missing heights were filled in using the average of adult heights, with individuals with resulting average heights and individual observed heights more than 4 inches different flagged and checked. Finally, all calculated BMIs greater than 50 plus two less than 15 were checked by hand, in keeping with guidelines from de Gonzalez and colleagues (2010). Unless an error was very obvious, such as being 4-11 in several observations but 5-11 in one year, all of this flagging and checking resulted in implicated values being changed to missing. All remaining BMIs above 50 reflect cases with long histories of being severely obese and/or trajectories of weight gain that made a high BMI quite plausible. Similarly, one of the two cases with a BMI below 15 showed weight loss that paralleled her sister, implying they may have been losing weight together and the low weight reported was valid. (The other case below 15 had widely varying heights that made judging which was correct impossible.) The BMI for women who reported being pregnant were also set to missing, as BMI is not valid in such cases.

Among the variables created by the managers of the study are a yearly Total Net Family Income variable, combining income across all sources from all co-resident family members related by birth or marriage (but excluding unmarried partners), and a Poverty Status indicator that compares the previous year's family income to federal poverty thresholds based on family size and location. The NLSY Total Net Family Income variable includes income from all sources, including unemployment benefits, child support and alimony, rental subsidies, food stamps, EITC, and other transfers. All of these can be described as non-market income sources that help smooth vagaries of earned income. This means the NLSY measure

assesses a form of adjusted income poverty (it does not adjust for taxes paid) and thus categorizes fewer families as poor than the poverty measure produced by the US Census Bureau. The proposed mechanisms connecting income instability to adiposity depend essentially on perceived stress which may or may not be related to nutritional instability. Future research can compare the effects of total income instability versus instability of market income to test whether the latter is likely to be experienced by the family as anxiety. The social insurance benefits of programs act in part through smoothing of income and consumption. The Food Stamp Program (now called Supplemental Nutrition Assistance Program), for example, has important dampening effects on volatility of income and food consumption, particularly among those most at risk of needing food stamps, for whom food makes up a higher proportion of the household budget (Gundersen & Ziliak, 2003). The availability of such social insurance lowers the risk of major disruptions in consumption due to changes in market income. Changes in final experienced income, however, should have direct effects on consumption and experiences of stress and privation.

The income needs, and alternately the stress felt at a particular income level, will vary across families based on family size. Establishing an equivalence scale to normalize income across different households is an important consideration in studies of income distributions and inequality. A common approach, attributed to work done with the Luxembourg Income Study by Buhmann and colleagues (1988, as cited in Coulter, Cowell, & Jenkins, 1992; Cowell & Mercader-Prats, 1999), divides income by household size raised to a certain power:

$$Y_h = \frac{X_h}{n_h^s} \quad (2.1)$$

where  $h$  indicates households,  $X$  is unadjusted household income,  $n$  is the household size, and  $s$  reflects “the elasticity of needs with respect to household size” (Cowell & Mercader-Prats, 1999, p. 418). “While this is clearly a very simple specification, we are reassured by Buhman et al.’s (1988) demonstration that this scale provides a good approximation to virtually all the different scales currently used in empirical studies of income distributions in developed countries including several scales that are based on other household characteristics in addition to size” (Coulter et al., 1992, p. 1069), such as weighting children and/or elderly

household members differently than adults. While the choice of equivalence scale can make major differences in examinations of individual level poverty or income inequality across countries (or time), this may be less of a concern in the current analysis. As Coulter and colleagues note, it appears to be most common to use  $s = .5$ , i.e. to use the square root of family size. For example, Iceland (2003) used the square root to scale relative poverty measures. All incomes in the current study were inflated to 2010 dollars using the Consumer Price Index and normalized using  $s = .5$ .

This brings up an issue regarding income measurement in the NLSY. While common practice (e.g., Classen, 2010; Smith, Stoddard, & Barnes, 2009) appears to be to treat the income or poverty status reported by NLSY by the data in a particular survey year as “belonging” to that year, the income data are specifically asked about the previous calendar year, while the codebook entry for the poverty status variable also indicates it is for the previous year. With the advent of alternate year surveys, however, the data compilers for NLSY do not have access to the family size for the prior year (or the residence, e.g., whether they lived in Alaska or Hawaii) to define the proper poverty threshold, without assuming it is no different than the family size reported at the time of the current survey or from the previous survey two years prior. Similarly, I am essentially forced to divide the prior year’s income by the current reported family size to the power of  $s$ . Using income reported in survey year  $YY$  as indicating income for year  $YY - 1$  appears to be in keeping with the way the data are collected by NLSY and builds in a lag, such that characterizing resources in this regard captures the idea that last year’s experience of poverty or instability affects this year’s stress level and consumption decisions. Of course, for many families, there is no lag between earning and spending.

The NLSY also includes a number of variables that more richly describe the socioeconomic status background of mothers and their children, including educational achievement, some assessment of wealth, whether the mother reported having lived in the South or in a rural area (combining the NLSY categories of “in country, not farm” and “on farm or ranch”) at age 14, and whether the mother was born outside the US or spoke a foreign language

growing up (specifically, other than English). Additional measures that might be related to both future economic circumstances and BMI include the mother's age at her first birth and her age at the subject child's birth, her religious upbringing (as another descriptor of the cultural background, recoded to Protestant, Catholic, Jewish, Other, and None), and her intellectual capacity, as captured by the Armed Forces Qualifying Test of math and verbal abilities. The AFQT was a subset of the Armed Services Vocational Aptitude Battery conducted with the NLSY79 sample as a special administration in 1980 designed specifically to renorm the ASVAB. The results became part of what is known as the "Profile of American Youth." Following the user's guide I used percentile scores based on renorming done in 2006. Information on the mother's familial educational achievement (i.e. that of her mother, father, and oldest sibling if any, assessed in 1979) was combined to form two variables capturing whether any relative had completed high school (if the highest grade completed was  $\geq 12$ ) and whether any relative had any post-secondary education (any degree or year of schooling beyond high school). Note that because this information was queried in the 1979 interview, the educational achievement may have come after the birth of some of the oldest sample members. Parallel variables were generated for the mother's education by the year *before* the subject's birth.

While the NLSY also includes a constructed net worth variable based on a series of questions about assets (investments, home, farm or business, etc.) and debts (mortgage, car loan, etc.), it appears asset questions were asked inconsistently before 1985 and not at all in 1991, 2002, and 2006. The home ownership question was asked more often than the other wealth items. Furthermore, the asset questions were asked in regards to the respondent (here, the mother) and any spouse, and the net worth variable does not cover the net worth of the whole family if the mother was still living with her parents. Due to these limitations, I use as an indicator of wealth only whether the mother (and/or her spouse) owned a home before the subject was born.

Race and ethnicity of the NLSY children was assigned based on the race/ethnicity assigned to the mother during the original NLSY screening process in 1978. As described in the

NLSY User's Guide, this assignment appears to have been based in part on interviewer discretion, and resulted in three possible categories: Hispanic (including Portuguese and Filipino), Black (which may or may not include Hispanics due to interviewer discretion), and Nonblack/Non-Hispanic. Fortunately, the main youth survey later asked respondents to self-identify, although this process resulted in some ambiguous categories (e.g., "English") and a known problem with identification of Native Americans. The unambiguous codes in the resulting data on sole or primary self-identified race/ethnicity were all Asian or Pacific Islander (e.g., "Chinese", "Vietnamese", or "Hawaiian"). I used this information to recode race and ethnicity, essentially pulling Asians and Pacific Islander out of the Nonblack/Non-Hispanic category and using the same NLSY definition of Hispanic. Due to the issues with Native American codes, any "true" Native Americans unfortunately remain in the not Hispanic, not black, and not Asian/Pacific Islander reference category, hereafter referred to as white, because they simply cannot be validly identified.

As with all longitudinal data, there are various patterns of missing data across the variables of interest. For example, many potential subjects are missing income data for key years. This may be due to the mother not answering the question or not being interviewed during the year, or it may be due to the child not living with the mother that year (as indicated in the interview), such that the income reported by the mother will not be counted as being experienced by the child. (The child must be reported as living at least part time with the mother for the income to apply.) As I will show below, other constructs frequently missing data include relatives' educational achievement (by the mother's father, mother, and oldest sibling if any), the mother's AFQT score, and whether she lived in the South at age 14. In addition, many potential subjects are missing constructed variables that refer to conditions at or before the child's birth (i.e. the mother's education and whether she owned a home) are not available because the adults were born in 1979 or earlier, such that there are no NLSY waves available before birth.

I conducted a moderately aggressive imputation strategy that reduced cases lost due to missing covariates but did not impute the financial variables. Specifically, the underlying

maternal interview data was imputed using a multivariate normal regression framework (implemented using the R package Amelia [Honaker, King, & Blackwell, 2011]), with categorical (including dichotomous) variables restricted to categorical predictions to avoid, for example, a value for whether the mother was foreign born of 0.28. Using the underlying maternal interview data allowed for using data from prior and following interviews to impute, for example, the mother's educational achievement in 1984. Doing the imputation on the raw mother data avoids giving siblings inconsistently imputed data. It also means that anything occurring before 1979 cannot be imputed. As we shall see, there remain 1145 cases missing the mother's education prior to the child's birth because 1145 cases were born in 1979 or earlier. I chose not to impute the financial data because I am trying to capture the effects of instability of income and poverty background, and the imputation would inherently smooth out income and poverty status. That is, I want to capture the effect of occasional (unconditionally if not conditionally random) extreme events, years where the mother's household income or poverty status does not resemble other years. The imputation procedure produced five versions of the underlying maternal data with different predicted values of missing data filled in to reflect uncertainty in the predictions, which were then processed into five versions of the final data. In addition, a sixth dataset averaging these five imputed datasets was necessary for such analyses as descriptive statistics and checking for outliers and non-linearity. The covariates used in each analysis and which were imputed are listed in Table 2.1.

Table 2.1: Covariate list

Covariate	Part 1:	Part 2:	Part 3:	Imputed
	Poverty	Income	Instability	
Any relative (grandmother, grandfather, and/or oldest aunt/uncle) finished high school	X	X	X	X
Any relative completed any schooling beyond HS	X	X	X	X
Mother completed HS by year of subject's birth	X	X		X
Mom any any post-HS education by subject's birth	X	X		X
Mother foreign born	X	X	X	
Foreign language spoken at home during mom's childhood	X	X	X	X
Mom lived in South at 14	X	X	X	X
Mom lived in rural area at 14	X	X	X	X
Subject gender	X	X	X	
Subject ethnicity (4 categories)	X	X	X	
Subject age	X	X	X	
Year BMI observed	X	X	X	
Mom's parents' religion (religion raised in; 5 categories)	X	X	X	X
Mom's age at first birth	X	X		
Years to subject's birth (since mom's first birth)	X	X		
Mom's pre-birth home ownership	X	X		
Mom's aptitude/intelligence in 1980	X	X	X	X

## 2.2 The basic sample

The data processing described resulted in a potential sample of 6361 adults with a legitimate BMI reported in the year the subject turned 19 or older. As seen in Table 2.2, the oldest subject is 38 years of age. Subjects were born between 1970 and 1991 to mothers aged 11 to 34. Tables 2.3 and 2.4 detail characteristics of the mothers and shows the effect of the imputation process. As noted, the imputation was able to fill in, for example, 294 missing values for whether the mother lived in the South at age 14, but not 1145 values of whether the mother completed high school or owned a home by the year before the subject's birth. The means and standard deviations for the imputed variables are quite close to those of the original data. The largest difference appears to be for relative post-secondary education, where the imputation process predicted what the oldest sibling's educational achievement would have been if the mother had an older sibling and also predicted a slightly higher rate of maternal parent (the subject's grandparents) post-secondary education.

Table 2.2: Basic young adult descriptive statistics

	Mean	SD	Min	1 <sup>st</sup> Qu	Median	3 <sup>rd</sup> Qu	Max	NA
Adult BMI	27.84	6.57	12.40	23.12	26.45	31.12	72.44	0
Age BMI observed	25.48	4.08	19.00	22.00	25.00	29.00	38.00	0
Female	0.49	0.50	0.00	0.00	0.00	1.00	1.00	0
Black	0.35	0.48	0.00	0.00	0.00	1.00	1.00	0
Hispanic	0.23	0.42	0.00	0.00	0.00	0.00	1.00	0
Asian/PI	0.00	0.05	0.00	0.00	0.00	0.00	1.00	0
Mom's age at S birth	23.11	4.27	11.00	20.00	23.00	26.00	34.00	0
Birth year	1983.81	4.39	1970	1981	1984	1987	1991	0

*Note:* SD is standard deviation, Min is minimum value observed, Qu is quartile, Max is maximum value observed, and NA indicates number of observations missing data. S indicates young adult subject.

Table 2.3: Basic mother descriptive statistics: Birth, education, aptitude, home ownership

	Mean	SD	Min	1 <sup>st</sup> Qu	Median	3 <sup>rd</sup> Qu	Max	NA
Mom's age at 1 <sup>st</sup> birth	20.55	3.89	11.00	18.00	20.00	23.00	34.00	0
1 <sup>st</sup> birth to S's birth, years	2.56	3.33	0.00	0.00	1.00	4.00	18.00	0
Any relative completed HS	0.73	0.44	0.00	0.00	1.00	1.00	1.00	78
(i) Any relative HS	0.76	0.43	0.00	1.00	1.00	1.00	1.00	0
Any relative some post-HS	0.28	0.45	0.00	0.00	0.00	1.00	1.00	78
(i) Any relative post-HS	0.38	0.48	0.00	0.00	0.00	1.00	1.00	0
Mom finished HS before S's birth	0.71	0.45	0.00	0.00	1.00	1.00	1.00	1145
(i) Mom HS before S's birth	0.71	0.45	0.00	0.00	1.00	1.00	1.00	1145
Mom some post-HS before S's birth	0.27	0.45	0.00	0.00	0.00	1.00	1.00	1145
(i) Mom post-HS before S's birth	0.29	0.45	0.00	0.00	0.00	1.00	1.00	1145
Mom's AFQT percentile score	33.20	26.46	0.00	10.81	26.90	50.47	100.00	273
(i) Mom's AFQT percentile	33.10	26.25	-20.69	10.99	27.09	49.87	100.00	0
Mom owned home before S's birth	0.32	0.47	0.00	0.00	0.00	1.00	1.00	1145

*Note:* SD is standard deviation, Min is minimum value observed, Qu is quartile, Max is maximum value observed, and NA indicates number of observations missing data. 1<sup>st</sup> birth to S's birth is years between mother's age at her first birth and her age at the subject's birth. (i) refers to average of 5 imputed values for each case.

Table 2.4: Basic mother descriptive statistics: Upbringing

	Mean	SD	Min	1 <sup>st</sup> Qu	Med	3 <sup>rd</sup> Qu	Max	NA
Mom foreign born	0.08	0.27	0.00	0.00	0.00	0.00	1.00	0
Foreign language spoken at home during mom's childhood	0.26	0.44	0.00	0.00	0.00	1.00	1.00	6
(i) Foreign language during childhood	0.26	0.44	0.00	0.00	0.00	1.00	1.00	0
Mom resided in South at 14	0.39	0.49	0.00	0.00	0.00	1.00	1.00	294
(i) Mom in South at 14	0.39	0.48	0.00	0.00	0.00	1.00	1.00	0
Mom lived in rural area at 14	0.20	0.40	0.00	0.00	0.00	0.00	1.00	27
(i) Mom in rural area at 14	0.21	0.40	0.00	0.00	0.00	0.00	1.00	0
Mom raised in other religion	0.10	0.30	0.00	0.00	0.00	0.00	1.00	25
(i) Mom other religion	0.10	0.30	0.00	0.00	0.00	0.00	1.00	0
Mom raised Jewish	0.00	0.06	0.00	0.00	0.00	0.00	1.00	25
(i) Mom Jewish	0.00	0.06	0.00	0.00	0.00	0.00	1.00	0
Mom raised Catholic	0.35	0.48	0.00	0.00	0.00	1.00	1.00	25
(i) Mom Catholic	0.35	0.48	0.00	0.00	0.00	1.00	1.00	0
Mom raised Protestant	0.51	0.50	0.00	0.00	1.00	1.00	1.00	25
(i) Mom Protestant	0.51	0.50	0.00	0.00	1.00	1.00	1.00	0
Mom raised with no religion	0.04	0.19	0.00	0.00	0.00	0.00	1.00	25
(i) Mom no religion	0.04	0.19	0.00	0.00	0.00	0.00	1.00	0

*Note:* SD is standard deviation, Min is minimum value observed, Qu is quartile, Med is median, Max is maximum value observed, and NA indicates number of observations missing data. (i) refers to average of 5 imputed values for each case.

### **2.3 Modeling concerns**

As noted in the introductory chapter, this project attempts to move beyond simple measures of whether an adult was ever poor as a child or some other single indicator intended to represent economic conditions in all of childhood. Instead, the goal here is to examine the effects of dynamics of economic experiences, borrowing from a broad literature on childhood SES and adult health. One advancement is to examine the timing of events. For simplicity, I label as timing models specifications that estimate the effects of dichotomous indicators of whether an event ever occurred within a developmental period, regardless of how often. Accumulation models, in contrast, highlight the effects of how frequently events occurred during childhood, as in the proportion of childhood spent in poverty. Finally, I test hybrid models that test the accumulation of conditions within developmental periods.

Theories and studies of child development frequently divide childhood into various stages, often comprising the prenatal period, infancy and toddlerhood (birth to age 3), the preschool period (until 5 or 6), middle childhood (until 12), and adolescence (Feldman, 2004). Sometimes coarser divisions are used, as in Greg Duncan and colleagues' research on the effect of poverty during ages 0 to 5 versus poverty from age 6 to 15 on secondary school completion (as cited in Morris et al., 2005). Investigations of biological mechanisms may separate the early postnatal period from infancy, or combine this period with the pre- and perinatal months (e.g., Martin-Gronert & Ozanne, 2010). The closest comparison to the current project is the study in the PSID by Ziol-Guest and colleagues (2009), who differentiated among survey results from the birth year, to account for prenatal and early postnatal effects, from characteristics from when the child was ages 1 to 5 and from the later childhood period of ages 6 to 15. In order to investigate whether poverty or instability during different developmental periods is important in setting up the individual for future obesity, I will investigate by similar periods. Specifically, the definition of poverty or instability under consideration will be assessed and entered into the model for the birth year through toddlerhood (0–3), the preschool years (4–5), middle childhood (6–11), and adolescence (12–17). Data limitations often require combining the 0–3 years and the preschool years to create a single, larger early

childhood experience, so many of the reported timing and particularly hybrid model results use three periods. Hypotheses about the effect of instability include comparing the effects of instability experienced by the mother before the birth of the child with that experienced while the child is present, so Part 3 adds the available pre-birth period up to a maximum of 10 years. The significant amount of income volatility in the US means that while incomes in these periods will be moderately correlated, these correlations are low enough to allow estimation of effects for different periods (Ziol-Guest et al., 2009). I would like to separate the birth year from infancy and toddlerhood in order to test the fetal origins/programming hypothesis, but the biennial nature of recent NLSY waves means that many offspring do not have mother interviews in the year they were born.

Exploration of potential non-linearity in the relationships between predictors and BMI, using both univariate residual plots and the multivariate Alternating Conditional Expectations algorithm of Raftery and colleagues (Hoeting, Raftery, & Madigan, 2002), indicated that using the natural log of BMI provided better fit. Unlike analyses with older samples that have found a curvilinear effect of age on BMI (Cook & Daponte, 2008; Komlos & Brabec, 2010; Walsemann, Ailshire, Bell, & Frongillo, 2012), in this younger sample adding a second term to capture deceleration of BMI growth and/or peak BMI over the age range has no modeling benefit. Extensive analysis of outliers and influential cases also found that fit was improved by Winsorizing BMI to the 1<sup>st</sup> and 99<sup>th</sup> percentiles—i.e. recoding all observations below the 1<sup>st</sup> percentile to the value of the 1<sup>st</sup> percentile, and all those greater than the 99<sup>th</sup> percentile to the 99<sup>th</sup> percentile.

Many studies of adiposity that rely on longitudinal data, including the work by Smith and colleagues (2009) upon which Part 3 is partly based, include a baseline BMI or obesity status. This baseline helps control for any unobserved, stable characteristics of the individual that might be predictive of later BMI, and means that these studies examine change in adiposity over time, or how the predictor of interest affects average BMI trajectories. Any putative drivers of adiposity that occur between baseline and final measurement thus affect whether BMI has increased or decreased. Similarly, maternal weight status is a powerful predictor of

child weight (Classen, 2010; Daniels, 2006; Laitinen, Power, & Jarvelin, 2001) but is likely endogenous to the family income–child adiposity relationship.

Beginning evidence indicates that experiences of poverty and SES can influence birth weight and BMI very early in childhood (Anderson, Butcher, & Schanzenbach, 2007; Balistreri & Van Hook, 2009; Laitinen et al., 2001), setting up the child for a lifetime of excess adiposity. The effect of maternal poverty or of income instability in the 0 to 3 period on later adiposity may thus be partially or fully explained by the effect on BMI by middle childhood. As noted, evidence indicates that initial BMI level is correlated with growth in BMI (Brown, Williams, Ford, Ball, & Dobson, 2005; Gius, 2011; Gordon-Larsen et al., 2009; Ruel, Reither, Robert, & Lantz, 2010). Thus, if one is interested in establishing the total effect of earlier income, one should leave out temporally intervening variables that might explain part of the effect on later adiposity. Ziol-Guest and colleagues (2009), for example, specifically included as control variables only time-consistent factors or conditions measured before or around the child’s birth, and did not include any baseline weight or BMI variable. While Lee and colleagues (2009) examined change in obesity status from Wave II to Wave III of Add Health as a function of poverty before age 18, participants were between 13 and 20 years of age at Wave II, and so for most the experience of poverty likely came before Wave II.

The selection of covariates was made to capture as many important influences on economic circumstances and/or BMI as possible. The focus here is on capturing the total effect of childhood background on later adiposity. In order to reduce potential endogeneity, I have endeavored to use controls theoretically exogenous to the material resources–adiposity relationship, generally by including as controls only invariant characteristics (e.g., gender, ethnicity) or measured variables that temporally precede the initiation of the relationship of interest, such as the educational achievement of the mother’s mother, father, and oldest sibling if any. As seen in Table 2.1, this necessitates different covariates, as the instability analyses of Part 3 include instability experienced by the mother before the subject’s birth. The BMI itself and the effects of various contributors are thought to vary by gender and

ethnicity, due to physiological differences, variation in experiences of poverty, cultural ideals regarding diet and body size, and other potential mechanisms. Therefore, initial models (Parts 1 and 3) include indicators for each of eight ethnicity (white, African American, Hispanic, and Asian/Pacific Islander) by gender groups. Part 2 explores the differential effects of income across these groups via interactions between the predictors of interest and indicators of gender and ethnicity (except for Asian/Pacific Islander, as there are too few in the sample).

Out of the 6361 offspring in the NLSY79 data with valid adult BMIs, 5337 are members of sibling groups represented in the data. Among the 3103 mothers represented, 2079 have multiple children, from two to eight, in the data. To account for the correlation of errors among family members, I estimate all models (i.e. throughout this project) with heteroskedasticity-consistent standard errors, clustering within families, to account for both a family-specific correlated error component and the usual idiosyncratic error.

The relationship between background material resources and future BMI may be due to factors predictive of both income and BMI. While some of these factors will be entered as controls, unobserved factors may create upwardly biased ordinary least squares (OLS) estimates of the parameters of interest. These might include other parent characteristics such as mental illness, physical maladies, or intelligence that are predictive of both poverty (via job loss, disability, etc.) and child BMI (via modeling of diet and exercise or inherited traits). These nuisance factors may directly or indirectly cause both poverty and adiposity, and thus the unexplained heterogeneity from these exogenous variables creates the bias in the OLS estimates (Cameron & Trivedi, 2005). Fixed effects (FE) models are often employed in obesity research to control for unobserved propensity toward obesity (e.g., Cohen-Cole & Fletcher, 2008; Eid, Overman, Puga, & Turner, 2008; Liu, 2007).

I conduct parallel estimates of all models in Parts 1 and 3 using a sibling or family fixed effect, essentially including a separate indicator for each mother to account for time-invariant unexplained heterogeneity. The analysis for all models compares the estimates

from the more efficient but biased (clustered) OLS and the FE estimation. The FE model parameters are estimated among sibling groups, creating a smaller sample size. Each model is identified to the extent that characteristics differ among siblings, resulting in further loss of efficiency due to the underlying differencing of observations from the family mean for each variable and accompanying loss of degrees of freedom (i.e. one per family). Since non-twins are born some time apart, the pre-birth and developmental periods may differ between siblings, and thus the economic circumstances for the relevant period as well as other controls will differ. Sibling pairs that do not differ on these characteristics—more likely with whole childhood descriptors such as the simple poverty dichotomy in Part 1—cannot be included in the estimation. Siblings that are identical on these variables do not contribute to the estimation. Any characteristic constant among families, such as parental ethnicity or level of education, cannot be included in a model using family fixed effect estimation. This means that models involving interactions with ethnicity cannot include “main” effects for Hispanics, African Americans, etc. (NLSY ethnicity for children is based solely on the mother’s ethnicity, so even children with different fathers cannot be listed as having different ethnicity). The fixed effects analysis also accounts for correlation of errors within families. FE estimation controls for only time-invariant unobserved heterogeneity; the assumption here is that any residual correlation between the material advantage conditions of interest and the error term due to time-varying unobserved factors approaches 0 net of the other variables in the model.

## Chapter 3

**PART 1: POVERTY DYNAMICS IN CHILDHOOD AND ADULT  
ADIPOSIITY****3.1 Background**

Social policies from the initiation of the War of Poverty to Head Start to State Children's Health Insurance Programs demonstrate the concern we as a nation have with the consequences of poverty, particularly among children. Major figures in the study of poverty, from Glen Elder to Gary Becker to Oscar Lewis, have been implicitly concerned with the effects of poverty on child development. Whether these effects are theorized to occur via reduced investment by parents, stress, or accompanying fluctuations in social and cultural capital, attention in such studies has been on the feared risks associated with growing up poor (Magnuson & Votruba-Drzal, 2009).

As with a variety of health outcomes, there is a well known relationship between obesity and measures of socioeconomic status (SES; Averett, 2012; Cook & Daponte, 2008; Huang, Drewnowski, Kumanyika, & Glass, 2009; Lee, 2011; Offer, Pechey, & Ulijaszek, 2010; Ogden, Lamb, Carroll, & Flegal, 2010). A subset of this literature focuses on experiences of poverty as a main driver of obesity. The connection between obesity and low SES or poverty has entered the public consciousness, and, as Marc Ambinder wrote in *The Atlantic*, "In fact, obesity has become a marker of sorts for lower socioeconomic status" (2010, p. 76). Much of this research attention to the poverty-obesity link is cross-sectional and among adults. Obesity is costly, in terms of health care, impaired school and work functioning, and self-esteem. Studies that have attempted to isolate the causal effect of excess weight have found evidence of a negative effect on wages and family income for women, particularly white women (Cawley, 2004; Conley & Glauber, 2007; Gortmaker, Must, Perrin, Sobol, & Dietz, 1993). A portion of the obesity-poverty relationship in cross-section may be due to the obesity promoting the poverty. What part of the concurrent correlation is due to the

opposite effect, of economic disadvantage causing excess adiposity over time?

In this analysis, I advance investigation of the relationship between poverty and body fat in several ways. First, I take advantage of linked mother and child data from the National Longitudinal Surveys of Youth 1979 cohort (NLSY) to associate maternal income with particular ages during the childhoods of now-adult sample members. Second, given criticisms of the usual US definition of poverty and prior evidence that this definition may not identify a group uniquely at risk for higher adiposity, I use multiple definitions of poverty. Finally, I apply these multiple definitions using different models of how economic background influences later health to examine the effects of timing and/or accumulation of poverty experiences in childhood on adult adiposity. I find little relationship between child poverty and adult adiposity across the sample: There is little evidence in this analysis that poverty experiences, by any definition, identify a unique group at risk for later excess body fat. Differential effects of poverty for different groups may explain part of this null finding. Similarly, as the focus here is on finding the total effect of child poverty, experiences after childhood—diet and exercise patterns adapted in early adulthood, smoking and other health behaviors—apparently moderate the effect of poverty.

### *3.1.1 Investigating the relationship between income and adiposity*

Amidst all the attention on obesity and economic factors, relatively little has provided evidence of a causal effect of economic conditions on adiposity. While a number of studies have connected earlier SES to later obesity in both adults and children, much of this work has concentrated on father's occupation, parental education, or one's own education, establishing the effect of low childhood SES on later adiposity (Laitinen, Power, & Jarvelin, 2001; Senese, Almeida, Fath, Smith, & Loucks, 2009) even controlling for adult SES (Langenberg, Hardy, Kuh, Brunner, & Wadsworth, 2003; Power et al., 2005). In such studies, the outcome related to earlier SES is either adult obesity or adult body mass index (BMI). BMI is the most common measure of adiposity. It is defined as weight in kilograms  $\div$  the square of height in meters:  $BMI = \frac{kg}{m^2}$ . Obesity is generally defined as having a BMI  $\geq 30$ , overweight as  $\geq 25$ .

Focusing on income, the Alameda County Study (Baltrus, Everson-Rose, Lynch, Raghunathan, & Kaplan, 2007) found that older males' own household income was significantly related to BMI growth, but this relationship did not hold among the younger males or either group of females. Furthermore, the positive finding for older males may suffer from the endogeneity of earlier BMI. Schmeiser (2009) found a significant positive effect of exogenous income increases on female but not male BMI among low income adults, while Smith and colleagues (Smith, Stoddard, & Barnes, 2009) found exogenous income to be associated with weight loss in their study of men of all incomes.

While most of the support for an income–obesity gradient is cross-sectional, the link between income and adiposity has attained the status of conventional wisdom. A special report in *The Economist* highlighted

one notable feature of the obesity epidemic. The rich and well-educated have mostly managed to stay slim, or at least have got less fat than the less well off. . . . Among the many explanations put forward for this link is that educated people may be better informed about health issues. They may also have better access to doctors, gyms, parks and healthy food. The poor may be under greater stress, making it harder for them to resist food that is cheap, tasty, convenience and relentlessly advertised. Whatever the cause, a lot of poor and uneducated people are also fat (Howard, 2012, p. 5).

Growing up in relative advantage means access to an enriched environment that promotes healthy development. “Doctors, gyms, parks and healthy food” all come at a cost that may be beyond segments of the population. In addition, wealthier neighborhoods not only have access to these goods but also to role models exhibiting good health behaviors.

The people most vulnerable to obesity, however, do not have access to healthy food, to role models, to solid health-care and community infrastructures, to accurate information, to effective treatments, and even to the time necessary to change their relationship with food. And if that is true for fat adults, it is even

more true for fat children, many of whose choices are made *for* them. Their vulnerability to obesity is much more the result of societal inequalities than of any character flaw. Indeed, for all the attention paid to fat's economic costs, the epidemic's toll on children is a stark reminder of its moral dimension (Ambinder, 2010, p. 74, italics in original).

Cross-sectional studies show that poor children are more likely to be overweight or obese (e.g., Anderson, Butcher, & Schanzenbach, 2007; Ogden et al., 2010; Phipps, Burton, Osberg, & Lethbridge, 2006; Singh, Siahpush, & Kogan, 2010; Storey, Forshee, Weaver, & Sansalone, 2003) , and “obese children in the poorer families are heavier than obese children in wealthier families” (Anderson et al., 2007, p. 10). A handful of studies have found relationships between childhood poverty or income levels and later BMI or obesity (Evans & Kutcher, 2011; Lee, Harris, & Gordon-Larsen, 2009; Ziol-Guest, Duncan, & Kalil, 2009), but by no means has the causal connection between childhood income levels and the development of obesity been firmly established, much less the mechanisms by which earlier disadvantage results in higher BMI.

Thus, we have an accepted relationship between early SES and later BMI that appears to be mostly based on studies of education or occupation, but seems to manifest itself in declarations that poor children are at high risk of developing obesity and attendant health risks. Obesity is frequently included in lists of childhood health disparities associated with poverty or low income (e.g., Conroy, Sandel, & Zuckerman, 2010). Furthermore, discussions of long-term health effects of low childhood SES frequently focus on conditions related to obesity, such as cardiovascular disease or diabetes, and often highlight health behaviors such as diet and exercise as mechanisms connecting lower SES to morbidity and mortality (e.g., Cohen, Janicki-Deverts, Chen, & Matthews, 2010; Conroy et al., 2010; Hayward & Gorman, 2004; Seeman, Epel, Gruenewald, Karlamangla, & McEwen, 2010). Countering arguments that a tax on sugar-sweetened beverages would be regressive, a health policy report in the *New England Journal of Medicine* noted the tax would have long-term health benefits for the poor and that “designating revenues for programs promoting childhood

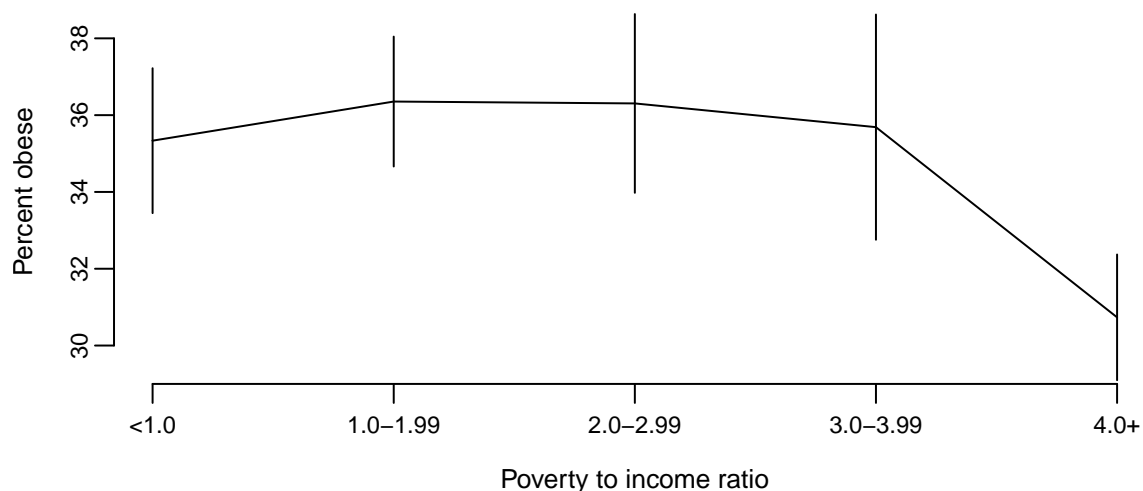
nutrition, obesity prevention, or health care for the uninsured would preferentially help those most in need” (Brownell et al., 2009, p. 1603). At the beginning of 2013, the UK health minister claimed that it is now easy to pick out which children in the schoolyard are poor “because their diet... means they tend to be fat.” In response to what was seen as incriminating poor parents, a British child poverty advocacy group claimed that “‘piles and piles of evidence. . . make it absolutely clear that the real reason why our obesity problem is going to get bigger in the years ahead is because our child poverty problem is going to get much bigger’” (Booth, 2013).

### *3.1.2 Testing poverty as a risk factor*

We believe low income puts children at risk of becoming obese and remaining obese into adulthood. Because income (specifically, family income with respect to needs) is continuous, this implies some threshold below which children are most at risk for the outcomes associated with such relative deprivation, including obesity. Experiencing income below this threshold implies increased risks and inability to access protective factors. Such a threshold is, along with decisions about what and whose income to count, how we operationally define poverty. We define and measure poverty in part because of a desire to identify who is at risk for poor outcomes.

Applying such a threshold consideration in a causal sense to concurrent data implies some balance between two prospects: 1) That there is some continuity in the economic standing of the subjects; or 2) Poverty acts quickly to cause adiposity. Figure 3.1 presents the proportion obese among adults as a function of poverty-income ratio, the ratio of reported family income to the appropriate federal poverty level (FPL), from National Health and Nutrition Evaluation Survey (NHANES) data pooled over the 2001–2002 to 2009–2010 waves for adults 20 to 74. Here we see little difference in obesity rates until income reaches four times federal poverty level ( $PIR = 4+$ ). Again, the apparent relative protective effect of high income may be a function of reciprocal relationships between socioeconomic conditions and adiposity (Kawachi, Adler, & Dow, 2010).

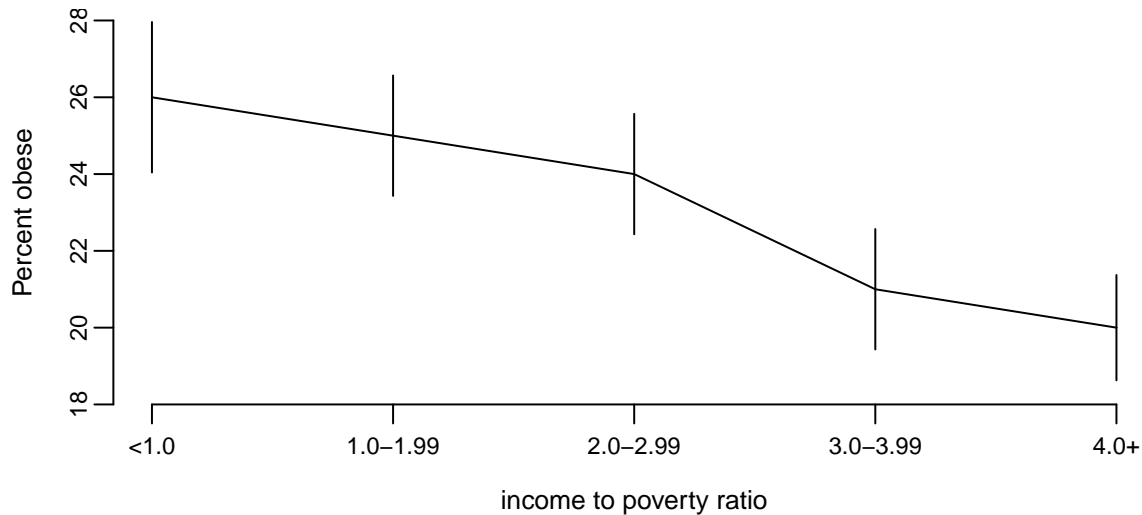
Figure 3.1: Obesity prevalence (with 95% confidence intervals) among adults 20 to 74 by poverty-income ratio group, NHANES 2001 through 2010



Analyses that control for other demographic correlates of BMI similarly find that BMI or obesity prevalence among the currently poor are little different than adults with household income up to three times the FPL, particularly among men (Kant & Graubard, 2007; Komlos & Brabec, 2010). In Figure 3.2 below, I graph the adjusted obesity prevalence among the adults in Kant and Graubard’s sample, controlling for demographics, lifestyles variables, and education. (The overall prevalence is lower than in Figure 3.1 due to period effects, as Kant and Graubard included NHANES I [1971–1975] through NHANES 2001–2002.) Adjusting for education, age, ethnicity, etc., the obesity prevalence among those with a PIR of three to less than four times FPL was significantly lower than that of those with lower incomes. That is, even adjusting for other relevant factors, we continue to see evidence that current income does not appear to be protective in terms of lower body fat until levels significantly above poverty level.

Among children ages 2 to 19, NHANES results from 2005 to 2008 (Ogden et al., 2010) divide the family income range into  $PIR \geq 350\%$ ,  $130\% \leq PIR < 350\%$ , and  $PIR \leq 130\%$  and demonstrate the usual gradient overall (in both genders). Poor children with family incomes below 130% of the usual FPL have a prevalence of obesity 3.5 to 3.7 percentage points higher than

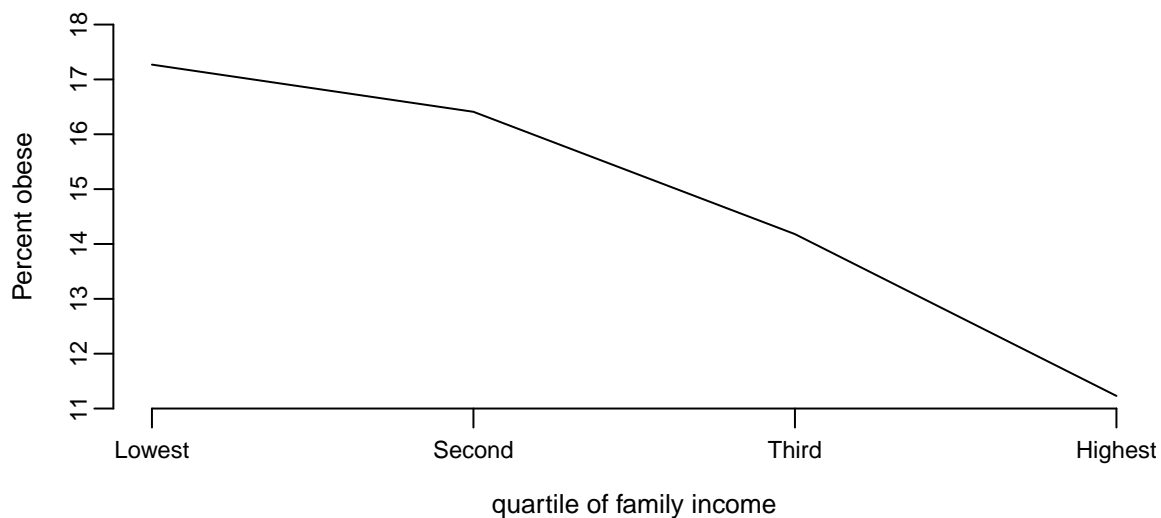
Figure 3.2: Predicted obesity prevalence (with 95% confidence intervals) among adults 25 to 74 by poverty-income ratio group, NHANES 1971 through 2002 from Kant and Graubard (2007)



those between 130% and 350%, who in turn are 3.8 to 5.5 percentage points more likely to be obese than those with incomes above 350% of FPL. In contrast, Figure 3.3 graphs the obesity rate among young children by income categorized by quartile of poverty-income ratio, from the analysis of NHANES data over 1999 to 2004 by Anderson and colleagues (Anderson et al., 2007). Here we see little difference in obesity prevalence between children in the first and second quartiles (about 17.3% and 16.4%). Above the median, prevalence drops notably.

For both adults and children, there appears to be little decrease in obesity prevalence until well above the usual poverty line. At least in cross-section, the group at high risk for excess adiposity includes those with incomes above what we officially count as poverty level. Turning to a potential causal effect of poverty, this implies that a different threshold for poverty might be more consistently predictive of obesity. Lee and colleagues (2009) used a slightly broader definition of poverty (household income below FPL or receipt of welfare at any point during childhood) in finding that those poor were more likely to remain obese

Figure 3.3: Obesity prevalence among children 2 to 11 by quartile of poverty-income ratio, NHANES 1999 through 2004, from Anderson et al. (2007)



(be obese in adolescence and young adulthood) and more likely to become obese (between adolescence and young adulthood).

Alternately, a relative measure of poverty may be more predictive of obesity than a static measure with a fixed threshold. Relative poverty measures, more common in Europe, are thought to capture a socially defined level of needs in a particular country. Relative poverty definitions still rely on a threshold, often 50% of median income, but as this threshold is pegged to a central tendency measure of the overall income distribution, this threshold rises (or falls) with the overall level of income in a society (Iceland, 2003; Meyer & Wallace, 2009). As with the usual absolute poverty measure in the US, research links relative poverty measures to adverse health outcomes (Emerson, 2009). Phipps and colleagues (2006) examined relative child poverty ( $\leq 50\%$  median normalized after-tax household income) among children 6–11 using NHANES 1988–1994. They found the extent of obesity (proportional severity or depth times prevalence) to be significantly greater among poor children than non-poor children. The difference in Canada was statistically significant but smaller than in the US. Although not corresponding exactly to 50% of the median, note that there is

little difference between children in the first and second quartiles in Figure 3.3 above, but a large difference between all those below and above (100% of) the median.

Measuring poverty is a decades-old phenomenon which expresses, among other things, the concern we as a society have for the effects of economic deprivation, particularly the effects on children. Our traditional poverty measure, however, may not fully account for the group at risk of these privation-related outcomes, like obesity. One task is therefore to assess the predictive validity of the usual definition of poverty and perhaps find another definition that works better. Below, I demonstrate a methodology for finding an optimal poverty threshold (both in the usual absolute poverty framework and a relative poverty definition, where the threshold is a fraction of relevant median income). Such an optimal threshold addresses one part of the poverty definition, keeping constant what sources of income accruing to whom are counted, in a predictive validity framework. If we are concerned about poverty because of what it portends, the best definition of poverty is the one that is most strongly linked to the negative outcome of concern.

The experience of poverty is not static, and those obese individuals with incomes above the usual poverty threshold who seem to be interfering with the assumed income gradient may well have been poor in the past (Jantti, 2009). The nature and timing of poverty offers potential insight into the mechanisms that cause obesity and policy and programmatic interventions that might reduce future obesity risk. The relationship between dynamics of childhood SES and later health has been conceptualized and investigated in two main models. The *timing* or developmental period model, also known as the latency model, focuses on the effects of SES-related factors during sensitive or critical periods during the child's development that manifest later in life (Cohen et al., 2010; Conroy et al., 2010). Similarly, the developmental literature highlights the importance of the timing of childhood experiences and conditions, including critical periods for interventions (e.g., Miller, Maguire, & Macdonald, 2011; Morris, Duncan, & Clark-Kauffman, 2005) as well as the timing of insults such as child maltreatment (e.g., Jaffee & Maikovich-Fong, 2011). The fetal origins hypothesis is a subset of the timing model focusing on the importance of the prenatal

through post-partum periods. For example, a study using data from the PSID (Ziol-Guest et al., 2009) compared the effects of family income (below \$25,000) during the prenatal and birth years, ages 1 to 5, and ages 6 to 15. Increased income at low levels during the first period only was inversely associated with BMI and weight status at 30 to 37 years of age, net of controls and income in the other periods. Although the authors interpret this as support for the physiological programming of obesity propensity due to income-related deprivation and insults, the primacy of this period may operate through the mother and thus could be more psychological in nature (stress reactions, diet and exercise habits, etc.).

The timing model implies that falling into poverty once at any point during a particular period sets in motion forces that promote adiposity. Others argue for an *accumulation* (Cohen et al., 2010) or cumulative effects or pathways model (Conroy et al., 2010). This approach places emphasis on the duration and extent, or dosage, of stress and deprivation, as in the concept of allostatic load, which relates chronicity of stress to dysregulation among various physiological systems, including metabolism (Seeman et al., 2010). Rather than a dichotomy of any childhood poverty versus never in poverty, Wells and colleagues (Wells, Evans, Beavis, & Ong, 2010), for example, tested an accumulation model via the proportion of time in poverty from birth to 9 years of age, and found a significant positive linear effect on BMI growth over time (it does not appear they tested a nonlinear effect in this mostly white sample). There appears to be no example of combining these models to test the differential effects of accumulation within developmental periods.

This study follows Ziol-Guest and colleagues (2009) in examining poverty dynamics using income data in multiple developmental periods over subjects' entire childhoods, and Wells and colleagues (2010) in testing the accumulation of poverty over childhood. In sum, I explore three models that move beyond the crude dichotomous poverty indicator often used in this research, to examine the accumulation of childhood poverty, the timing of poverty, and a hybrid model that tests the effects of the proportion of each developmental period spent in poverty. I furthermore demonstrate how different conclusions might be drawn from such analyses with different definitions of poverty. For this initial investigation, I control

for gender and race/ethnicity but do not test for differential effects of poverty dynamics by gender and ethnicity groupings.

### 3.2 Modeling

In this analysis, the concern is with modeling the effect of being in poverty at any point during childhood (defined in various ways) on later adiposity, and then advancing the complexity of poverty measurement to examine whether considering the dynamics of childhood poverty provides better prediction of adult BMI than a simple dichotomy. A simple model might thus be

$$\ln BMI_i = \beta_0 + \beta_1 POV_i + \beta_2 P_i + \beta_3 C_i + \beta_4 T_i + \varepsilon_i \quad (3.1)$$

where  $i$  indicates individual, and  $P$  and  $C$  represent vectors of parental and child covariates, including age of the adult child as well as gender and ethnicity and family education and wealth indicators, maternal age at first birth and age at subject child's birth.  $T$  is a linear time trend capturing secular increases in BMI and  $\varepsilon$  is the usual error term.  $POV$  is the measure of poverty from birth to age 17, initially a simple dichotomy. The assumption here is that the eating habits or physiological adaptations underlying the connection between poverty and later obesity are set by 18 (and I leave for future research whether these effects hold net of adult economic contextual factors). The control variables isolate the effect of interest ( $\beta_1$ ) as well as account for factors that may be associated with both poverty and obesity which otherwise would bias the estimated effect of poverty. Extensive examination of potential nonlinear relationships between predictors and BMI found that using a log transformation of BMI improved model fit. The resulting linear models are thus accounting for percent changes in adult BMI.

The first task here is to find an optimal absolute poverty threshold that outperforms the usual definition in terms of predictive power. It may be that a different threshold, say at 150% or 250% of the poverty line, is more predictive of BMI. Alternately, some experts prefer a relative poverty measure, thought to capture a socially defined level of needs in a particular country. As noted, relative poverty definitions often use a threshold of 50% or 60% of median income, so the search for a threshold that is most predictive of

later adiposity applies. These optimal definitions can be compared to the usual poverty definition or to categorizations of low income that are used as eligibility criteria, such as 185% of 200% of FPL, which are often used by states to denote eligibility for assistance such as the Supplemental Nutrition Assistance Program (SNAP, formerly known as the Food Stamps program), the supplemental program for Women, Infants, and Children, and Children’s Health Insurance Programs.

So far, this analysis repeats the “important limitation of most large-scale studies that link early childhood socioeconomic conditions to adult health outcomes [which] is their reliance on relatively crude measures of socioeconomic status, such as” being in poverty at any time in childhood (Ziol-Guest et al., 2009, p. 528). As described above, there are various ways to move beyond such a simple dichotomization to examine when and how much poverty in childhood appears necessary to promote adiposity in adulthood. These improvements come via considering the timing of poverty, the accumulation of poverty, or both.

To explore whether poverty during different developmental periods is important in setting up the individual for future obesity, I follow a plan similar to Ziol-Guest and colleagues (2009). Specifically, the definition of poverty under consideration will be assessed and entered into the model to capture any observed year in poverty during the birth year through toddlerhood (0-3), the preschool years (4-5), middle childhood (6-11), and adolescence (12-17). The significant amount of income volatility in the US means that while incomes in these periods are moderately correlated, these correlations are low enough to allow estimation of effects for different periods (Ziol-Guest et al., 2009). The resulting model will thus resemble

$$\ln BMI_i = \beta_0 + \sum_{a=1}^4 \gamma_a POV_{ai} + \beta_2 P_i + \beta_3 C_i + \beta_4 T_i + \varepsilon_i \quad (3.2)$$

with four  $\gamma$  coefficients replacing  $\beta_1$  and corresponding to the four age ranges subscripted by  $a$ . Comparing results for the model represented by equation 3.1 and the model represented by equation 3.2 will add to the literature about the timing of poverty in affecting behavioral health outcomes.

It is possible that different levels of poverty may have more or less impact on later adiposity during different developmental periods. If more resources are required to ensure optimal nutrition during infancy than are needed during middle childhood, for example, this implies different thresholds for defining poverty in each period. On the other hand, it is possible that period-specific thresholds would not differ enough to have any policy implications. The multiply developed nature of adiposity indicates that many poor children will turn into non-obese adults, and many children who did not experience poverty will become obese. It is therefore likely that period-specific threshold estimates would not significantly differ from each other. If applying this method of threshold selection to overall childhood poverty is successful, future research can pursue the question of different thresholds by developmental period.

A second method to examine poverty dynamics, the accumulation model, is to measure the proportion of childhood spent in poverty, as in Wells et al. (2010). A single year in poverty might not create permanent change in glucose tolerance or diet and exercise patterns. A larger proportion of time in poverty might lead to stocking up and increased consumption of cheap foods when income later increases, whereas a childhood spent almost entirely in poverty might result in constantly restricted diet. This analysis will thus replace the dichotomous poverty indicator (*POV*) in the basic model above (3.1) with the proportion of years during childhood (among those with three or more poverty status observations available) that the household income was at or below the poverty level, using the various definitions discussed above.

Ideally the poverty indicator in the developmental period model (3.2) would be the proportion of time during the period in poverty. This hybrid model would allow the most sensitivity to poverty dynamics. Doing so, however, requires at least two years of income observations per period. Given the biennial nature of the NLSY after 1994 and the vagaries of missing data, very few cases have income recorded for the years they turned 4 *and* 5. In order to test this hybrid model, I combine the preschool years with toddlerhood to examine accumulation of poverty during early childhood versus middle childhood and adolescence.

The control variables (in  $P$  and  $C$  in the equations above) were selected in order to account for multiple and often overlapping influences on both economic circumstances and BMI while allowing the investigation to isolate the total effect of background poverty experiences. The child covariates included are those known to affect BMI, including age and interactions between ethnicity and gender. Investigation of potential nonlinearity indicated that, unlike analyses with older samples that have found a curvilinear effect of age on BMI (Cook & Daponte, 2008; Komlos & Brabec, 2010; Walsemann, Ailshire, Bell, & Frongillo, 2012), in this younger sample adding a second term to capture deceleration of BMI growth and/or peak BMI over the age range has no modeling benefit. The eight gender by ethnicity (white<sup>1</sup>, African American, Hispanic, and Asian/Pacific Islander) indicators allow for fitting separate intercepts to account for known BMI differences between, for example, African American women and white men.

The choice of maternal covariates is more germane to issues of total effect and endogeneity. I model the effect of childhood poverty dynamics controlling for characteristics of the mother that temporally precede, although may still be correlated with, the child's poverty experiences. These include the education of the mother's own family and her own education as of the child's birth, whether the mother was foreign born or spoke a foreign language growing up, her religion during her childhood, her age at her first birth and the years between this birth and the subject's birth, if any, whether the mother lived in the South or in a rural area at age 14, whether she (and/or her spouse) owned a home at any time before the child's birth, and the mother's percentile score on the Armed Forces Qualifying Test, an assessment of aptitude.

Although controls such as ethnicity, maternal background education, and an indicator of wealth (home ownership) likely account for many processes that might be correlated with both experiences of poverty and BMI, there likely remain constructs unaccounted for that might bias ordinary least squares (OLS) estimates of the effect of poverty on later adiposity.

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<sup>1</sup>Due to NLSY coding issues, it is not possible to reliably pull Native Americans and Alaskan Natives out of the group neither black, Hispanic, nor Asian/Pacific Islander. Therefore, "white" here includes both Caucasian Americans and some Native Americans.

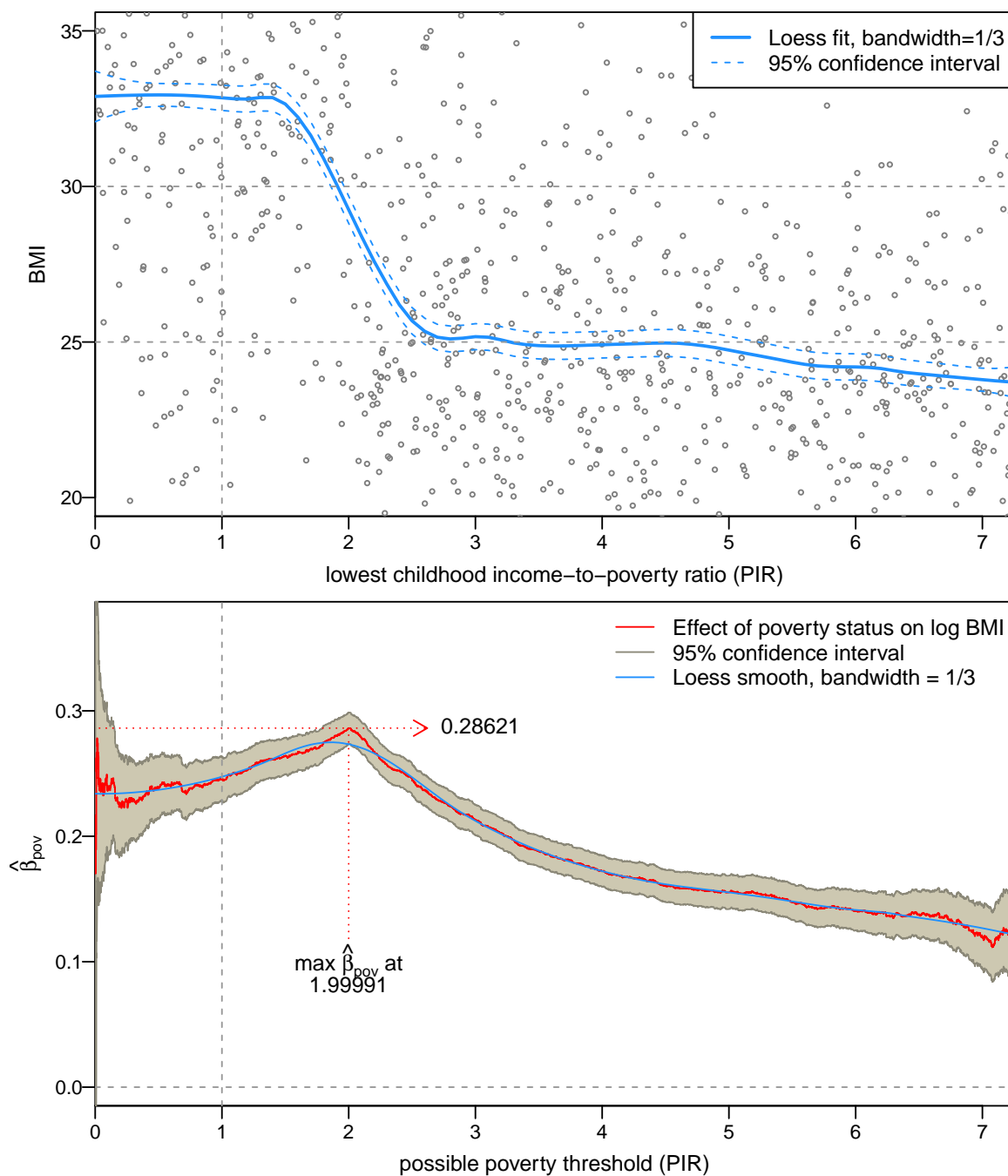
I take advantage of the numerous sibling groups present in the NLSY Child and Young Adults data—out of the 6361 offspring with valid adult BMIs, 5337 are members of sibling groups representing 3103 mothers—to implement sibling or family fixed effects to account for unobserved, time-invariant heterogeneity in family-level propensity towards poverty and adiposity. The modeling relies on differences within siblings in poverty experiences as well as the covariates. As a result, each fixed effects model is estimated on a smaller sample with fewer covariates.

### **3.3 Finding an optimal absolute threshold**

The choice of which of potentially infinite poverty thresholds has maximum predictive power can be conceptualized as a problem of maximizing the model fit in the basic model (3.1) where *POV* is an indicator for whether income was ever below a candidate cutoff, over all (reasonable) potential values of the cutoff. I implement essentially a grid search, creating poverty indicators for whether the lowest value of the ratio of a year’s household income to that year’s appropriate poverty threshold (in parallel with the NLSY construction of the Poverty Status variable) is less than or equal to the threshold in question, over all observed income-to-poverty ratios. Specifically, for each iteration, each subject’s lowest observed childhood income-to-poverty ratio (using the official threshold) is compared to the candidate threshold (a fraction or multiple of the official threshold). If the subject’s lowest poverty-income ratio (PIR) is less than or equal to the candidate threshold, the subject is judged to have been in poverty at some point during childhood. This poverty indicator is then used in the basic model and the ordinary least squares (OLS) coefficient estimate for the poverty indicator,  $\beta_1$  in equation 3.1, is recorded. One way of finding the best threshold is then identifying the cutoff with the highest estimated coefficient.

For illustration, in the top panel of Figure 3.4, I present predicted BMI over a range of lowest observed childhood PIR for a data generating process in which all hypothetical subjects had a small probability of having an additional 10 BMI points, but those with a low PIR of 2 or below had an additional 70% probability. This could be taken as one interpretation of the conventional wisdom regarding income, poverty, and BMI, where in

Figure 3.4: Effect of child poverty on adult BMI: Simulation of optimal threshold search



general those at the lower end of the income distribution have higher BMIs. In the bottom panel, I present the resulting  $\beta_1$  estimates from the optimal threshold search described above applied to this hypothetical sample and identify the poverty threshold associated with the highest difference in BMI between those at or below the threshold and those above. The procedure finds a clear peak in terms of the poverty threshold best differentiating those with higher versus lower BMIs, with the largest difference between the poor and non-poor at a threshold of 1.9999. Those designated as poor by this threshold have, on the average, a BMI almost 29% higher than those above the threshold. In the simulated data, this puts those poor by this optimal definition at significantly higher risk of being obese ( $\text{BMI} \geq 30$ ).

Note that this threshold search is different than the usual variable selection problem in that I am trying to determine which single predictor out of many, rather than which subset of predictors, maximizes fit, for the basic analysis of the effect of ever experiencing childhood poverty net of covariates in (3.1). I am capitalizing, however, on multiple inferences and chance significance. The basic effect of poverty estimated here is thus the maximal relationship in this data and should be considered an upper-bound estimate of the effect of poverty defined using that poverty threshold. This analysis sets the groundwork for not only replication in other samples but also testing the resulting threshold in the dynamics models. Indeed, one underlying hypothesis of this work is that this measure—a simple dichotomous indicator of ever being in poverty—constitutes a weak model relative to models accounting for dosage or timing of poverty. Furthermore, recall that unlike other analyses we are trying to isolate the total effect of childhood poverty, not its effect over and above maternal BMI, adult SES factors, or other risks.

As described, the procedure to search for an optimal threshold to define absolute poverty tests the effect of all candidate poverty thresholds and finds the threshold with the highest effect. As with all analyses, this is accomplished across five multiply imputed datasets<sup>2</sup>. For finding the optimal absolute poverty threshold, this procedure examined 3157 unique

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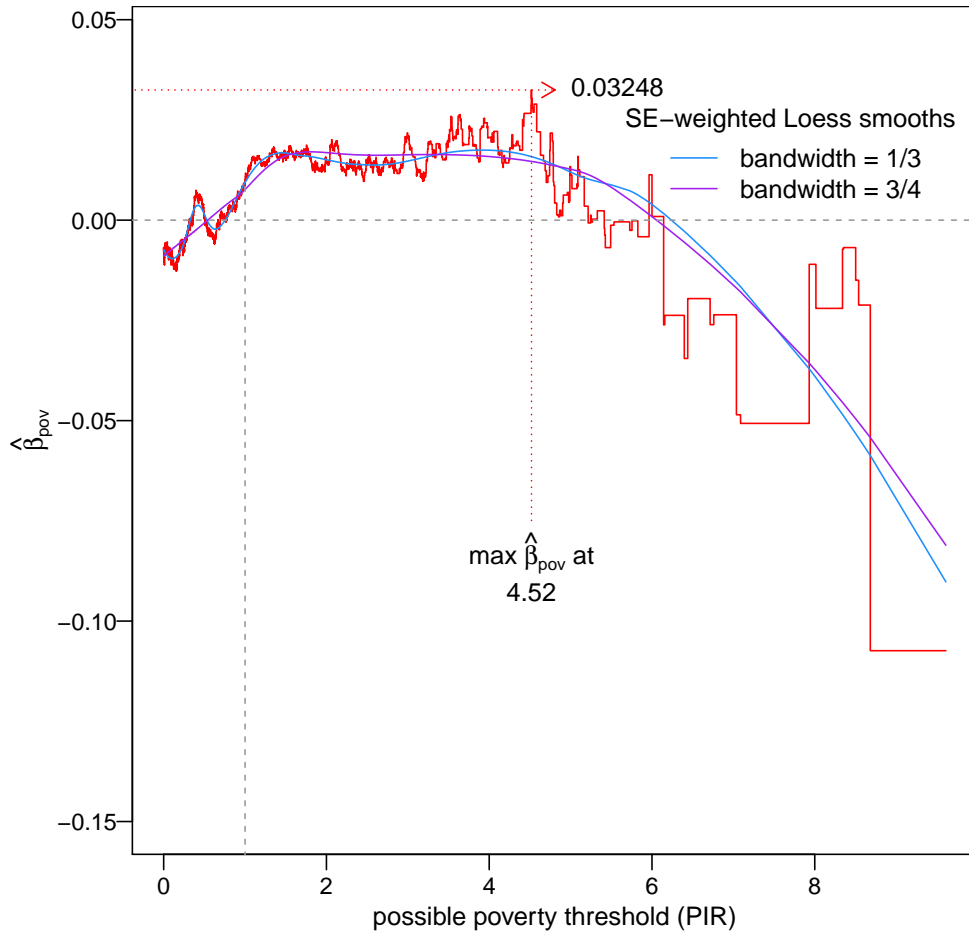
<sup>2</sup>Only the covariates are imputed. So as to not introduce smoothing of what are likely varied poverty experiences, income or poverty itself is not imputed.

lowest PIR values, ranging from 0 to 8.68. Note that for absolute poverty this analysis inherently examines the effect of the traditional (NLSY-defined) poverty level (i.e. a PIR of 1) and compares it to other possible thresholds. In fact, I found that being poor by the usual threshold was associated with a 9/10 of 1% increase in BMI net of the covariates.

Figure 3.5 plots the coefficient estimates for each of the 3157 candidate thresholds. The general trend of effect on the natural log of BMI is represented by two loess locally-weighted non-parametric smooths with spans of  $1/3$  and  $3/4$ . These smooths actually have two weights, with the inverse of the standard error associated with each estimated effect combined with the standard tri-cubic within-span weighting. The coefficients for extreme thresholds are obviously less stable than those in the middle, as very low thresholds will define almost everyone as never poor, and very high thresholds will define almost everyone as poor. This is more of an issue with high thresholds: There are 476 observations out of 5185 with a low observed childhood income of 0, and 484 with a low PIR of less than .001, while only 1 subject would be defined as not poor with the highest candidate threshold, and 8 (.15%) would be defined as “poor” at a threshold of 8. There are naturally more candidate thresholds in the lower end of the distribution than the higher end, as illustrated by the length of the steps in the figure.

While the usual threshold (represented by the dotted line at  $\text{PIR} = 1$ ) may be associated with a positive effect on later BMI, it is apparent that the effect of poverty status increases as the poverty threshold increases beyond 1. In fact, it appears that any threshold above 1 up to about 4.75 should produce a larger effect on later BMI than using the usual threshold. The maximum effect was observed for a threshold of 452% of the usual poverty line ( $\text{PIR} = 4.52$ ). Compared to adults whose childhood income never fell to or below 4.52 times the Federal poverty level, adults whose income did fail to exceed this level in at least one observed year had a BMI an average 3.3% higher, net of all covariates. This is obviously a high poverty threshold, which I explore further below.

Figure 3.5: Effect of poverty status on log BMI by absolute poverty threshold



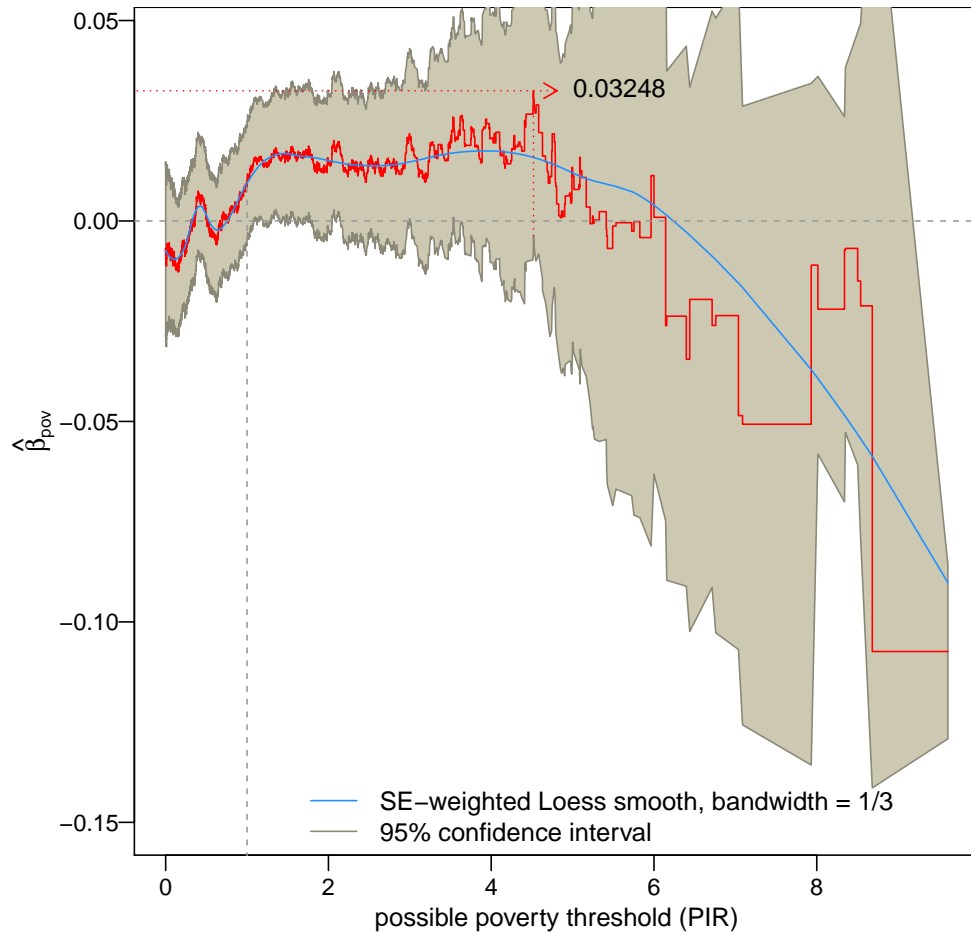
The loess smooths in Figure 3.5 indicate that the effect on adult BMI has an initial peak around 1.5 times the poverty level. The effect plateaus until a second peak in the range of 3 to 4 times the poverty level. While the estimated effects vary around this trend, this variation is generally  $\pm 1/10th$  of a percentage point around an average effect of an additional 1.5% in adult BMI. The maximum effect found here, while surrounded by a handful of similarly high effects, occurs in a range of candidate thresholds with a generally decreasing effect on later BMI. The maximum effect indicates that the 5102 sample members defined as poor by this threshold have a significantly higher average BMI, net of covariates, than the 1.6% with more privileged childhoods.

Given the likely imprecise estimates associated with a definition of “poverty” which assigns less than 2% of the sample to one category or the other, it may well be that our initial optimal threshold is not estimated to be significantly different than 0. Furthermore, we might be concerned as to whether the effect of such a high threshold is any different statistically than the estimated effect from a lower threshold more consistent with what we think of as poverty. Figure 3.6 below incorporates 95% confidence intervals for the coefficients (with just the more sensitive of the two loess smooths). Now we see that our initial optimal threshold is not significantly different than 0, but there are several lower thresholds (still above 1) for which the 95% confidence interval does not contain 0. Thus, rather than a clear peak in the average difference between those defined as poor and not poor by threshold seen in the simulation in Figure 3.4 (page 69), we have a plateau of effects in which a threshold of 400% of FPL accounts for essentially the same difference in later BMI as a threshold of 200% of FPL, and our highest effect appears to be an unstable product of random variation.

Our optimal threshold should come from a subset of poverty thresholds that produce estimated BMI increases significantly different than 0. In this sample, there are 380 observed low childhood PIR values, ranging from 1.082 times the usual poverty level to 3.54, that result in significant coefficient estimates. The highest estimated effect comes with a PIR of about 3.53. Defining poverty at this level creates two groups whose average BMIs differ by an estimated 2.62%, net of the covariates. As seen in Figure 3.7 below, this candidate threshold is in a range where the estimated effects are increasing, toward our original optimal threshold of 4.52, but also becoming more imprecise. A threshold of 3.53 leaves just 4.1% of the sample not poor. Because of this imprecision as well as correlation with the covariates, the highest proportion of variation in adult BMI accounted for by the poverty definition plus covariates (the highest  $R^2$ ) is found with a cutoff of about 1.35.

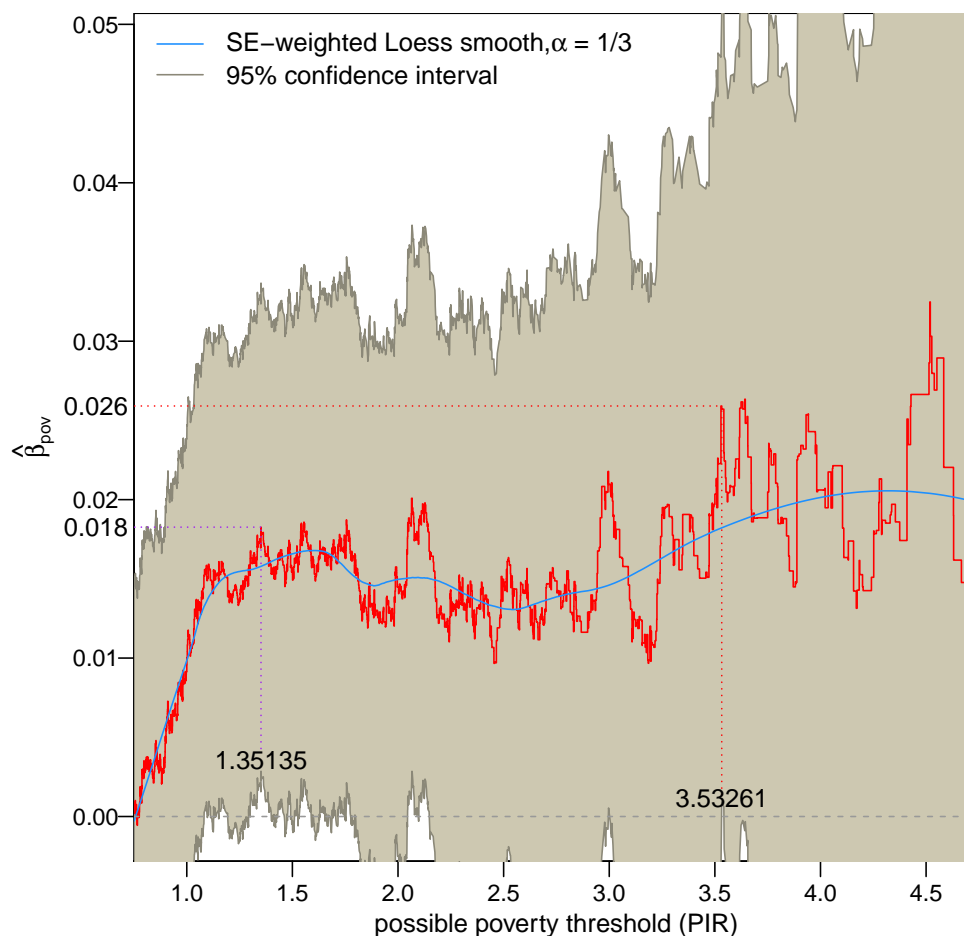
Defining poverty at a threshold of 1.35 results in a model explaining 11.64% of the variance in adult BMI, versus 11.58% with the higher threshold of 3.53. This new optimal threshold accounts for an average BMI 1.8% higher in those categorized as poor versus

Figure 3.6: Effect of poverty status on log BMI (with confidence intervals) by absolute poverty threshold, highlighting initial optimal threshold



those more fortunate. Again, while selecting on model fit likely provides an upper bound of the effect of poverty so defined, we will go on to test this effect in multiple models. Furthermore, note that selecting via  $R^2$  in this case is no different than selecting based on the Bayesian Information Criterion since all models have the same sample size and number of predictors. Note that 1.35 is a more satisfying poverty threshold than 3.53, and is similar to the eligibility cutoff specified for Medicaid expansion under the federal Patient Protection and Affordable Care Act (138% of poverty).

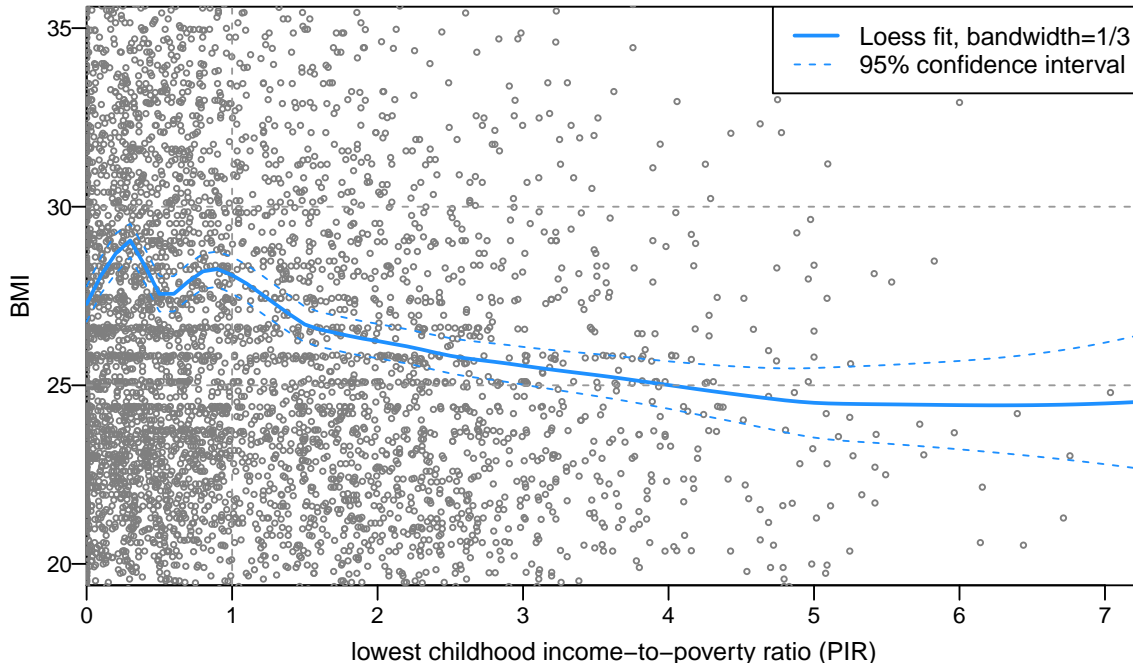
Figure 3.7: Effect of poverty status on log BMI (with confidence intervals) by absolute poverty threshold, comparing two possible optimal threshold choices



Selecting a threshold of 1.35 instead of 3.53 on the basis of accounting for an additional 0.06% of the variance in adult BMI demonstrates again the lack of a clear threshold—rather than a peak, we obtained a plateau. Although the hypothetical data in the simulation exhibited large variation in BMI at a particular level of income (see the top panel of Figure 3.4), the procedure was still able to reproduce the threshold of 200% of FPL. In the observed data, however, Figure 3.8 shows even more variation and even less of a threshold in the simple distribution of adult BMI as a function of lowest observed childhood PIR. The loess fit demonstrates not a large drop-off in BMI but rather a more modest decline from a PIR of

just below 1 to around 1.5, amounting to perhaps 2 BMI points. Furthermore, substantial variation or nonlinearity in the relationship between a marginally higher low PIR and adult BMI is apparent below 100% of FPL.

Figure 3.8: Bivariate relationship between adult BMI and lowest observed childhood income



### 3.4 Finding an optimal relative poverty threshold

Relative poverty is usually defined by whether the family income was less than or equal to some fraction of median family income (for the appropriate family size). This fraction is often 40% or 50%. The search for an optimal threshold to define relative poverty, then, uses the lowest observed childhood median income ratio (MIR), or the ratio of family income to median family income. Following Iceland (2003) and other relative poverty research, I obtained yearly estimates from the Current Population Survey of the median family income for a family of 4, converted these to real terms (2010 dollars), adjusted the medians by an equivalence scale consisting of the square root of family size (i.e.  $\div 2$ ) to convert them

to individual normalized median income<sup>3</sup>, and created the MIR for each observed year. The optimal threshold search procedure compares this value to each candidate threshold to define relative poverty. In this data, those under the 40% threshold have a BMI an average of 1.55% higher than those above this threshold, net of the covariates. The 50% definition creates an average increase in adult BMI of 1.78%. While both are statistically significant, a different threshold may produce a larger difference in adult BMI between those under and those above the threshold.

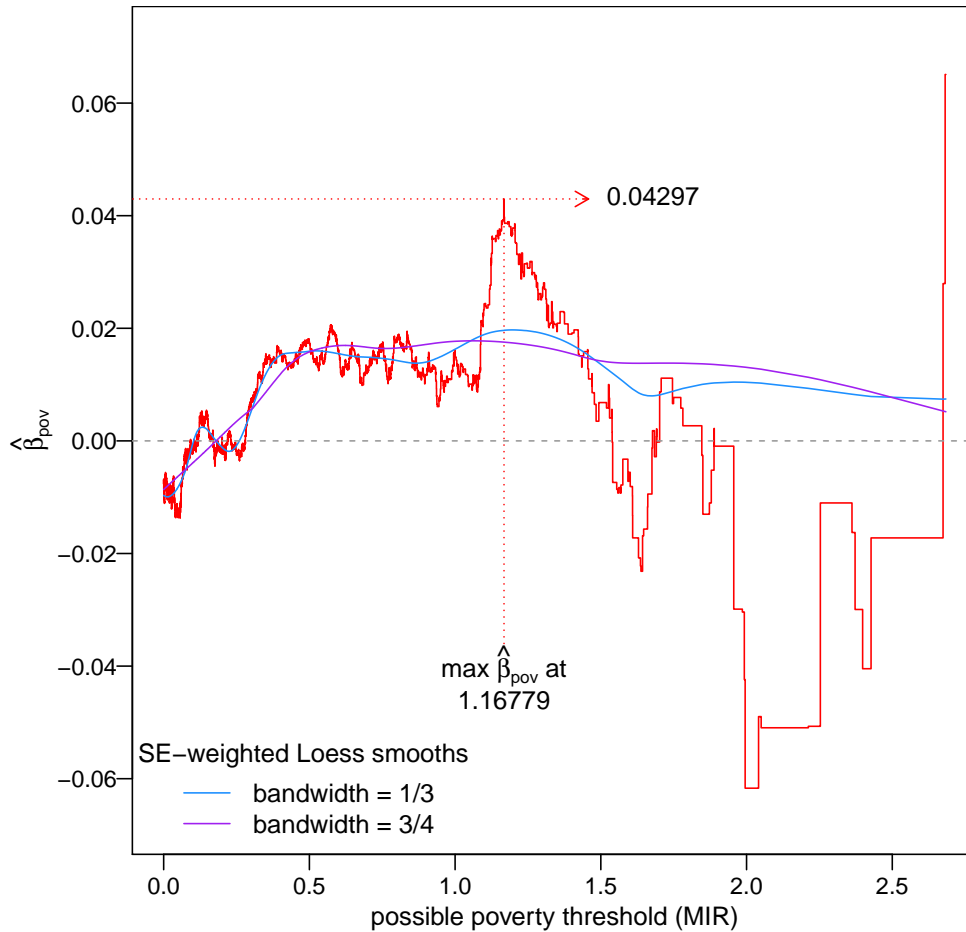
The search procedure tested each of 3190 unique observed values (from 0 to 2.68) to define poverty status and tested the effect of the poverty indicator. As seen in Figure 3.9 below, it was actually this highest value that had the highest estimated effect. As noted, such a result is likely to be highly unstable, besides not being reflective of “poverty.” It is also not consistent with the general trend of effects, which steadily decrease from a peak at around 1.168.

Unlike the clear peak in the simulation, the loess smooths show that there is a false peak (to continue the mountaineering metaphor) in effects around 45% of median (for the more responsive smoother) to 60% of median income (for the smoother loess). Immediately above this initial peak, the estimated effects are generally flat or decrease slightly before climbing again above 100% of median income. The higher-bandwidth smooth shows little difference in the effect on later BMI for relative poverty status defined by thresholds anywhere between 0.5 and 1.5. The more responsive smoother finds a maximum near the aforementioned peak around 1.168 times the normalized median income for the appropriate year. This peak in effects by relative poverty definition appears to be closer to the demonstration with simulated data in Figure 3.4 (page 69) than in the optimal absolute threshold search. Taking a relative poverty definition of having income less than or equal to 116.8% of the median results in an estimated 4.39% higher BMI, on average, for those so designated as

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<sup>3</sup>A common method of normalizing income to account for the different needs of households of different sizes is to divide the income by household size raised to some fractional power (Coulter, Cowell, & Jenkins, 1992; Cowell & Mercader-Prats, 1999). As Coulter and colleagues note, it appears to be most common to use .5, i.e. to divide income by the square root of household size. For example, Iceland (2003) used the square root to scale relative poverty measures.

Figure 3.9: Effect of poverty status on BMI by relative poverty threshold

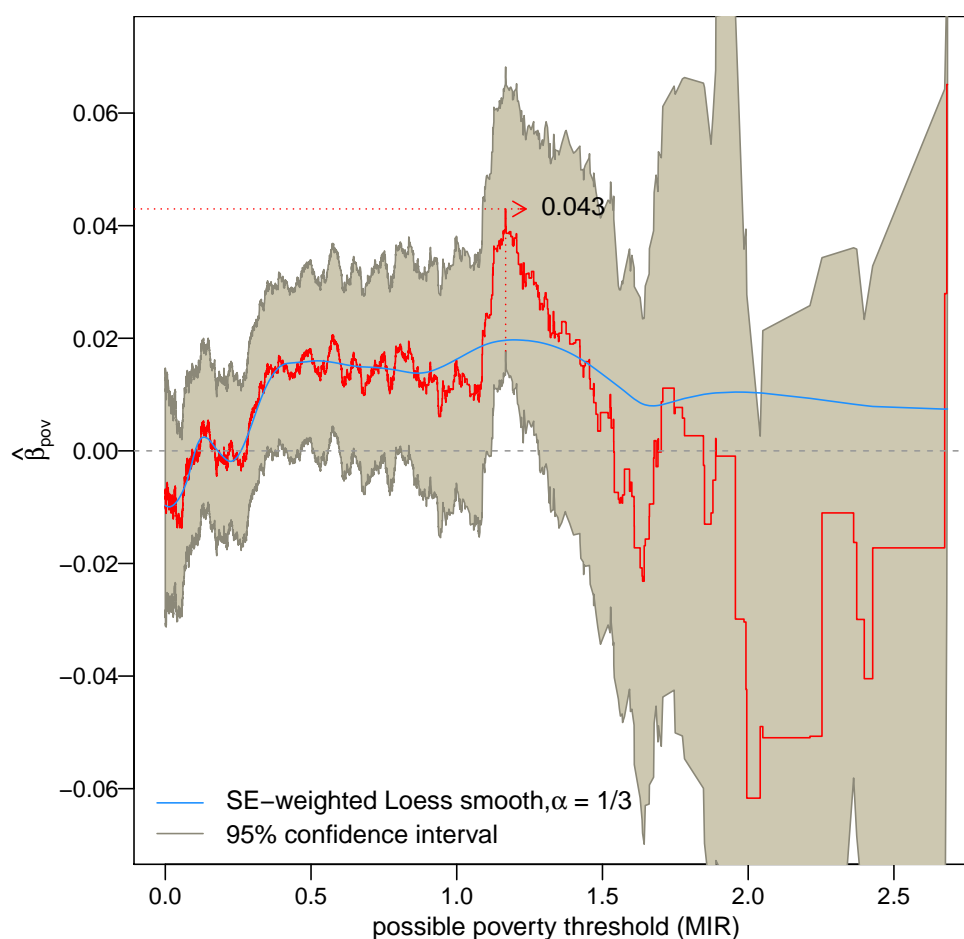


poor during childhood than those not. This is our initial optimal threshold candidate, based on the search for the highest estimated effect on adult BMI.

This initial optimal relative poverty threshold is high and may provide an unstable estimate of BMI differences between those below and above this cutoff, as we saw with the initial absolute poverty threshold. In Figure 3.10 I include 95% confidence intervals for the  $\beta$  estimates. Now we see that the 1.168 threshold is in the middle of a stretch where all thresholds between 1.1202 and 1.2809 are statistically significant, even though they define between 4963 and 5052 of the 5185 sample members as “poor.” This stretch of significant thresholds implies that moving above 1.12 times median income consistently moves

high BMI individuals from above to below the threshold, up to 1.168. In other words, this sample apparently has a significant handful of people who grew up in relatively affluent households (keeping in mind that this analysis is based on the *minimum* observed ratio of family income to median family income) with high BMIs.

Figure 3.10: Effect of poverty status on log BMI (with confidence intervals) by relative poverty threshold, highlighting initial optimal threshold



There are 354 candidate relative poverty thresholds that account for differences in BMI significantly greater than 0. These range from 0.350 to 1.282 times normalized median income. Although the 1.168 threshold accounts for the largest increase in BMI *and* the most variance in BMI (i.e. the highest  $\beta$  and the highest  $R^2$ ), our set of ideal candidates

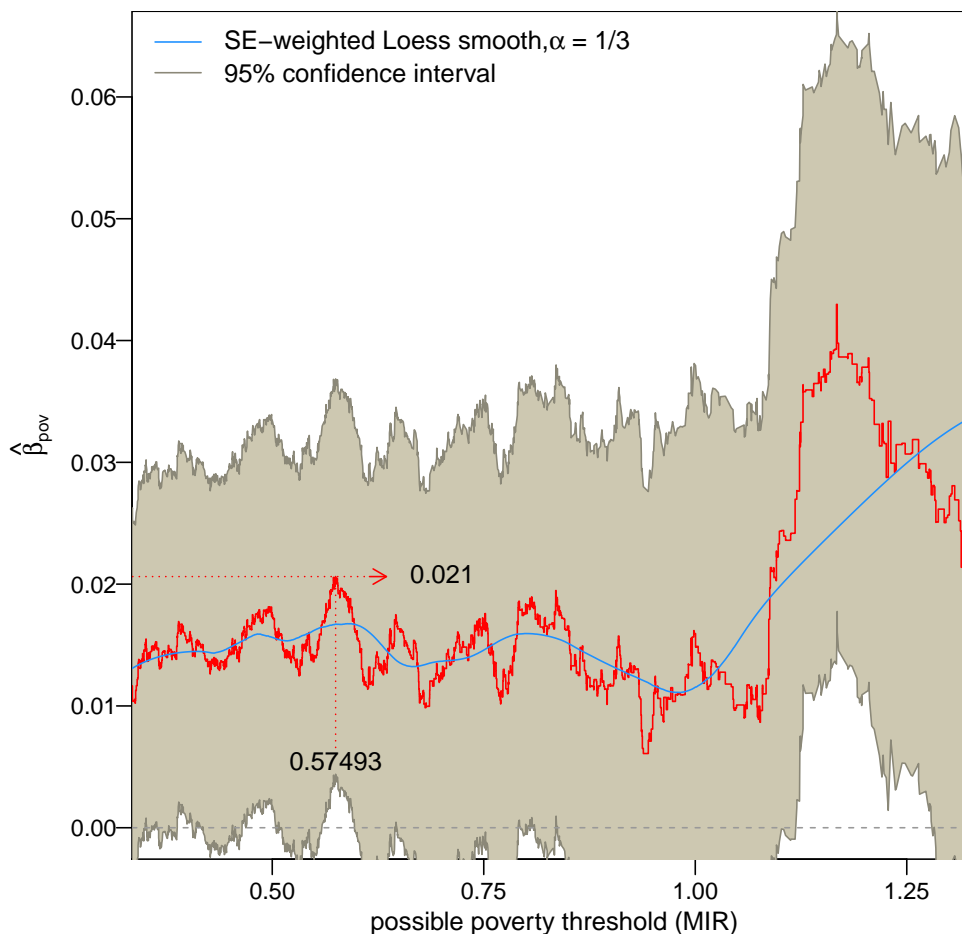
should really be those values below 1 times the median that explain a significant difference in adult BMI. The construct of relative poverty is intended to capture those unable to afford the median standard of living, so while a cutpoint of 1.168 accounts for the most variance in adult BMI, this is not a feasible poverty definition for research or practice, however interesting this finding might be.

Restricting our search to significant thresholds below 1 (100% of relevant median income), there are 284 such candidates. Turning to Figure 3.11, we see that the candidate in this subgroup that accounts for the largest difference in adult BMI is 57.5% of median income. Those so defined as poor have adult BMIs an average of 2.08% higher than those whose lowest childhood income never fell to 57.5% of median income or lower. Using a simple dichotomy of ever in poverty defined by this threshold in the full model (3.1) accounts for 11.65% of the variance in adult BMI.

### ***3.5 Exploring the poverty definitions***

The two optimal thresholds were estimated empirically in the context of a simple dichotomous model of whether the adult was ever in poverty as a child. This simple dichotomy likely masks multiply varied effects of poverty which we will investigate via the timing, accumulation, and hybrid models. A poverty definition “optimal” in the dichotomous sense may not be best within a model accounting for poverty dynamics. I leave for future efforts a more flexible optimal threshold search within a poverty dynamics model and here simply compare the two optimal threshold definitions with the usual NLSY-defined poverty and two practice definitions corresponding to thresholds often used for program eligibility, 200% and 185% of FPL. Before moving on to the rest of the analyses, let us take a look at how the different poverty definitions are distributed in the sample. I present kernel density and box plots of the distribution of the proportion of all childhood years spent in poverty by each definition, in Figure 3.12, along with the proportion of the sample never in poverty by that definition and the proportion in poverty every observed year.

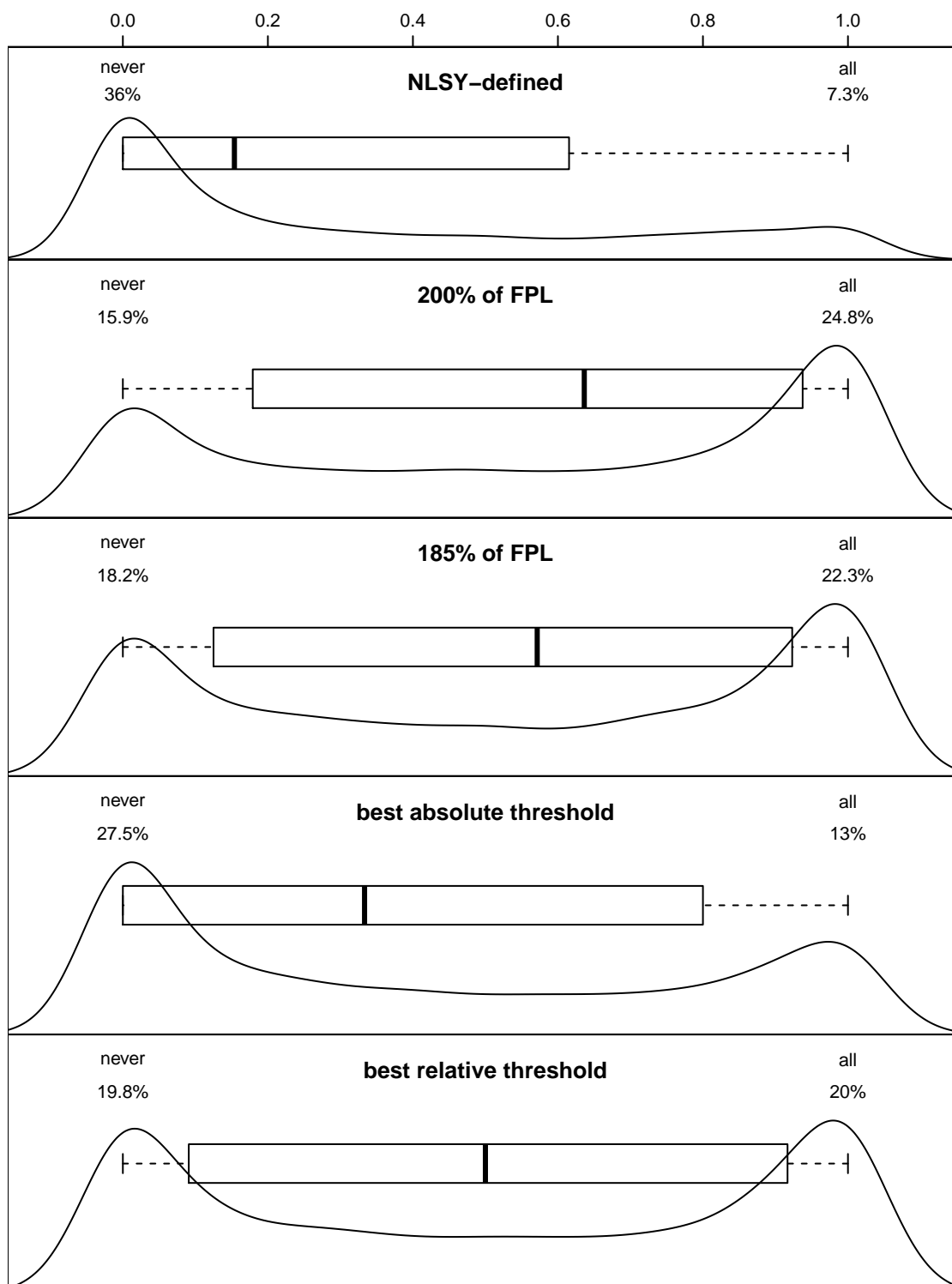
Figure 3.11: Effect of poverty status on log BMI (with confidence intervals) by relative poverty threshold, highlighting final optimal threshold



The new best absolute threshold of about 135% of FPL is, of course, somewhere between the usual definition (100% of FPL) and the common practice threshold of 185% of FPL in terms of the proportion of the sample never poor during childhood, the proportion whose entire childhood was spent under the threshold, and the median proportion of childhood spent poor. The optimal relative threshold is, in turn, between the best absolute threshold and the 185% threshold. The optimal relative threshold represents a value of \$36,415.23 for a family of four in 1985, in 2010 dollars, while poverty level for a family of four in 1985 was \$22,312.24. The optimal absolute threshold is thus \$30,152.76 for the same situation,

while 185% of poverty is \$41,277.64. We essentially have a nice array of poverty definitions associated with different levels of family income with which to compare models connecting poverty dynamics in childhood to future BMI: NLSY-defined poverty, the optimal absolute threshold, the optimal relative threshold, 185% of FPL, and 200% of FPL.

Figure 3.12: Distribution of proportion of childhood in poverty by poverty definition



## 3.6 Modeling results

### 3.6.1 OLS models

This analysis is essentially comprised of a competition among poverty definitions and models of poverty dynamics, in the service of discerning what it is about poverty that may result in excess adiposity. I will briefly focus on effects, but most of the focus here is on model fit and the pattern of effects. In the first row of Table 3.1 I include the estimates from the basic model of the effect of ever being in poverty, which essentially replicates the optimal threshold searches above. In the second and third panels, I present the estimates for the timing models with both four periods and three periods, respectively. In the four period model, ever having an income  $\leq$  185% FPL in early childhood is significantly positively related to later BMI, net of ever being in poverty in the other three periods (and the covariates). The primacy of early childhood under this definition is confirmed in the three period model, where the 0-to-3 period is combined with poverty experiences during ages 4 and 5. With the additional precision, the 0-to-5 period becomes significant under the 200% FPL definition, and middle childhood becomes significant under the optimal absolute threshold definition. The proportion of all childhood years (observed, with a minimum of 3 required observations) spent in poverty under the three highest definitions (optimal relative, 185% FPL, and 200% FPL) is also significantly related to adult BMI.

All significant coefficients in the OLS models indicate a positive effect of (more) poverty on later BMI. As far as the timing of poverty experiences, the indicator of any poverty within a period was significant four times, three for early childhood (including 4–5) and once for middle childhood, but note that this is out of 35 chances. In this analysis, it is clear that poverty during adolescence appears to have little effect. The hybrid model, assessing proportion of each (of three) periods in poverty, never found significant effects for poverty. Finally, the accumulation model found a significant effect for three of the five definitions.

Table 3.1: Effects of poverty measures on lnBMI, full OLS models

Model	Effect	NLSY-defined	Optimal absolute	Optimal relative	185% FPL	200% FPL
Dichotomous	Ever	0.010 (0.008)	0.018 (0.008)*	0.021 (0.008)*	0.012 (0.009)	0.013 (0.009)
	0 to 3	0.005 (0.009)	0.013 (0.009)	0.016 (0.009)	0.022 (0.009)*	0.017 (0.009)
	4 to 5	-0.012 (0.01)	0.003 (0.01)	0.01 (0.009)	0.011 (0.009)	0.015 (0.009)
Timing, 4 pd.	6 to 11	0.007 (0.009)	0.017 (0.01)	-0.001 (0.009)	0 (0.009)	-0.003 (0.009)
	12 to 17	0.009 (0.009)	-0.014 (0.009)	-0.004 (0.009)	-0.011 (0.009)	-0.003 (0.009)
	0 to 5	-0.005 (0.009)	0.01 (0.009)	0.018 (0.009)	0.022 (0.009)*	0.021 (0.009)*
Timing, 3 pd.	6 to 11	0.007 (0.009)	0.018 (0.009)*	0.006 (0.009)	0.007 (0.009)	0.004 (0.009)
	12 to 17	0.007 (0.009)	-0.012 (0.008)	-0.004 (0.008)	-0.008 (0.008)	-0.002 (0.008)
Accumulation	Prop. childhood	0.01 (0.013)	0.018 (0.012)	0.023 (0.011)*	0.024 (0.011)*	0.024 (0.011)*
	0 to 5	-0.006 (0.014)	0.009 (0.013)	0.015 (0.013)	0.016 (0.013)	0.012 (0.013)
Hybrid model	6 to 11	0.006 (0.014)	0.024 (0.013)	0.02 (0.013)	0.022 (0.013)	0.016 (0.012)
	12 to 17	0.008 (0.012)	-0.019 (0.012)	-0.011 (0.011)	-0.013 (0.011)	-0.003 (0.011)

Note: \* p < .05. Cluster-robust, multiple-imputation-corrected standard errors in parentheses. All models control for age, year, demographics, mother's background characteristics, etc. See text for full covariate list.

Only the 185% of FPL definition appears to have resulted in significant findings for the effect of poverty across the sample beyond chance. The usual definition was never significant across the four models. The two alternate definitions were each significant in two models, but one of these was the dichotomous poverty model in which the optimal thresholds were selected. In Table 3.2 I present model fit statistics across the five tested models and five definitions of poverty, including overall model Bayesian Information Criterion scores and the change in BIC associated with the poverty parameter(s). For the models with a single poverty parameter, we get different rankings of the models when comparing  $R^2$  and model BICs due to different sample sizes. The  $R^2$  always prefers the accumulation model over the dichotomous model within definitions, but BIC prefers the dichotomous model with the optimal absolute threshold over the accumulation model. The change in BIC for the poverty parameter—i.e. the difference in BIC for models with and without the poverty measure—strangely prefers the simple dichotomy for all but the practice definitions (185% and 200% of FPL) in terms of doing the least harm to model fit (as all are positive).

Across the developmental models (the simpler dichotomous within periods—presented above for early childhood and preschool years separate and combined—and the hybrid accumulation within periods), the best fitting model appears to be the hybrid model under the 185% of FPL definition: It is the only model among those with three poverty parameters (three age groups) to have an  $R^2$  above 12% (the 4-period timing models often do so thanks to the extra parameter) and the three poverty parameters as a group do the least harm to model fit. Inherent in the timing hypotheses, however, is the idea that one period may matter more than the others, but we saw that no period under the hybrid model was significant net of the others. This contrast may be due to mixed effects of accumulation in different periods for different groups. The 185% of FPL definition also had the best model fit in the accumulation models, marginally. The BIC for the latter is of course much better than for the hybrid model thanks to the penalized extra parameters of the hybrid model.

While the accumulation and hybrid models using the 185% of FPL poverty definition exhibit marginally better fit, the estimated effects of poverty experiences are small. In the

Table 3.2: Model fit by poverty dynamics model and definition, full OLS models

Model	n	Fit measure	NLSY-defined	Optimal absolute	Optimal relative	185% FPL	200% FPL
Dichotomous		$R^2$	0.116	0.118	0.116	0.116	0.116
	5185	BIC	-414.694	-420.710	-419.833	-414.764	-415.035
		BIC poverty	6.393	2.078	1.255	6.323	6.052
Timing, 4 pd.		$R^2$	0.120	0.121	0.121	0.121	0.121
		BIC	-285.224	-292.558	-289.498	-293.299	-291.966
	4134	F poverty	0.892	2.716*	1.954	2.900*	2.568*
		BIC poverty	29.716	22.381	25.442	21.640	22.973
Timing, 3 pd.		$R^2$	0.118	0.120	0.119	0.120	0.120
		BIC	-335.075	-342.062	-339.686	-342.428	-340.894
	4529	F poverty	0.743	3.060*	2.271	3.181*	2.672*
		BIC poverty	23.012	16.025	18.402	15.660	17.193
Accumulation		$R^2$	0.117	0.117	0.118	0.118	0.118
	5139	BIC	-415.097	-417.538	-420.426	-420.710	-420.497
		BIC poverty	7.691	5.250	2.362	2.078	2.291
Hybrid model		$R^2$	0.118	0.120	0.120	0.120	0.119
		BIC	-334.062	-340.883	-341.967	-343.575	-340.422
	4529	F poverty	0.407	2.669*	3.028*	3.562*	2.516
		BIC poverty	24.025	17.204	16.120	14.513	17.665

*Note:* \*  $p < .05$ . F poverty and BIC poverty indicate F-statistic and change in BIC for addition of poverty measure(s) to base model with no poverty measure(s). Lower BIC values indicate better fit; negative BIC poverty values would indicate better fit with the poverty measure(s).

accumulation model, spending one's *entire* childhood below 185% of FPL is associated with an adult BMI 2.4% higher than someone never below 185% of FPL. In that same model, this is roughly equivalent to the estimated BMI difference for white women versus white men (2.4% higher, *ceteris paribus*). A single additional year of age accounts for an additional 1.5% higher BMI net of the other covariates, so the effect of an entire childhood in poverty versus no year in poverty is smaller than the estimated BMI growth associated with being two years older. Breaking up childhood into developmental stages in the hybrid model, spending all of early childhood and the pre-school years (0 to 5) below the 185% of FPL threshold is associated with a BMI 1.6% higher than someone never poor in this period, controlling for poverty in the other periods and the covariates. Spending all of middle childhood in poverty is associated with a BMI 2.25% higher than someone not below this threshold ages 6 through 11. Again, neither of these marginal effects were statistically significant. Combining them implies an a BMI 3.8% higher for someone poor from age 0 through age 11 versus someone never poor, which is slightly higher than the protective effect of having a foreign-born mother (3.6% lower BMI) and about 60% of the effect of being a black female versus a white male (6.4% higher BMI). Thus, while being poor from 0 through 11 might push someone from a BMI of 25.00 to 25.95, never having been poor but being 29 instead of 25 years of age might push that BMI from 25.0 to 26.5.

While there is some evidence that the timing of poverty is important, and some evidence that the accumulation of poverty is what matters, there is little evidence here (pooled across genders and ethnicities) that a higher proportion of, say, early childhood spent in poverty is predictive of significantly higher BMI. In addition, there appears to be no clear threshold under which children are strongly at risk of increased BMI in adulthood. This lack of a clear threshold in the analysis of whether the adult was ever under the threshold in childhood—the plateau of effects in Figures 3.5 and 3.9—likely contributed to the lack of a clear winner among models. More importantly, considering only whether a child was ever in poverty masks a variety of dynamics—metabolic programming, cumulative stress, development of diet and exercise habits—that are only partly captured by a single dichotomy. It is likely that the accumulation of low income experiences that contribute to adiposity, for example,

are experienced at a higher level of income—below a higher threshold—than the threshold that captures the most difference in BMI in the dichotomous model. As we saw, the higher 185% of FPL definition appeared to perform better in the poverty dynamics models than the optimal definitions, but in the dichotomous model this poverty indicator was not significant. Finally, note that we are trying here to capture the effect of poverty as a risk factor across the whole sample, averaging what might be different reactions to poverty for different groups.

### *3.6.2 Fixed effects models*

Despite capturing background covariates likely related to both socioeconomic conditions during childhood and adiposity, there are likely numerous other factors associated with both income and BMI, from genetic predispositions to motivations to time preferences, that may bias estimates of the effect of poverty on BMI. One common way to deal with such unexplained heterogeneity is to implement a fixed effects model in which a grouping indicator decomposes the error variance into an idiosyncratic portion and a portion attributable to unobserved constant (across the group, be it BMI observations within an individual or individuals within a family) factors. Such fixed effects are often employed in obesity research to control for unobserved propensity towards obesity. For example, Eid and colleagues (Eid, Overman, Puga, & Turner, 2008) replicated the usual associations between measures of residential sprawl and mixed-use land use in subjects' neighborhoods and BMI in OLS models. Exploiting the panel nature of their data (NLSY) via individual fixed effects (first differencing), however, eliminated these neighborhood effects: "Our results strongly suggest that neither residential-sprawl nor a lack of mixed-use causes obesity in men or women, and that higher obesity rates in 'sprawling' areas are entirely due to the self-selection of people with a propensity for obesity into these neighborhood" (p. 387). Closer to the current study, Liu (2007) examined an accumulation model of the effect of earlier childhood poverty on obesity among children in the NLSY Child and Young Adult sample using sibling fixed effects and concluded "that the results obtained via naive probit or linear probability regressions might be biased downward. In particular, after controlling for unobserved heterogeneity, we find that extensive poverty exposure increases a child's risk of being obese" (p. 18).

As noted, implementing family or sibling fixed effects (FE) to control for unobserved, time-invariant heterogeneity in family-level propensity towards poverty and/or adiposity relies on between-sibling differences in poverty experiences. Twins cannot be included. Differences are more likely in the developmental models and the accumulation model than in the dichotomous model: It is more likely for siblings to differ on the number of years of early childhood spent in poverty than on whether they were ever poor during their (presumably) overlapping childhoods. In fact, the number of families present within which there is any variation in dichotomous childhood poverty status ranges from 135 for the most stringent definition of poverty, the NSLY designation, to only 93 for the highest threshold, 200% of FPL. Thus, I do not consider the basic dichotomous models in the fixed effects analyses. Other factors that do not differ within families are excluded from the model, such as the mother's relatives' education and her age at her first birth. With this data, this also includes "main" effects of ethnicity, as the ethnicities of children in the NLSY are determined by those of their mothers. For some of the resulting models—specifically, all but the accumulation models—the indicator of being an Asian American or Pacific Islander female is also dropped due to lack of variation among siblings with complete data.

As differences in estimated effects of the poverty measures between the OLS models above and the FE models could be due to the latter being estimated only among families with more than one child, for comparison I present in Tables 3.3 and 3.4 the full OLS models re-estimated on a subset of subjects who have siblings represented in the data. Although this restricts the OLS sample to a sample similar to that used in the FE analyses, eliminating singletons, the effect of the poverty measures are, of course, not estimated using the same information. This does create differences with the full OLS models above, indicating a potentially larger effect among only children (or at least adults whose siblings were not included in the NLSY) or a larger effect among multiple-child families.

In the four period timing model, for example, the early childhood period was previously significant under the 185% of FPL definition, where the estimate was 0.022, but not in the restricted sample, where it drops to 0.007. Accounting for fixed family factors, this

Table 3.3: Effects of poverty measures on lnBMI, OLS vs. FE estimates, timing models

Model	Effect	Estimator	NLSY-defined	Optimal absolute	Optimal relative	185% FPL	200% FPL
0 to 3	OLS	-0.004 (0.011)	0.002 (0.012)	0.001 (0.011)	0.007 (0.011)	0.007 (0.011)	0.007 (0.011)
	FE	0 (0.014)	-0.003 (0.015)	-0.006 (0.016)	0.003 (0.016)	-0.001 (0.016)	-0.001 (0.016)
4 to 5	OLS	-0.008 (0.012)	0.012 (0.012)	0.02 (0.011)	0.016 (0.011)	0.016 (0.011)	0.016 (0.011)
	FE	-0.021 (0.015)	-0.001 (0.014)	0.009 (0.013)	0.013 (0.013)	0.014 (0.013)	0.014 (0.013)
6 to 11	OLS	0.003 (0.011)	0.019 (0.012)	-0.005 (0.012)	-0.001 (0.012)	-0.002 (0.012)	-0.002 (0.012)
	FE	-0.021 (0.018)	-0.013 (0.019)	-0.028 (0.019)	-0.019 (0.017)	-0.005 (0.018)	-0.005 (0.018)
12 to 17	OLS	0.021 (0.011)	-0.013 (0.011)	-0.003 (0.011)	-0.01 (0.01)	-0.002 (0.01)	-0.002 (0.01)
	FE	-0.034 (0.018)	-0.037 (0.019)*	-0.022 (0.018)	-0.029 (0.018)	-0.021 (0.018)	-0.021 (0.018)
0 to 5	OLS	-0.014 (0.011)	0.006 (0.011)	0.008 (0.011)	0.011 (0.011)	0.015 (0.011)	0.015 (0.011)
	FE	-0.002 (0.016)	0 (0.017)	-0.017 (0.017)	-0.012 (0.017)	-0.007 (0.018)	-0.007 (0.018)
Timing, 3 pd. 6 to 11	OLS	0.007 (0.01)	0.023 (0.011)*	0.008 (0.011)	0.01 (0.01)	0.007 (0.011)	0.007 (0.011)
	FE	-0.012 (0.017)	-0.011 (0.018)	-0.027 (0.018)	-0.021 (0.016)	-0.011 (0.017)	-0.011 (0.017)
12 to 17	OLS	-0.03 (0.01)	-0.03 (0.01)	-0.03 (0.01)	-0.03 (0.01)	-0.031 (0.01)	-0.031 (0.01)
	FE	-0.025 (0.017)	-0.038 (0.018)*	-0.022 (0.017)	-0.026 (0.017)	-0.024 (0.016)	-0.024 (0.016)

*Note:* \*  $p < .05$ . Cluster-robust, multiple-imputation-corrected standard errors in parentheses. OLS models estimated only on sibling groups. All FE models control for covariates that vary within sibling groups with complete data for that model.

Table 3.4: Effects of poverty measures on lnBMI, OLS vs. FE estimates, accumulation and hybrid models

Model	Effect	Estimator	NLSY-defined	Optimal absolute	Optimal relative	185% FPL	200% FPL	
Accumulation	Prop.	OLS	0.015 (0.015)	0.027 (0.014)*	0.025 (0.013)	0.027 (0.013)*	0.026 (0.013)	
		FE	0.022 (0.009)*	0.022 (0.009)*	0.022 (0.009)*	0.022 (0.009)*	0.022 (0.009)*	
	0 to 5	OLS	-0.014 (0.017)	0.009 (0.016)	0.012 (0.016)	0.011 (0.016)	0.007 (0.015)	
		FE	-0.033 (0.03)	-0.029 (0.026)	-0.02 (0.025)	-0.01 (0.025)	-0.021 (0.026)	
	Hybrid model	6 to 11	OLS	0.001 (0.016)	0.031 (0.016)	0.019 (0.015)	0.021 (0.015)	0.014 (0.015)
			FE	-0.043 (0.025)	-0.045 (0.029)	-0.029 (0.029)	-0.014 (0.028)	-0.009 (0.027)
12 to 17		OLS	0.026 (0.015)	-0.018 (0.014)	-0.005 (0.013)	-0.005 (0.013)	0.007 (0.013)	
		FE	-0.023 (0.024)	-0.064 (0.026)*	-0.029 (0.024)	-0.015 (0.024)	-0.019 (0.024)	

Note: \* p < .05. Cluster-robust, multiple-imputation-corrected standard errors in parentheses. OLS models estimated only on sibling groups. All FE models control for covariates that vary within sibling groups with complete data for that model.

effect decreases further towards 0. In the fixed effects analysis, the only significant finding in the four period timing model is for being below the optimal absolute threshold during adolescence, the effect of which is approximately three times larger than in the OLS models with both the full and the reduced sample. Similarly, where any poverty in the combined early childhood period (0 to 5) under the two practice thresholds was significantly predictive of later BMI in the whole sample, and the optimal relative threshold indicator approached significance ( $t = 1.95$ ), these effects were smaller among only sibling groups and become negative and non-significant when accounting for family-level propensity towards higher BMI. The positive relationship between being poor during middle childhood under the optimal absolute threshold also becomes negative in the fixed effects models. The effect of poverty in adolescence as defined by the optimal absolute threshold is again significantly negative (and approximately the same size in both the three period and four period models) in the FE models. This significant protective effect of adolescent poverty holds for the hybrid model, but again only for the optimal absolute threshold definition.

In the full OLS models, the accumulation of poverty was significant under the optimal relative threshold and the two practice definitions. The strength of this model appears to be confirmed in the FE models, where the effect of the proportion of childhood spent in poverty is remarkably consistent across the definitions. This analysis does not consistently confirm Liu's (2007) conclusion that "naive" estimates underestimate the effect of child poverty on later adiposity (here BMI in adults, there obesity in children). Accounting for family factors influencing BMI, the estimate of the effect of the proportion of childhood poor under the usual NLSY definition increases by about 129%, and the estimate for the optimal absolute definition increased by about 27%. The other three estimates decreased by 10% or less from the full sample OLS estimates. Part of the large increase is due to differences between singletons and sibling groups, and the FE estimates are fairly close to the OLS estimates among sibling groups. Regardless of the definition, those who spent all of their childhoods in poverty have BMIs around 2% higher than those who were never poor.

These results are robust to a linear specification (i.e. with untransformed BMI as the outcome) but somewhat less likely to be significantly different than 0 (not shown). Only the proportion of (all) childhood years spent in poverty was significantly associated linearly with adult BMI, although the protective effect of adolescent poverty under the optimal absolute threshold neared significance. The accumulation model predicts an additional 0.65 BMI points for an adult who spent all of his or her childhood in poverty versus one never poor for all poverty definitions.

While the developmental models, whether measuring any poverty or the proportion of the period spent in poverty, found a consistent effect only for adolescence, this effect was negative. In fact, a remarkable result of the FE models of period effects is that any positive effect found in the OLS models becomes negative when accounting for fixed family factors. Furthermore, *every single estimate* is negative. Even if not indicating a significantly heightened risk for poverty in that period, the consistency of this finding is striking, especially when judged against the *positive* effect estimated for the proportion of all childhood. Put another way, the accumulation model indicates more poverty results in a higher BMI, but every other FE model indicates a lower BMI with more poverty.

Taking the optimal absolute threshold definition, which as seen in Table 3.5 was the best performing definition in the timing and hybrid developmental models and a marginal second (to the deeper NLSY poverty threshold) in the accumulation model, let us further explore this apparent conundrum. Spending any part of adolescence in poverty has a protective effect on later BMI. The hybrid model further indicates that, while not statistically significant, spending more of early childhood (0 to 5) and middle childhood in poverty also lowers adult BMI. While poverty within periods appears to have a protective effect, the accumulation of poverty experiences across all of childhood is associated with a higher BMI. In fact, hybrid models with any combination of the proportion of the developmental periods—any one, two, or three of the periods—using this definition results in all the estimates being negative (not shown). Spending all of adolescence at or below about 135% of FPL is associated with an adult BMI about 6% lower than someone never poor, holding constant the proportion of

the other periods poor. Spending all of childhood poor, compared to someone never poor, is associated with a BMI about 2% higher according to the basic accumulation mode, but almost 14% lower according to the hybrid model. The hybrid model is estimated on 3328 siblings in 1398 families, while the accumulation model is estimated on 3894 observations in 1617 families, with the additional sibling groups allowing for inclusion of an indicator for Asian/Pacific Islander female (a tiny portion of the sample). Estimating the accumulation model on the same sample as the hybrid model finds the estimates are highly robust (0.20 or 0.21 for all definitions) to these differences.

The diverging predictions for someone with a high proportion of childhood spent in poverty between the accumulation and hybrid models suggests, again, some nonlinearity in the effect of the proportion of childhood spent poor. The within-period (hybrid) model is estimating the effect of more poverty within the period, holding the other periods constant, which could be constantly high, low, etc. The average effect across the whole sample for more poverty within a period may be negative, but the effect of high proportions of poverty might still be positive among the subgroup so afflicted. Preliminary analysis of nonlinearity in the effect of the proportion of childhood spent under NLSY-defined poverty showed a potential small differential effect of proportion of NLSY-defined poverty above 50% versus below. There was also some indication of further nonlinearity below .5, but that may be accounted for by breaking poverty accumulation out by periods.

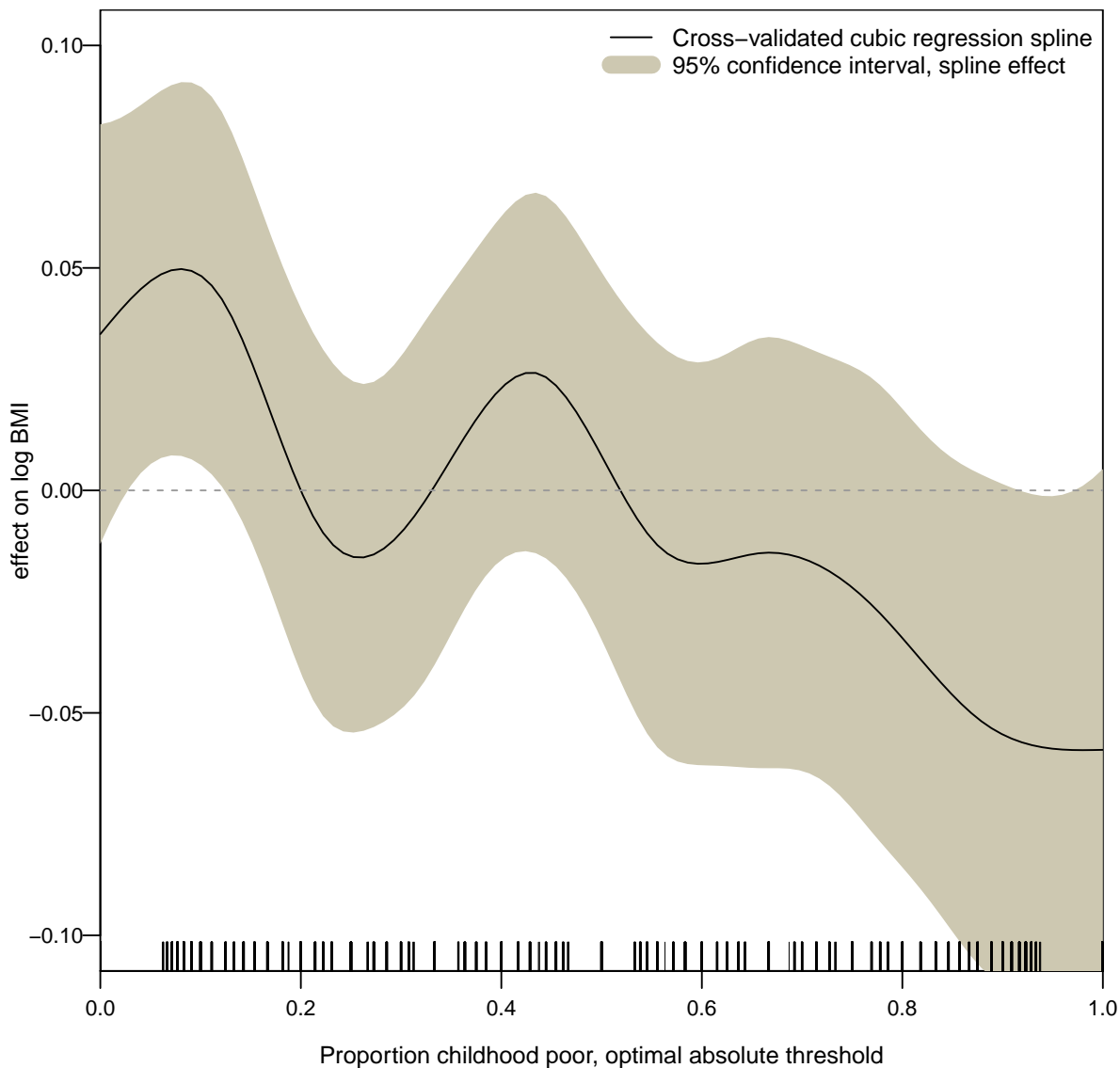
To explore and visualize this, in Figure 3.13 I present the estimated marginal effect for the proportion of childhood spent under the optimal absolute threshold using a generalized additive model (GAM) with a cubic spline for the effect of interest. This GAM includes linear additive terms of the same covariates as the FE models plus, to make it as comparable as possible, dummy variables for each mother, and was estimated on a sample without singletons. The rug plot at the bottom indicates the approximate location of sample members on the proportion of childhood poor. The spline effect indicates that, while there is some nonlinearity below around 0.45, the general trend is downward, with marginally more of childhood spent in poverty associated with a marginally lower adult BMI.

Table 3.5: Model fit by poverty dynamics model and definition, FE models

Model	n	Fit measure	NLSY-defined	Optimal absolute	Optimal relative	185% FPL	200% FPL
Timing, 4 pd.	2935	$R^2$	0.101	0.100	0.100	0.101	0.099
		BIC	-208.439	-206.630	-205.663	-207.218	-203.768
Timing, 3 pd.	3328	F poverty	1.379	1.121	0.984	1.205	0.714
		BIC poverty	22.256	24.065	25.031	23.477	26.926
Accumulation	3894	$R^2$	0.099	0.100	0.099	0.099	0.098
		BIC	-248.135	-253.403	-250.131	-249.712	-247.706
Hybrid model	3328	F poverty	0.853	1.867	1.237	1.156	0.770
		BIC poverty	19.894	14.626	17.899	18.317	20.324
Accumulation	3894	$R^2$	0.108	0.109	0.108	0.107	0.107
		BIC	-355.040	-356.704	-353.922	-351.298	-351.365
Hybrid model	3328	BIC poverty	3.731	2.067	4.849	7.473	7.406
		$R^2$	0.099	0.101	0.098	0.098	0.098
Hybrid model	3328	BIC	-249.983	-257.620	-247.525	-244.763	-245.583
		F poverty	1.208	2.680*	0.735	0.204	0.362
Hybrid model	3328	BIC poverty	18.046	10.409	20.505	23.266	22.446

*Note:* \*  $p < .05$ . F poverty and BIC poverty indicate F-statistic and change in BIC for addition of poverty measure(s) to base model with no poverty measure(s). Lower BIC values indicate better fit; negative BIC poverty values would indicate better fit with the poverty measure(s).

Figure 3.13: Marginal effect of childhood poverty, accumulation model



The non-parametric effect of poverty accumulation in childhood in Figure 3.13 seems to conflict with the parametric models above, where the accumulation effect was always positive. Instead of a 2% increase in adult BMI for those with all observed childhood years below the optimal absolute threshold over those never in poverty so defined, the GAM results indicate a nearly 9% decrease. Rather than a linear relationship, the spline reveals

that this positive effect is averaging over a somewhat nonlinear effect, and appears to be dominated by the positive slopes at the low end, below around 8%, and in the range of around 25% to 43%. Otherwise, the effect of slightly more time spent in poverty appears to be negative, consistent with the within-period hybrid model. The GAM results, then, seem to indicate that nonlinearity renders the results of the simple accumulation model invalid. Returning to Table 3.5, the best fitting model appears to be the one that most flexibly captures poverty dynamics, particularly with the optimal absolute threshold definition.

### **3.7 *If not poverty...***

In some parts of the income–obesity literature, “low income” is used as the risk factor. Whether we call it poverty or low income, this may be interpreted as positing some threshold below which children are at risk of becoming obese. This process has become common wisdom and generated numerous discussions of what it is about relative deprivation that is associated with excess adiposity. Poverty has essentially been identified as a risk factor for childhood and later adult obesity and the attendant health problems associated with obesity.

Past research has shown that current poverty status, by the usual definition, is not in and of itself a strong predictor of adiposity. Little difference is seen between those below the poverty level and those just above. This implies that a higher poverty threshold might provide a categorization that better accounts for risk of obesity. Applying that logic to a longitudinal framework to assess the predictive validity of various poverty thresholds, however, found little clear relationship across all adults. Testing all possible absolute poverty thresholds showed that any threshold between about 1.5 times the usual Federal poverty level and about 4.5 times FPL produces a difference in BMI between those categorized as poor and those not poor of around 2%. A similar plateau was found in the effects on BMI of poverty status by different possible relative poverty thresholds. While this 2% difference was often—but not always—statistically significant, an increase in BMI from 25 to 25.5 or 29.5 to 30.1 may not represent a practically significant leap in health risk.

There are several possible explanations for the lack of a clear threshold in the relationship between ever being in poverty in childhood and adult BMI. One possibility addressed here is the idea that this dichotomous measure of poverty, even with an improved threshold, is still a crude measure of the deprivation experienced by a child growing up. Some have promoted a developmental timing model of socioeconomic effects on health. As applied to poverty and BMI, this implies that poverty during particular developmental periods will have lasting effects on BMI. Most have identified early childhood as the key period, implying that poverty becomes ingrained in psychological and biophysical processes such as taste habituation and endocrine effects of cheap, high sugar diets (e.g., Brownell et al., 2009), decreased mastery and self-esteem (e.g., Cohen et al., 2010), or dysregulation of interrelated nervous and biochemical systems (Cohen et al., 2010; Huang et al., 2009) that develop relatively early in life and have cascading effects, regardless of later prosperity. In contrast, others take the view that not the timing but the density or accumulation of poverty experiences might best explain the connection between poverty and later adiposity (or health in general). Rather than a triggering event, such as experiencing poverty once in early childhood, risk increases with duration of exposure to related conditions, consistent with the allostatic load hypothesis (Seeman et al., 2010) or giving more time to cement poor diet and exercise habits adapted as a response to stress, privation, and time poverty. Cohen and colleagues find that there is support for both the timing and the accumulation models relating life course SES to adult cardiovascular risk factors, morbidity, and mortality, but that previous studies are limited by reliance on SES measures at only one or two points during childhood. A more flexible conceptualization of poverty dynamics would combine the timing and accumulation models. These dynamics also imply that a different measure of poverty—a different threshold to define risk—might apply than in the simple conceptualization of ever being in poverty. That is, accumulation of years under a higher poverty threshold may be more powerful predictor than ever being under a deeper (lower) threshold.

This paper tested these models of the dynamics of life course poverty on adult BMI (a major cardiovascular risk factor). The OLS models, controlling for a host of variables

related to both SES and adiposity, found some support for the timing model, particularly the positive effects of poverty in early childhood (defined either as 0 to 3 or 0 to 5) on later BMI, and some support for the accumulation model. More years spent under the three highest thresholds considered was related to higher BMI in adulthood. The lowest threshold, the usual (NLSY-assigned) poverty definition, was never significantly associated with later BMI, implying that much of the risk lies above the usual threshold. Indeed, the best definition in the OLS models appeared to be the 185% of FPL definition of poverty or low income. Adults who were ever below this threshold in early childhood or who spent more years of childhood overall in such a state had significantly higher BMIs than other adults. The effects were relatively small. Ever being poor in early childhood accounted for a 2% higher BMI, as did being in poverty 100% of childhood versus never, a marginal effect smaller than those of ethnicity differences or an additional year or two of age.

Although the OLS models accounted for aspects of the mother's background—her own education and that of her family of origin, her home ownership before the subject was born, her aptitude, etc.—that might affect both later income and her child's BMI, there are likely other family-level factors not included in the model (or in the NLSY) that might bias the estimated relationship between poverty and BMI. This analysis employed family fixed effects to account for such potential biases. Unlike Liu (2007), I do not find that ignoring unobserved heterogeneity *underestimates* the relationship between poverty experiences and later BMI. Instead, many of the apparent relationships not only became nonsignificant in the FE models, but became negative: Any (timing model) or more poverty (hybrid model) in specific periods, particularly adolescence, has a protective effect on later BMI. These differences are not accounted for by the difference between the samples used in the OLS models and the FE models, the latter of which are estimated only on sibling groups. This analysis seems to join the ranks of fixed effects models that have “broken” the connection between purported causes such as social networks (Cohen-Cole & Fletcher, 2008) or neighborhood sprawl (Eid et al., 2008) and adiposity.

Lower income during *childhood* appears to be associated with higher *childhood* BMI, and lower income during *adulthood* appears to be associated with higher *adult* BMI, but in this analysis, using a large national dataset, poverty during childhood does not appear to be related to higher adult BMI, even using multiple definitions of poverty. What else might account for this seeming inconsistency? Not everyone may react to poverty experiences, even poverty at the same age, in the same way. Access to social capital may moderate the nutrition or exercise effects of a relatively poor childhood (Evans & Kutcher, 2011). There may be differences in stress tolerance or reactions to stress. Longitudinal research in England, for example, identified diverging BMIs in reaction to work stress based on initial BMI, at least among men: Essentially, the thin got thinner and the fat got fatter in response to the demands and strains of work environments (Kivimaki et al., 2006). Similarly, women may be more likely to eat in response to experienced stress (e.g., Chang, Hillier, & Mehta, 2009). Other coping skills, such as smoking or exercise, may be substitutes for emotional eating and moderate the relationship between experiences of deprivation and stress and later BMI. These or similar mechanisms may explain differential relationships between SES factors and adiposity by gender (e.g., Baltrus et al. 2007; Lee et al., 2009; Power et al., 2005; Senese et al., 2009; Suglia, Duarte, Chambers, & Boynton-Jarrett, 2012), ethnicity (at least in cross-section; e.g., Kumanyika & Grier, 2006; Ogden et al., 2010), or both (e.g., Scharoun-Lee et al., 2011).

So-called “headcount” measures of poverty are crude measures of deprivation, even with an “optimal” threshold. Whether the resulting measure is a proportion of years or a dichotomy within periods, the poverty indicators used here simply capture whether an individual’s family income in a given year was below the threshold, ignoring how far below the threshold and thus how deep the attendant privation and distress may have been (Blank, 2008; Meyer & Wallace, 2009). In addition to measuring the poverty gap or depth, one can account for the severity of poverty by squaring this difference between income and poverty threshold, to put more weight on those in deeper poverty (Foster, Greer, & Thorbecke, 1984, as cited in Averett, 2012). While the developmental models and accumulation model are an improvement over the simple dichotomous conceptualization of child poverty, they nonethe-

less average poverty effects among those \$10 below the threshold, \$100 below, and \$10,000 below. Capturing not only duration of poverty but also the intensity of poverty would better capture the dosage effect of childhood poverty put forth by Cohen and colleagues (2010).

Measuring poverty is inherently applying an outsider's view to an individual's experience. A given poverty threshold is, from the perspective of the individual, arbitrary and meaningless. What is experienced is (absolute) income, as well as perhaps relative deprivation (comparing one's income to a reference group) or relative rank (Kawachi et al., 2010). The experience of poverty may be less about "do I have less than this amount?" and more about "what can I afford to spend on food this month?" There is beginning evidence at the national level (Offer et al., 2010), within adults (Smith et al., 2009), and developmentally (Olson et al., 2007) that instability of food and related resources may be as predictive if not more predictive of obesity than one's permanent (average) level of income. This view focuses attention not on falling below an external threshold but rather variations from one's own accustomed level of income and consumption. The stress of low income may arise not from becoming nominally eligible to be called poor or receive food stamps, but from having to worry a little more than last year or last month about feeding one's family. A fortunate week or month may promote stocking up or gorging on favored foods (Olson et al., 2007). A downward income shock might impinge upon sleep duration, which has been associated with appetite dysregulation (Knutson & van Cauter, 2008), or inspire trying a cheap, high-sugar and/or -fat convenience food that becomes a viable solution to the problem of how to feed yourself or your family on a tight budget (Jastran, Bisogni, Sobal, Blake, & Devine, 2009).

### **3.8 Next steps**

This analysis advanced conceptualization of the relationship between childhood poverty and adult BMI in two ways, via an improved poverty definition (focusing on an improved threshold), and via models that capture the timing or chronicity of poverty experiences. Despite this, the results indicate that poverty has no clear effect on later BMI, and on the average may be predictive of lower rather than higher BMI. A single poverty cutoff does not appear to be an effective tool for identifying those at risk for later obesity, which may

result from potential nonlinearity in the effect of income on later BMI. The next step for this research is to examine this potential nonlinearity of the relationship between childhood income and adult adiposity by gender and ethnicity groupings to explore whether the pattern of how income affects BMI is consistent across groups. Later, I will take up the question of whether income instability may be a better indicator of obesity risk. A lack of difference in adult BMI between those below and above an external poverty threshold in childhood may be due to different experiences of stability of income, as those with more childhood poverty may actually experience less instability than those with fewer years below poverty level.

## Chapter 4

**PART 2: DIFFERENTIAL RELATIONSHIPS BETWEEN INCOME DYNAMICS IN CHILDHOOD AND ADULT ADIPOSITY****4.1 Background***4.1.1 Conventional wisdom*

When reviews of the obesity literature, public officials, and newspaper and magazine stories alike cite a relationship between poverty or lower socioeconomic status and obesity, we can say such a link has become conventional wisdom. That wisdom holds that there is an income gradient to excess body fat, and is consistent with evidence of economic disparities in other health behaviors and outcomes. For example, an article in an issue of *The Future of Children* dedicated to childhood obesity proclaimed that “low-income children are at excess risk of obesity” (Kumanyika & Grier, 2006, p. 190). The reality, of course, is much more complex. This complexity may reflect timing and extent of poverty, familial and cultural traditions around coping with stressful situations and food, genetic factors, access to social capital and services, and so on.

This paper adds to the literature on potential drivers of excess body fat by exploring the complexity of how income in childhood might be related to adult adiposity. While much of the existing literature relies on current income and current body mass index (the most common measure of adiposity) or obesity prevalence, this analysis places the relationship in a developmental context. The result is a nuanced picture of how childhood income affects the potential development of obesity and thus the health, economic, and social consequences of excess body fat—a picture that belies the conventional wisdom.

#### 4.1.2 *A complex relationship*

Snapshot views of the link between poverty or income and adiposity both illustrate and paper over the complexity of how economic conditions might be related to weight. Analysis of the effect of current income on body mass index ( $\text{BMI} = \text{weight in kilograms} \div \text{square of height in meters}$ ) or obesity ( $\text{BMI} \geq 30$ ) often, for simplicity, divides income into ranges—for example, quintiles within the observed data or *a priori* ranges such as \$20,000 to \$25,000—and appear to show not a steep drop-off in adiposity above the lowest income categories, but rather little difference until well above poverty level. This phenomenon has been illustrated among adults (e.g., Kant & Graubard, 2007) and among children (e.g., Anderson, Butcher, & Schanzenbach, 2007). The non-linearity found, of course, depends on the discretization of income. Some results show higher risk among those just above the lowest income category than among those with the lowest income levels (e.g., Schmeiser, 2009). Restricting the Continuing Survey of Food Intake by Individuals (CSFII) to those with incomes less than 130% of poverty found a positive effect of income on probability of obesity and on BMI for women and no effect for men, net of other demographics, homeowner status, food stamp participation, and other covariates (Chen, Yen, & Eastwood, 2005).

Much evidence suggests the usual gradient between income and BMI or obesity prevalence exists only for women in general and white women in particular. In their flexible analysis of data from multiple waves of the National Health and Nutrition Evaluation Survey (NHANES), Komlos and Brabec (2010) modeled BMI as increasing with income below 100% of the federal poverty line (FPL) for black females and then decreasing, and increasing up to 250% and 300% of poverty for white and black men, respectively. The decline of BMI with income appears mainly for women and only above the poverty line. Peak BMIs for males appear well above poverty level. With higher income above 300% of FPL, white men appear to have slightly lower BMI while black men exhibit little relationship between income and BMI.

These differences by gender and race or ethnicity may reflect interactions among genetics, culture, and exposure to environmental forces thought to drive obesity. Multiple factors

likely interact in non-specific ways to create multiple pathways to relative obesity or relative health for specific groups (Braveman et al., 2011; Huang, Drewnowski, Kumanyika, & Glass, 2009). Family proclivities for weight gain and sensitivity to stress will interact with social norms around self-comforting behaviors (such as eating versus smoking versus drugs and alcohol), all in the context of more or less obesogenic environments. Animal models, for example, demonstrate that chronic stress combined with normal or reduced calorie availability results in reduced adiposity, but stress in the context of a high fat and high sugar diet produces excess adiposity (Kuo et al., 2008). Minority neighborhoods tend to have increased access to fast food and convenience stores and decreased access to supermarkets, often net of income or other SES indicators (Powell, Han, & Chaloupka, 2010; Walker, Keane, & Burke, 2010). Blacks living in a census tract with a supermarket are more likely to meet dietary guidelines for total and saturated fat and fruit and vegetable intake than those in census tracts without supermarkets, but no such effect is found for whites (Cawley, 2006; Ford & Dziewaltowski, 2008). Some evidence indicates African American children are exposed to 60% more food-related television advertising than white children (Caprio et al., 2008) and that low income and minority children are exposed to more marketing overall (Kumanyika & Grier, 2006).

SES and race/ethnicity are often confounded in scholarly attention to health and other outcomes (LaVeist, 2005), and evidence is growing that they are independently and jointly associated with “multiple environmental and social risk factors... over a prolonged period of time,” contributing to the development of chronic conditions (Suglia, Duarte, Chambers, & Boynton-Jarrett, 2012, p. e1174). A given level of income or poverty status does not have the same meaning in terms of deprivation and disadvantage for all groups. At a given level of income, wealth and debt levels vary by ethnicity (Caprio et al., 2008; LaVeist, 2005). An income-based indicator of “poverty” gives an incomplete accounting of an individual’s context-specific experience of material disadvantage. Among the poor, the poverty gap—the amount by which family income is below the relevant poverty threshold—is larger for nonwhites (Meyer & Wallace, 2009). Blacks and Hispanics are more likely than whites to be poor in multiple months of a year or all months over a period, and have longer spells of

poverty (Jantti, 2009), and family income for black female-headed households recovers more slowly after the initiation of a poverty spell than among white female-headed households (Card & Blank, 2008).

Any estimate of the effect of poverty or income on adiposity is thus at risk of combining what might be disparate effects—varied trajectories for those just below the poverty line versus those well below, those with more advantage than others at the same income level, those prone to emotional eating versus those prone to other coping mechanisms, etc. Longitudinal analysis of BMI trajectories among children, for example, finds that initial BMI is higher with more family income for white children and Hispanic children of native-born parents, but not among Hispanic children of immigrant parents. BMI change is independent of income for both Hispanic groups, while white children exhibit lower BMI growth with higher family income (Balistreri & Van Hook, 2009). There is a small literature that holds that instability of income and thus food supplies may be more important than permanent income in driving adiposity (Offer, Pechey, & Ulijaszek, 2010; Olson, Bove, & Miller, 2007; Smith, Stoddard, & Barnes, 2009). Considering the dynamics of exposure—the proportion of childhood or the developmental periods during which exposure takes place—also provides more nuanced investigation of how childhood circumstances contribute to later health (Cohen, Janicki-Deverts, Chen, & Matthews, 2010; Conroy, Sandel, & Zuckerman, 2010). Ignoring these multiple reactions or trajectories amounts to measurement error that attenuates estimated effects.

Controlling for family of origin educational background and an indicator of wealth, in previous work I found that childhood poverty—measured in five different ways and across four models of childhood dynamics—had little effect on adult BMI, and what effects were found in ordinary least squares models became negative when unexplained heterogeneity—as might occur with genetic endowments and shared environments—was controlled for via sibling fixed effects. That is, if anything childhood poverty contributes to lower adult BMI. Poverty is a coarse measurement of economic resources and related phenomena. A simple dichotomy ignores the depth of deprivation and tells us nothing about how far below—or

above—this externally set threshold one must be to set in motion forces related to later health. The effect, or lack thereof, of poverty may thus miss different effects throughout the income range. In this paper, I more flexibly examine the effects of childhood income on adult adiposity across gender and ethnicity groups and across developmental periods.

## **4.2 Methodology and data**

### *4.2.1 Exploring the effects of childhood income and adult BMI*

Much of the literature cited above that has noted the complexity of the relationship between income and adiposity relies on cross-sectional data. Modeling the effect of current income on current BMI or obesity status has two main problems that preclude making a causal conclusion. First, numerous studies have suggested that obesity affects future educational achievement and wages (Cawley, 2004; Conley & Glauber, 2007; Gortmaker, Must, Perrin, Sobol, & Dietz, 1993). Second, appealing to the power of current income assumes that level of income has been in place long enough to set in motion the forces that drive an obviously developmental process. Excess adiposity develops over time, as calories in exceed calories out and the unexpended energy is stored, partly as glycogen and protein but mostly as fatty tissue (Schoeller, 2008). I therefore take a long-run developmental approach, modeling adult adiposity as a function of childhood income dynamics.

I take advantage of linked mother-child data from the National Longitudinal Survey of Youth-1979 cohort. The NLSY is a premier source of information on labor market experiences and income dynamics, in part due to supplemental samples of African Americans, Hispanics/Latinos, and low-income whites.<sup>1</sup> Specifically, I connect data on adult children in the NLSY Child and Young Adult sample with background characteristics and income data in the mother's interviews dating back to 1979, and characterize the mother's reported household income in terms of how old the child was (e.g., income reported for the year the child turned 10, etc.). There are over 6000 adult children in the data with legitimate

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<sup>1</sup>Specifically, "whites" in the NLSY are youth designated as neither African American nor Hispanic. In this project, I remove from this category mothers who self-identified as Asian or Pacific Islander. Child race/ethnicity status in the NLSY Child and Young Adult sample is derived from that of the mother.

BMI observations at age 19 up to age 38. In order to avoid artificially smoothing out the dynamics of income, I do not impute the household income measures, but do impute other covariates using a multivariate normal regression framework. No imputation was attempted for information prior to 1979 (or, more generally, for any year with no interview), so adult children born before 1979 are not included in the analyses here. The maximum age is thus 30, and the adult children in the analytical sample were born between 1979 and 1991. The analyses presented are carried out on a dataset representing the average of five imputed datasets and thus do not control for uncertainty of imputation.

I model the natural log of BMI (which partly addresses some observed non-linearity between BMI and some predictors) in a general model

$$\ln BMI_i = \beta_0 + \beta_1 Y_i + \beta_2 P_i + \beta_3 C_i + \beta_4 T_i + \varepsilon_i \quad (4.1)$$

where  $Y$  is a measure or set of measures describing normalized family income during childhood (specified as a multiple of the federal poverty level for that year),  $C$  are exogenous child covariates known to be related to BMI (age, gender, and ethnicity),  $T$  is a time trend capturing secular changes in BMI, and  $P$  are parental, mostly maternal, characteristics potentially related to income during the subject's childhood and/or diet and exercise practices. These include theoretically time-invariant characteristics, such as the timing of the mother's first birth and the subject's birth, the mother's intelligence or aptitude (as measured by the Armed Forces Qualifying Test [AFQT]), whether the mother was foreign born or spoke a foreign language at home growing up, what religion she was raised in, the education of her family of origin, and whether she lived in the South or in a rural area at age 14. It also includes time-varying characteristics measured up to the year of the subject's birth, thus capturing the mother's own educational achievement and, as an indicator of wealth, whether she or her spouse owned a home prior to the measurement of income considered in  $Y$ . Income is normalized for family size (by dividing family income by the square root of family size) and expressed as a multiple of the usual poverty threshold.

While average income during childhood, essentially the normalized permanent income, might have important effects, it remains a relatively crude measure that ignores dynamics

of the timing of the income that might have important ramifications for future health, as represented by BMI. In keeping with a broader research on how SES in childhood affects adult health, I explore the timing of the income. This is consistent with literature in a variety of fields—developmental psychology, child welfare, and health—that cite the importance of events during particular sensitive periods. The fetal origins hypothesis, for example, cites the importance of conditions and experiences in utero and during the early months of life. Maternal diet during pregnancy is frequently cited as an important factor, as overnutrition is thought to essentially program the child’s biology to consume high calorie diets (Cohen et al., 2010; Muhlhausler & Ong, 2011). Analysis of the Panel Study of Income Dynamics (Ziol-Guest, Duncan, & Kalil, 2009), for example, found that more income among those with low income ( $< \$25,000$  in 2005 dollars) during the prenatal and birth year was associated with lower adult BMI, lower overweight prevalence, and lower prevalence of morbid obesity ( $BMI \geq 40$ ), all controlling for income when the adult was 1 to 5 years and 6 to 15 years of age.

The fetal origins hypothesis can be seen as embedded in a more generalized developmental period approach which recognizes that different mechanisms connecting SES to later health will be more or less prominent during different periods (Cohen et al., 2010). Early childhood may involve more physiological processes, in which the presence of a high sugar and/or fat diet may set in motion irreversible metabolic or epigenetic processes that affect appetite, insulin regulation, and adipose tissue development (Conroy et al., 2010; Fall, 2011; Ziol-Guest et al., 2009). Conditions during the school-age years may highlight the importance of schools, while “adolescence may be a sensitive period during which modeling of poor health behaviors such as smoking and poor dietary habits may increase lifetime risk for” obesity and related health conditions (Cohen et al., 2010, p. 47). Due to data limitations, specifically the biennial nature of NLSY data collection after 1994 plus otherwise missing income data, I break childhood into three developmental periods covering early childhood (from the year the child was born through the year the child turned 5), middle childhood (6 to 11), and adolescence (12 to 17).

In addition to known differences in observed obesity prevalence and BMI across gender and ethnic groups (e.g., Flegal, Carroll, Ogden, & Curtin, 2010; Ogden, Lamb, Carroll, & Flegal, 2010), and the above discussed differences in the effects of low income across groups, there are arguments for considering gender and race/ethnicity in the measurement of adiposity itself. The same prevalence or a specific value or range of BMI does not have identical meaning across all groups. As a measure of adiposity, BMI does not differentiate between fat and fat-free mass, so that an additional 10 pounds of muscle will increase BMI the same as an additional 10 pounds of fat. Differences in BMI therefore do not necessarily imply differences in body fat (Flegal, et al., 2010) and BMI tends to produce varying degrees of misclassification of obesity status across gender and race/ethnicity groupings when judged against a more direct measure of adiposity (Burkhauser & Cawley, 2008). Meanwhile, there appear to be different patterns of fat distribution and metabolism by ethnicity and thus differences in health consequences at the same BMI or same category of BMI, although these differences are much smaller than those *between* adiposity categories (Caprio, et al., 2008).

In this paper, therefore, I explore differences in the effect of childhood income on adult BMI across gender and ethnicity groups as well as by developmental period. In order to incorporate the non-linearity apparent in the literature discussed above, I use in place of  $Y$  in the general model (4.1) a cross-validated cubic regression spline, fitting a curve to the marginal effect of childhood income (overall or by period) while controlling for the effects of covariates in their usual linear, parametric form in a Generalized Additive Model (as implemented in the R package `mgcv` [Wood, 2011]). This allows exploration of how increased income contributes to adult BMI through sub-ranges of the income spectrum net of the effect of maternal wealth, educational background, conditions describing the mother's upbringing, and so on, without relying on or imposing *a priori* parametric specification of categories of income. I furthermore compare a spline effect estimated for the whole sample, which essentially averages the effects of income across gender and ethnicity group, with group-specific splines to explore how a marginally higher childhood income at different levels relates to adult adiposity uniquely for different groups.

### 4.3 Results

#### 4.3.1 *The effects of whole childhood income*

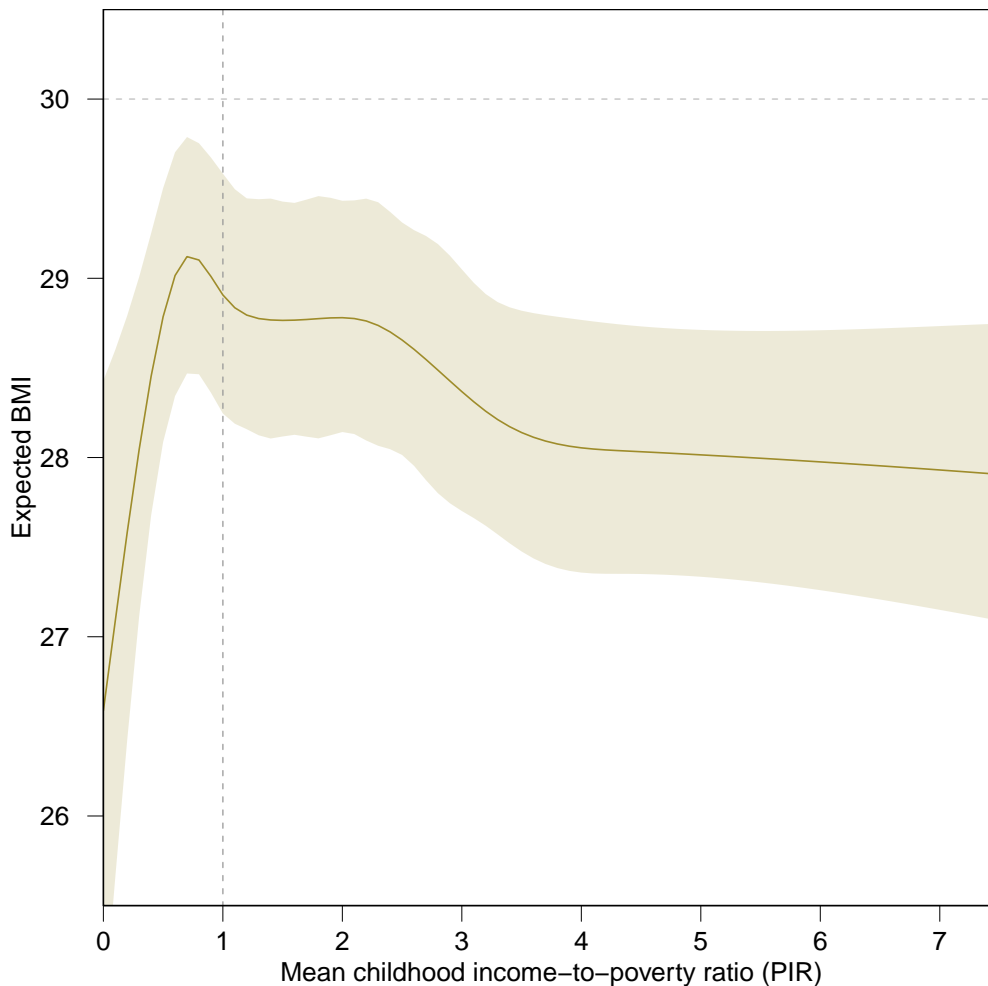
While a number of research efforts involving contemporaneous income and adiposity have noted that BMI or obesity risk does not significantly decrease until well above poverty level, fewer have noted that BMI actually increases with income at lower income levels. Most of those efforts, and the nature of the resulting patterning of the income–BMI (or income–obesity) relationship, are dependent upon the discretization of income selected. Putting all those with an income at or below poverty level together in one large group (e.g., Kant & Graubard, 2007) thus misses the increase in obesity risk evident with more income within the lowest income levels (e.g., Schmeiser, 2009; Ziol-Guest et al., 2009). A more flexible approach allows investigation of important nonlinear patterns. The current analysis applies a similar approach as that of Komlos and Brabec (2010), with a cross-validated cubic spline in lieu of a loess curve, to permanent income levels during childhood.

Average income during childhood, of course, measures a somewhat different phenomenon than a simple dichotomy of ever being in poverty, something closer to a consideration of the accumulation of poverty experiences. At a given level of income instability, a mean childhood income near the poverty line implies more time spent under that poverty threshold than a higher mean income. Very low average income implies a childhood full of deprivation and thus more chance for the purported mechanisms that might connect poverty to obesity—cheap food, lack of exercise opportunities, metabolic effects, etc.—to take hold.

The effect illustrated in Figure 4.1 instead shows quite low BMI associated with the lowest levels of childhood income, and a positive gradient up to around two-thirds the usual poverty line. The estimated slope in this range is quite steep, representing a difference of over two BMI points ( $2 \frac{kg}{m^2}$ ). The spline fit in the figure, which flexibly captures the effect of income net of the covariates described above, is estimated across all gender-by-ethnicity groups present in the data, with BMI predicted here for black females. It shows that high levels of adult BMI are expected, on the average, only for those with an average

childhood poverty-income ratio (PIR) in the range of 50% to 120% of FPL. Contrary to the conventional wisdom, the deeper childhood poverty associated with very low average income appears to be as protective, in terms of adult BMI, as average income above three or four times poverty. By this view, there appear to be “two poverties” with different risk profiles for later adiposity, which may partly explain a lack of effect of poverty on later BMI overall.

Figure 4.1: Marginal effect of mean childhood income on adult BMI

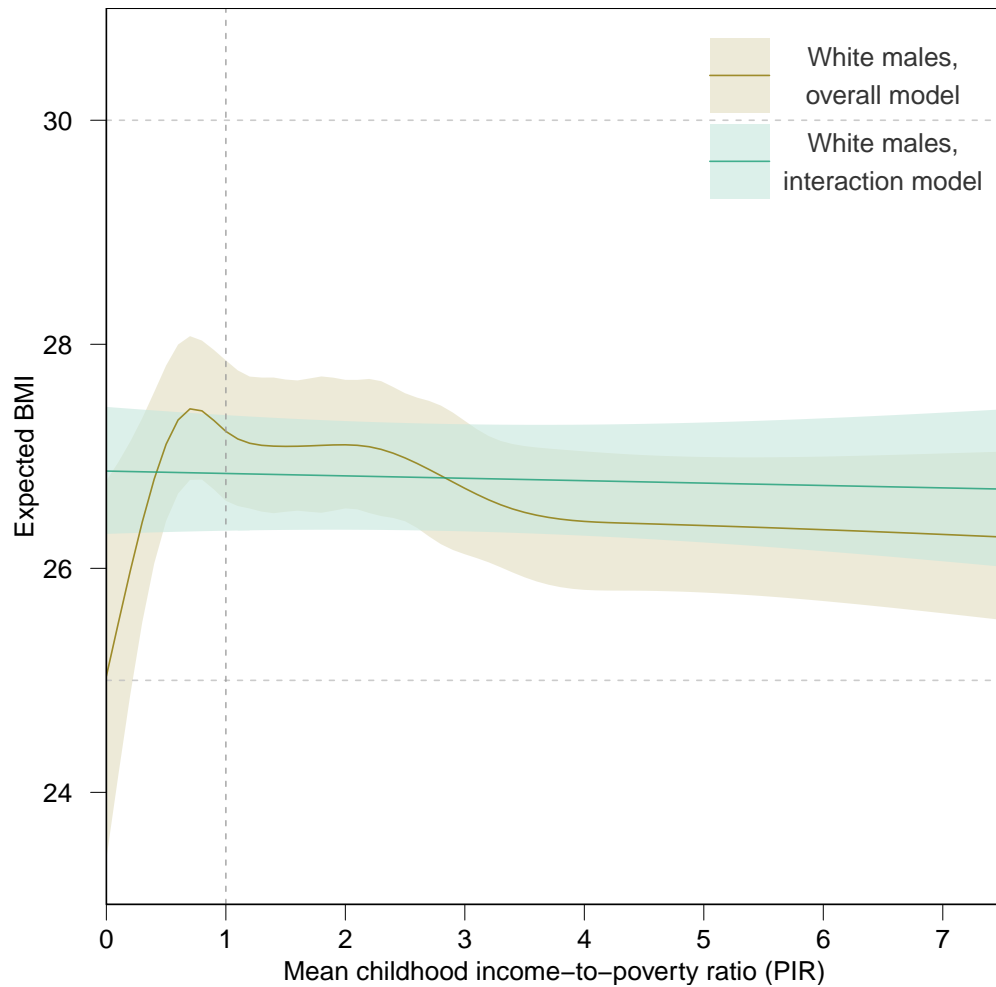


The overall spline illustrated in Figure 4.1 was estimated across all groups, essentially a “main effects” spline averaging over all groups the effect of a little more income within

a small window of income, with separate intercepts estimated for Hispanic females versus white males, etc. (including controlling for a small number of Asian/Pacific Islander men and women). One question is whether this overall pattern of “two poverties” is apparent for all groups, at least in this sample. In the figures that follow, I compare this overall spline, merely shifted up or down for each group, with a group-specific spline estimated via the spline equivalent of an interaction model. For example, the overall model curve for white males in Figure 4.2 is lower than the curve in Figure 4.1 reflecting the lower expected BMI over the income range shown for white males versus black females. As for Figure 4.1, these expected BMIs are estimated with all covariates held constant at their sample-wide means or modes (e.g., age 25.5 years, with a mother who did not own a home and had not completed any education beyond high school before the child was born, etc.). Different values of these covariates would merely shift the curves up (e.g., older age or lower maternal AFQT score) or down (e.g., having a foreign-born mother or one whose mother, father, or oldest sibling had any education beyond high school). Each figure thus contrasts the predicted BMI curve for that group from the overall model with the predicted BMI spline for the group-specific model.

Comparing the group-specific spline effect for white males in Figure 4.2 for that of white females in Figure 4.3 shows quite disparate effects of income on later BMI. For the latter, income has a positive relationship with adult BMI up to just below poverty level, and a negative relationship from poverty level to around three times FPL. For white women, there is little protective effect of more income from three times the poverty level through the 7.5 times FPL maximum explored here, while for white males, this apparent independence of adult BMI and childhood income extends throughout the range. These results are quite different from those of Komlos and Brabec (2010), who found an increase of less than one BMI point from the lowest *current* income level to around 250% of FPL for white males, and a negative gradient for white females up to 600% of FPL. Note that while the spline selection procedure found a linear function to be the best fit for white males, the overlap of the 95% confidence intervals does not fully reject the hypothesis that white males may exhibit a curvilinear effect of marginally more childhood income, particularly at the lowest

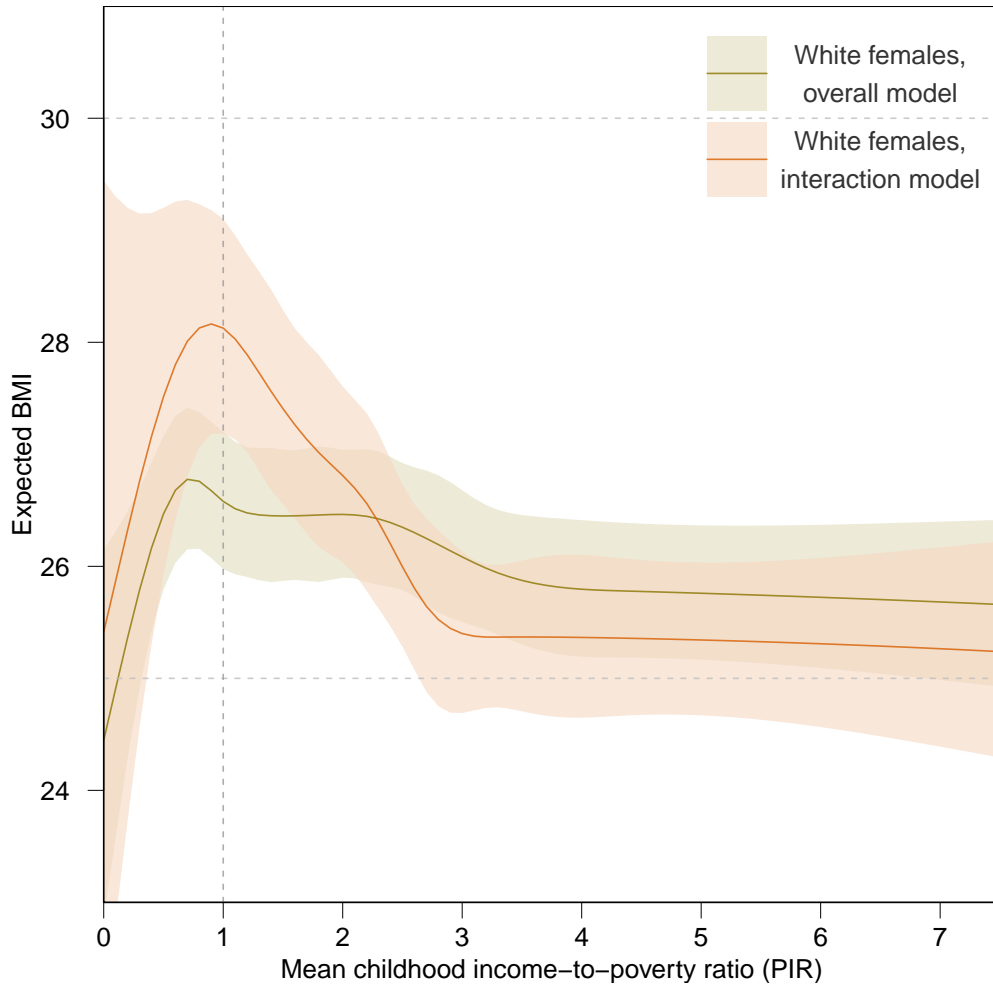
Figure 4.2: Marginal effect of mean childhood income, white males



income levels.

In this analysis, controlling for family educational background and other maternal characteristics, the average white female is expected to have a lower BMI than the average white male when both had average childhood incomes at the very lowest levels, below 25% of poverty, and when both had permanent childhood incomes above 300% of FPL. In between, white females illustrate the “two poverties” phenomenon while white males show essentially no effect of childhood income on adult adiposity and no non-linearity at low income levels. Note again that the confidence intervals indicate that white women do not differ

Figure 4.3: Marginal effect of mean childhood income, white females



significantly from the overall pattern, except perhaps for an exaggerated effect of average childhood income around poverty level. Note as well that the confidence interval for white women becomes quite large below around 35% of FPL and does not preclude a protective effect of higher income, indicating much uncertainty about the positive marginal effect of childhood income at the lowest levels. I return to this uncertainty below.

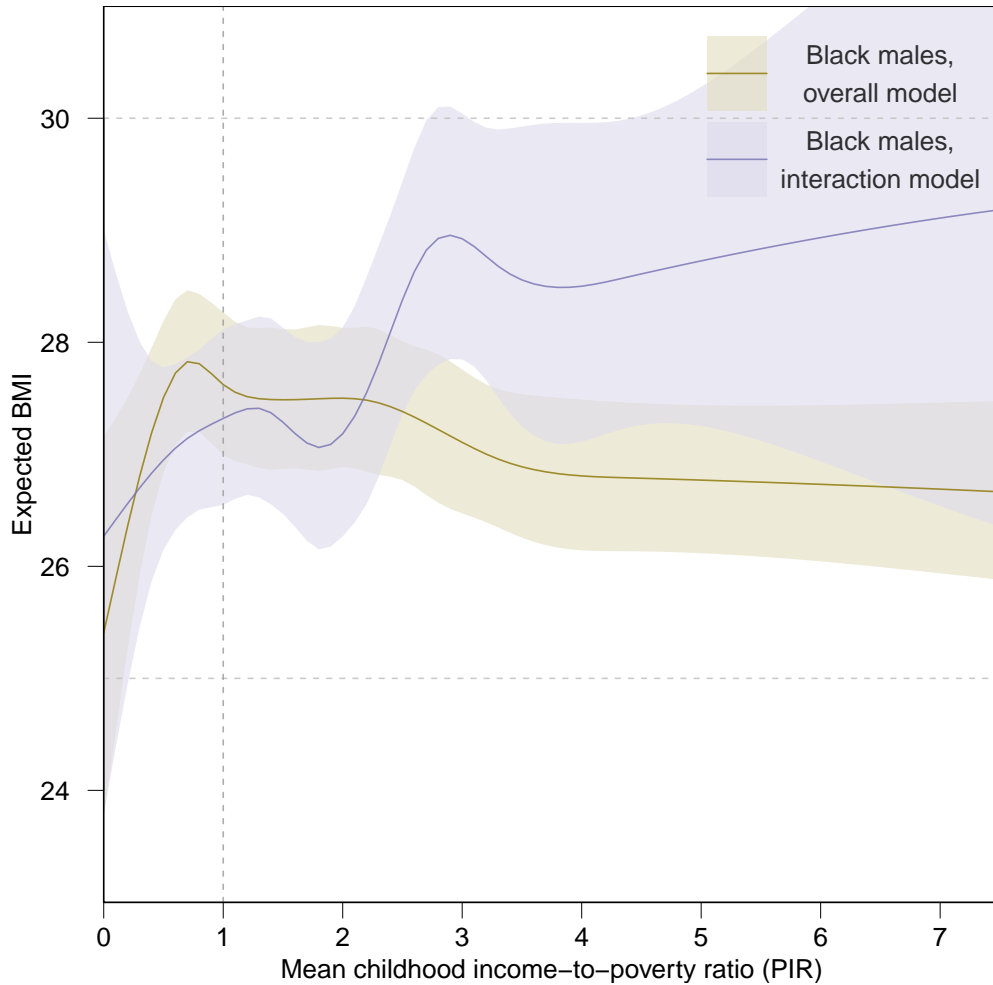
African American males and females also show contrasting effects of childhood income. As seen in Figure 4.4, black males have something of a “two poverties” effect, with higher BMI associated with increased income up to around 133% of FPL. After a short stretch of

negative gradient, the curve jumps to a higher BMI level, in a significant departure from the overall pattern. Black males with an average childhood income near 300% of poverty appear to have a significantly higher risk of obesity than other groups. Above 400% of FPL, more income is associated with a higher adult BMI, although with a much shallower slope than the range from 200% to around 280% of FPL. Overall, as with current income in Komlos and Brabec (2010), the trend for black males appears to be higher BMIs with higher incomes, although the confidence interval above around 480% of FPL does not rule out a protective effect of more income. Similarly, the confidence interval indicates much uncertainty about the positive marginal effect of average childhood income below around 50% of FPL.

For black females, there is a clear and quite linear negative gradient and no evidence of the “two poverties” phenomenon. This implies vastly different adult BMIs for otherwise similar adult black males and females who grew up with average childhood incomes below around 250% of FPL or above around 500%. For such hypothetical pairs, relative poverty implies a higher BMI for the woman, while relative fortune implies a higher BMI for the man. Note, of course, that while these analyses control for a host of factors likely associated with both childhood income and family diet and exercise practices, they do not control for genetic or other unobserved factors that might affect both income and BMI. Furthermore, the confidence intervals do not rule out the hypothesis that black females exhibit the same null effect of additional childhood income between 350% and 750% of FPL as in the more precise overall model.

Hispanic males, as seen in Figure 4.6, appear to experience a marginally more protective effect of higher income than white males (in Figure 4.2). Essentially, higher permanent childhood income, at least through 7.5 times FPL, appears to have little effect on the BMI of adult Hispanic males. The specific effect for Hispanic males is little different than the overall, cross-group spline, at least above the lowest levels of childhood income. For Hispanic females (Latinas), in contrast, the effect is more similar to that for black females, which is to say the expected negative gradient, although wide confidence intervals indicate the

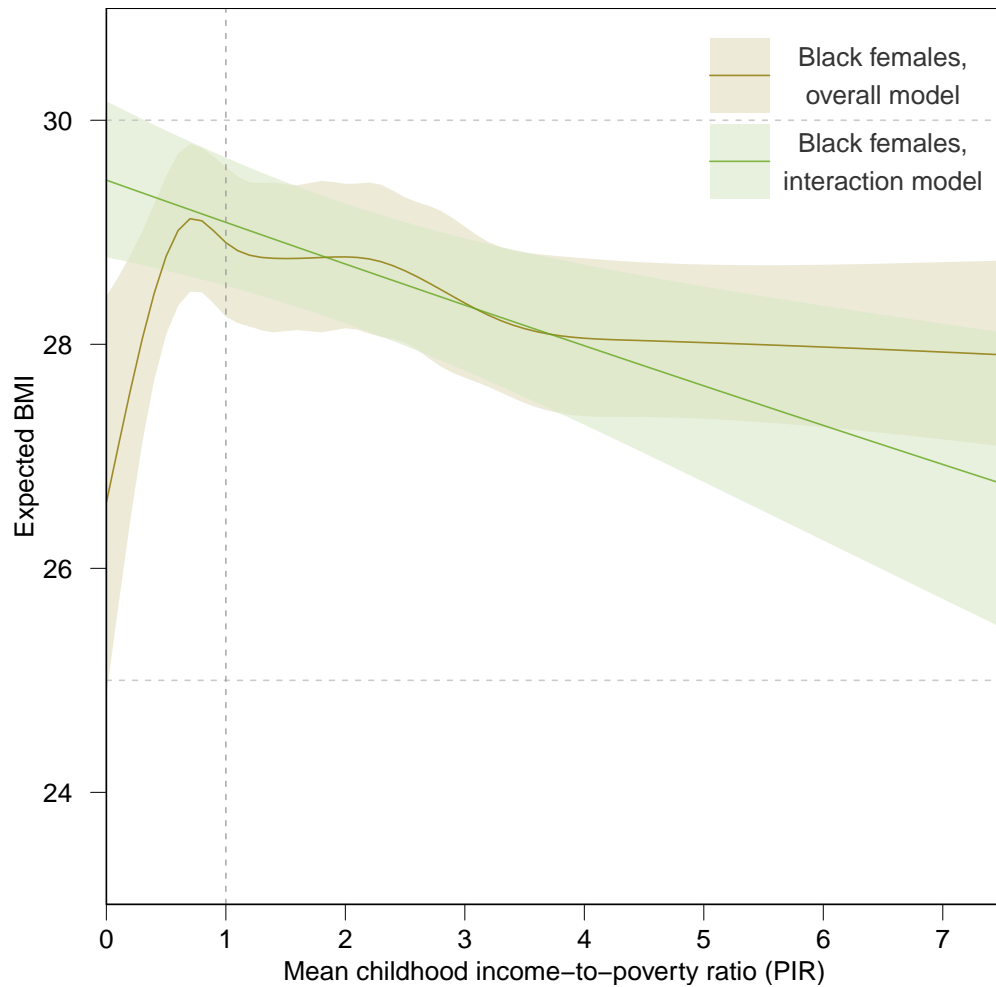
Figure 4.4: Marginal effect of mean childhood income, African American males



marginal effect of higher average childhood income may well be close to 0 for both African American and Hispanic women.

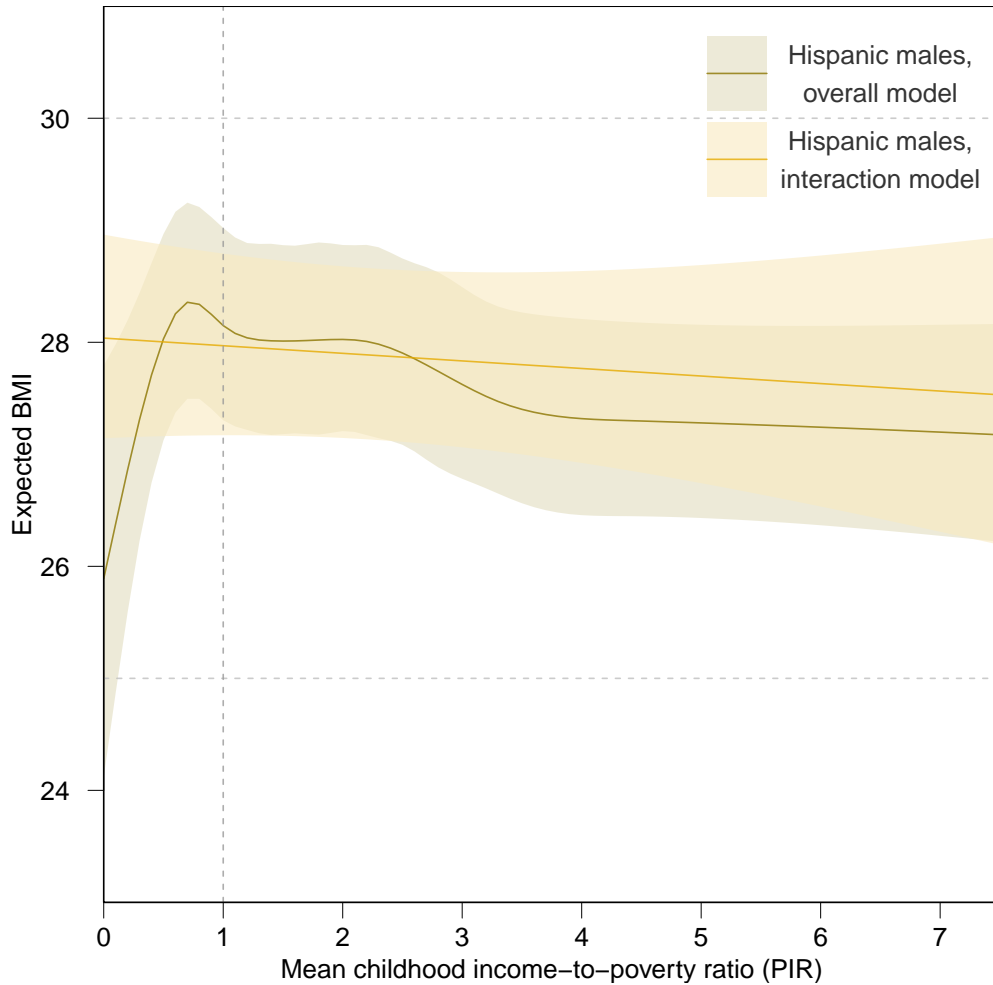
Extreme deprivation during childhood, as measured by low average childhood income, is not associated with lower adult BMI for Hispanics as it appears to be for white women and black males. *Ceteris paribus*, the highest expected BMIs in adulthood (again, predicted here for subjects age 25.5 whose mothers and maternal relatives each had only high school educations, etc.) are for black females with very low permanent childhood income and black males with high levels of childhood income, and the lowest expected BMIs for more fortunate

Figure 4.5: Marginal effect of mean childhood income, African American females



Latinas and the least fortunate white females and black males. Over all of childhood, the “two poverties” effect seems to apply mostly to white females and to a somewhat lesser extent to black males, while the usual negative gradient is apparent only for black females and Latinas. Childhood income does not have a consistent protective effect in terms of reduced future body mass.

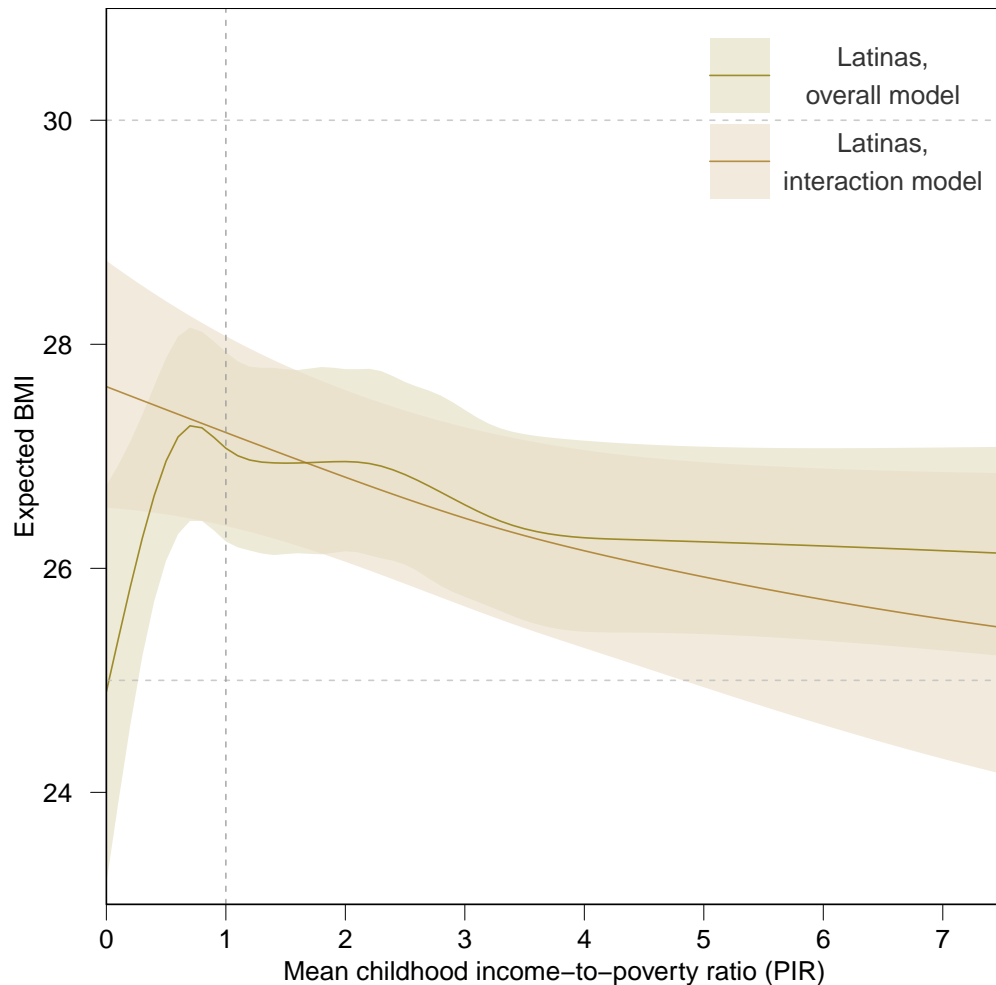
Figure 4.6: Marginal effect of mean childhood income, Hispanic males



*Sensitivity analysis: The effect of zero average childhood income*

The reader will have noticed that the spline effects are presented including estimates for adults with reported permanent childhood income of \$0. The NLSY includes extensive querying of income sources for all family members, including military pay and veteran's benefits, various forms of transfer programs and social insurance, support from relatives, and investment income and rents. A reported income of \$0 represents either literally reporting no income from all these sources for all family members for the year, or a sum counteracted by business or farm loss (there are no negative incomes—net losses are bottom coded at 0).

Figure 4.7: Marginal effect of mean childhood income, Hispanic females



Having a business may imply a stock of wealth into which the family can tap to smooth consumption, while a farm implies growing one's own food, but in both cases a true income of \$0 represents extensive family stress, economizing on food choices, and so on. The NLSY does not, of course, assess barter or black market income, and the total net family income variable created by the NLSY does not include income from household members not related by blood or marriage (thus excluding, for example, the mother's spouse's parents), and may therefore in some cases miss important sources of food and other necessities. The decision to include only family income rather than all household income naturally represents a trade-off in terms of avoiding aggregating the incomes of unrelated roommates but perhaps classifying

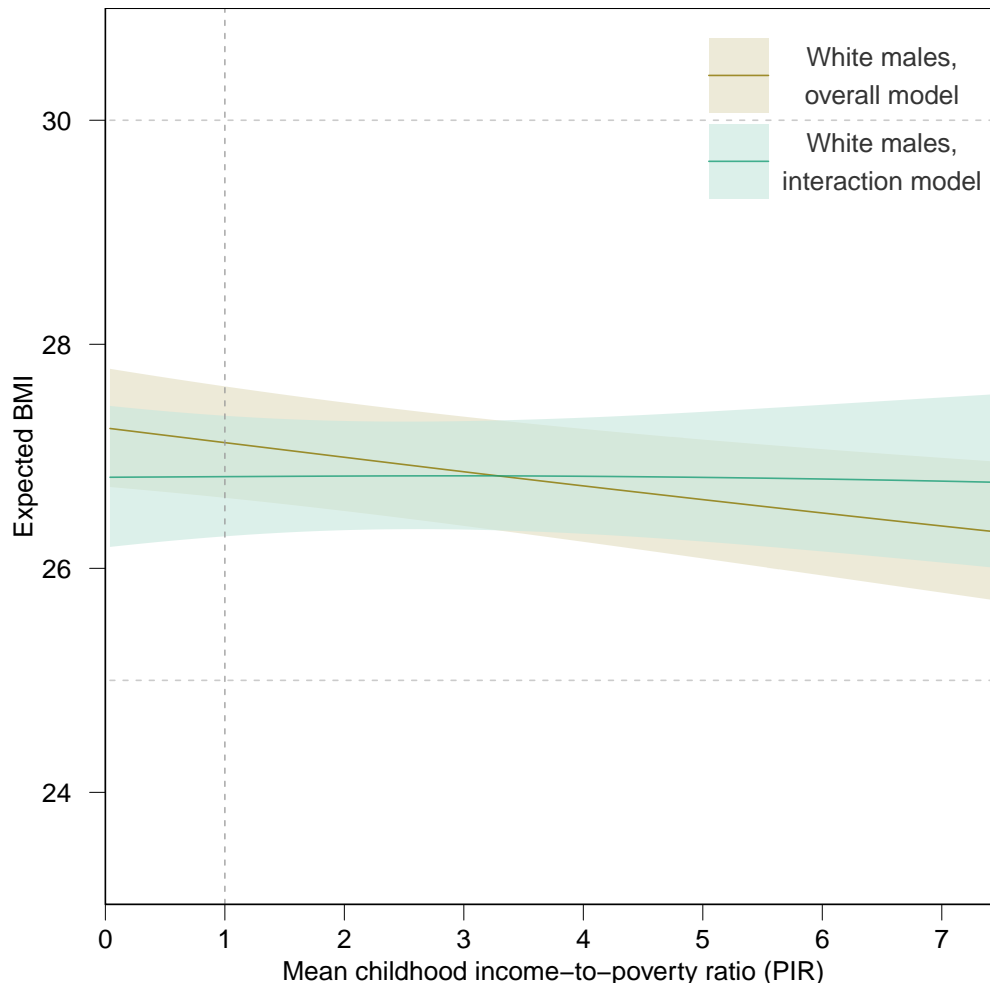
some families as poor that may well be enjoying a more comfortable existence due to support from other household members.

Exploration of the dataset revealed eight observations with a mean childhood income of 0, including two with only a single year of income reported for all of childhood. Because this group defines the low end of the spline—the lower boundary—one question is to what extent conclusions about the positive marginal effect of income at low levels are dependent upon this bound. Similarly, but less of an issue here as we are concentrating on the lower end of the income scale, those with the maximum average childhood income set the upper boundary, and their *ceteris paribus* average BMI relative to those just below on the income scale determines the curvature fit in that range. One common method of testing the robustness of income effects is to trim both the upper and lower ends of the income range. A robust effect should remain, perhaps attenuated, even in the trimmed sample.

The lower bound of the overall model splines above indicates the group with zero reported net childhood income has a lower conditional mean adult BMI than those with marginally higher incomes, particularly those represented by the peak of the “two poverties” phenomenon around 80% of FPL (where the spline procedure placed a “knot” defining the curve). If there is a consistent, significant, positive marginal effect of income among those with the lowest levels of childhood income, the spline procedure should continue to find a positive slope in that range even with the slightly higher bound resulting from eliminating those with a mean childhood income of 0.

Instead, as seen in Figures 4.8 through 4.13, the “two poverties” effect seen in the overall model disappears when I exclude this small group of adults with low BMIs and 0 reported childhood income. Without this group of apparent outliers, the spline cross-validation evidently decides that any improved fit from non-linearity at low incomes did not outweigh the penalty the procedure applies for “wiggleness”. (The figures presented are based on GAM models conducted after also trimming those with the highest observed mean childhood income—specifically, two observations with average childhood incomes more than 33 times

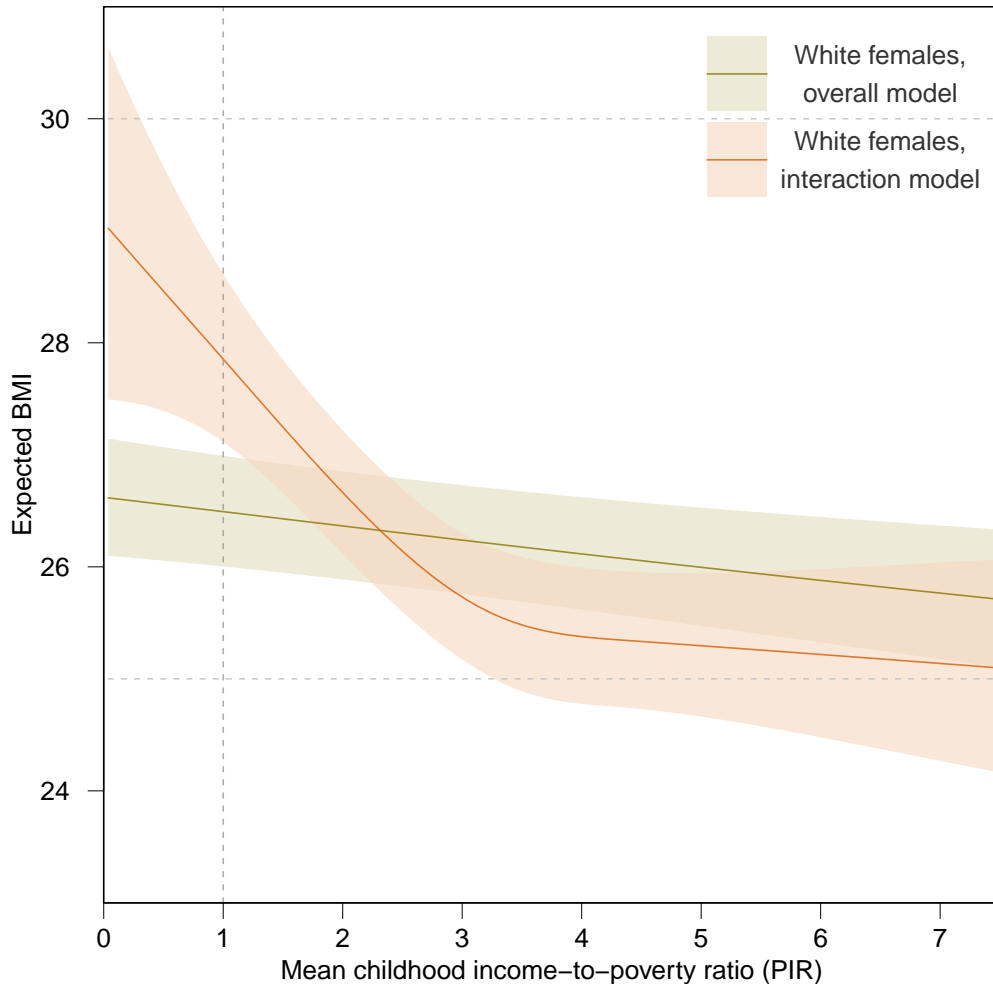
Figure 4.8: Marginal effect of mean childhood income, trimmed sample, white males



poverty level—although trimming the top end of the income distribution had no effect on the splines in the range presented here.) Instead, the smoothing algorithm concluded that a simple linear negative gradient provided the best fit, at least over the range of just above 0 (with the revised lower bound) to 7.5. While this is consistent with the conventional wisdom, note that the overall effect between these two endpoints is around 1 BMI unit. Controlling for maternal characteristics including education and home ownership (prior to the subject’s birth), subject age, etc., the model estimated on the trimmed data finds that the average BMI difference between those with an average childhood income of 7.5 times poverty level and those with an average childhood income below poverty level (now eliminating those

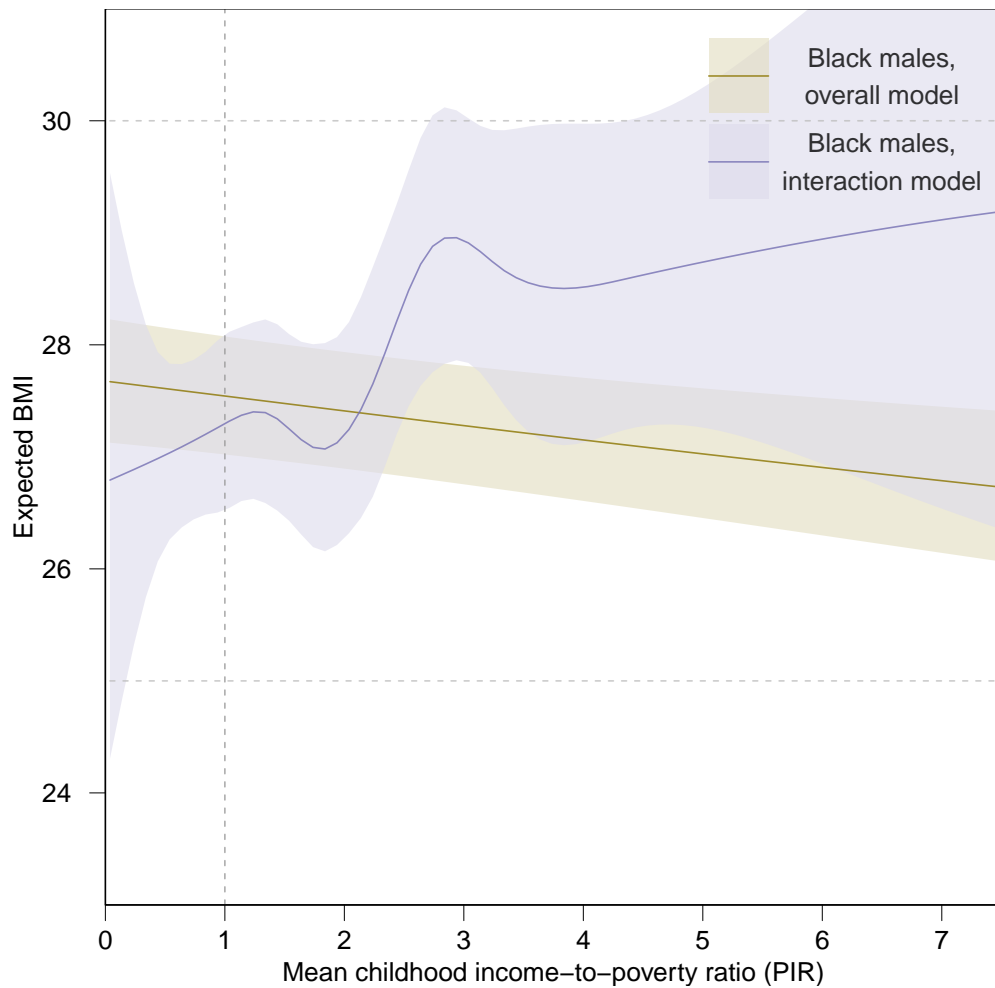
with 0 reported income) is less than 1 BMI point.

Figure 4.9: Marginal effect of mean childhood income, trimmed sample, white females



Most of the group-specific splines appear to be unchanged. The generally null effect of childhood income for Hispanic males is robust to the presence or absence of Latinos with zero reported childhood income, and none of the group with no reported net family income in childhood were white males (thus not changing the estimated spline for that group). Eliminating this group of apparently influential cases (two of whom were black males) resulted in the elimination of a knot in the spline for African American males at around 60% of FPL and a slightly increased lower boundary, but the general pattern is

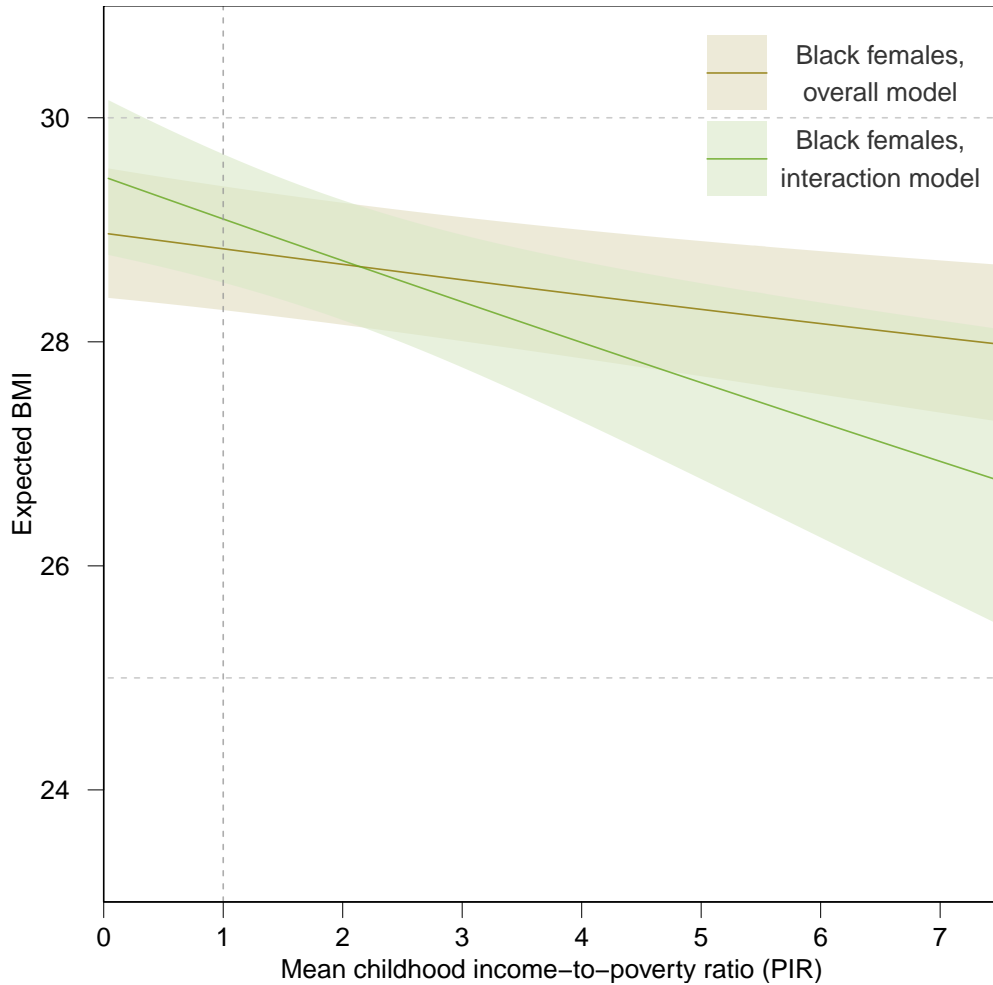
Figure 4.10: Marginal effect of mean childhood income, trimmed sample, African American males



quite similar. Little if any change is observed for the splines for black or Hispanic females (one each of the group of eight with zero reported childhood income).

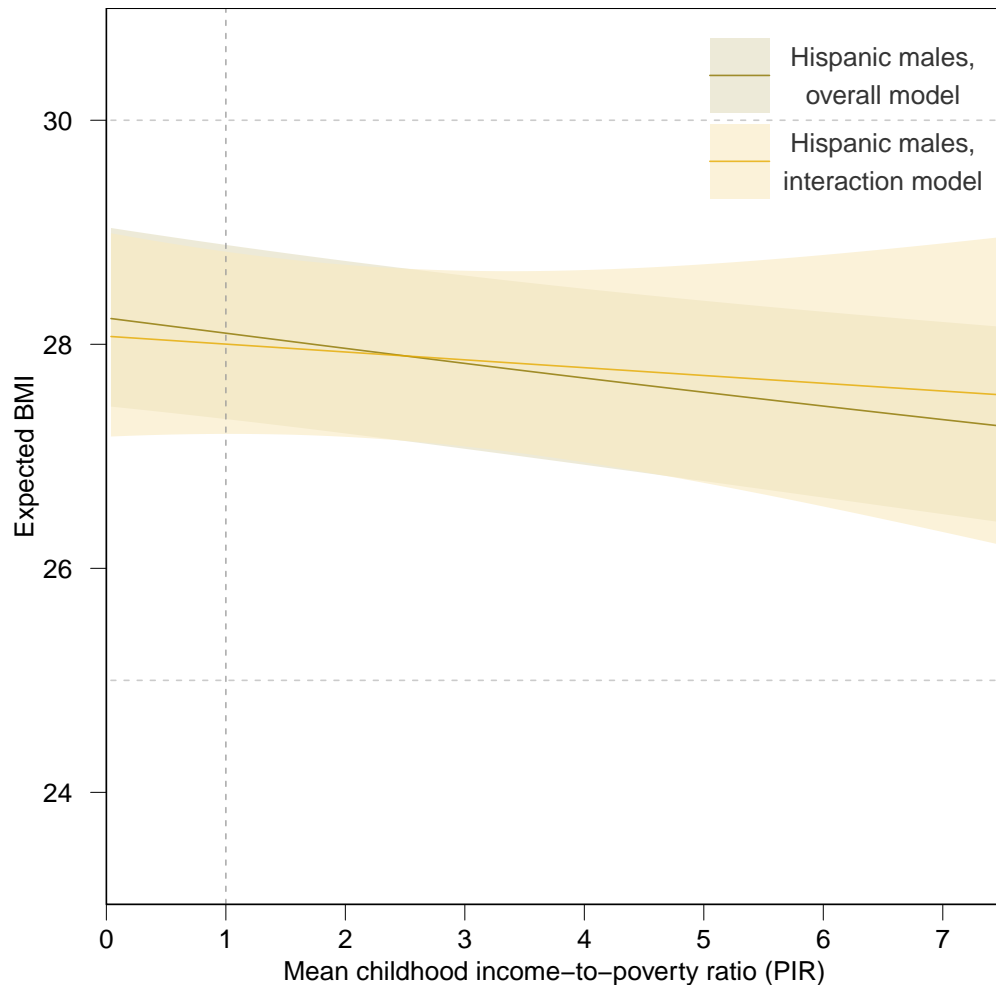
The only group-specific result largely affected by dropping these eight subjects is that for white females, even though only two of the eight were white females. Without them, the spline procedure finds a much simpler curve relating childhood permanent income to adult BMI, with (in the range presented) only a single knot around 3.25 times FPL, in contrast to the original spline with a knot just below 1 and two more between 2 and 3. The result

Figure 4.11: Marginal effect of mean childhood income, trimmed sample, African American females



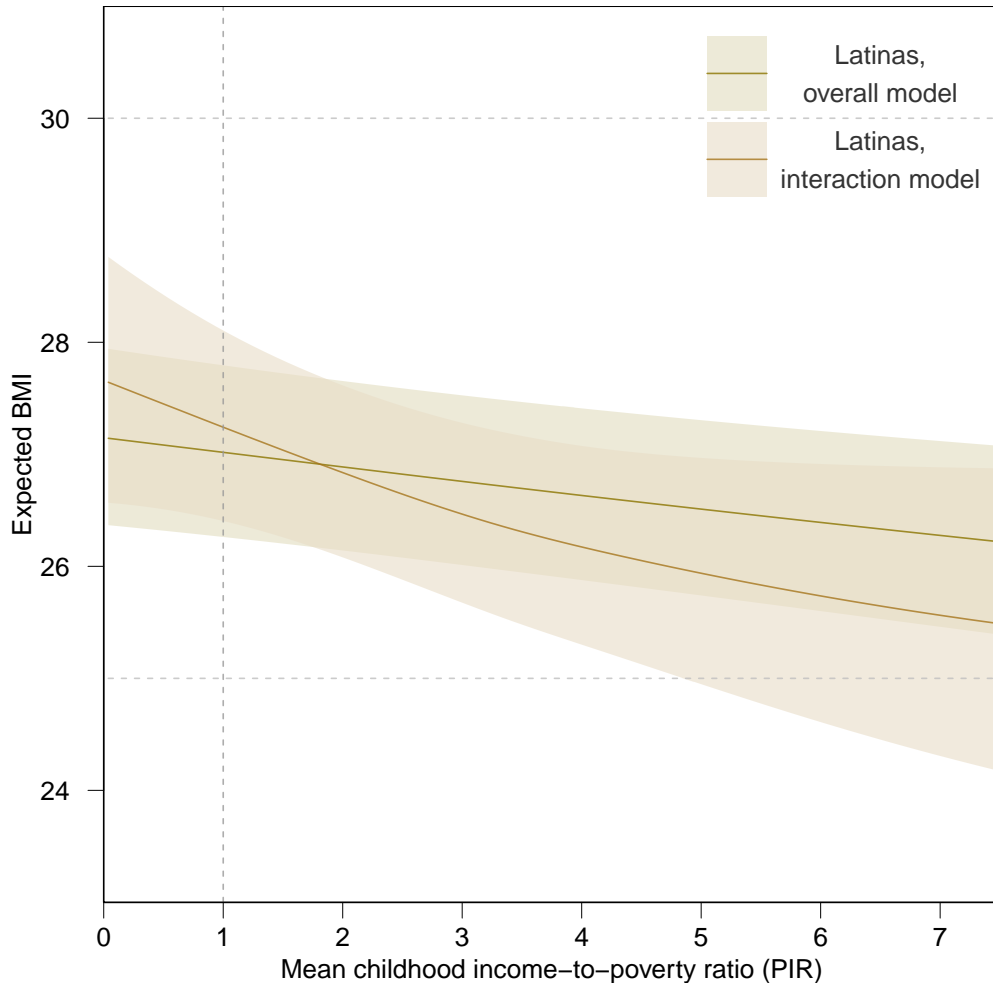
is no finding of a positive marginal effect of childhood income below poverty level. Recall that with the full sample above the rapidly expanding confidence interval below around 35% of FPL indicated uncertainly about the positive marginal effect of mean childhood in this range. Instead, with the slightly trimmed sample, white women exhibit a clear negative gradient up to around 300% of FPL and no effect above that level, and a significantly different pattern than the overall model.

Figure 4.12: Marginal effect of mean childhood income, trimmed sample, Hispanic males



Thus, while the positive marginal effect of childhood income on the adult BMI of black males is robust to excluding those with 0 reported childhood income, the positive marginal effect previously observed among lowest income white females, and the “two poverties” effect, appears to have been largely driven by two observations with observed adult BMIs of 20 and 23. These two observations—sisters whose mother gave birth to their oldest sibling at age 15, and who had seven and nine childhood income observations—appear to be different from their peers with slightly higher reported childhood incomes. Removing these two observations and changing the lower bound of the spline results in a markedly different estimated marginal effect of income at low levels of income.

Figure 4.13: Marginal effect of mean childhood income, trimmed sample, Hispanic females



For the other five groups, the modeled patterns of the effects of permanent childhood income on adult BMI are consistent with what we saw above, specifically a generally positive gradient among black males (with some potential non-linearity between around 125% and 375% of poverty), the usual negative gradient for black women and Latinas, and no effect of childhood income on white and Hispanic males. For the profile used for illustration here (25.5 years of age, with a mother with no education beyond high school and no home ownership as of the child's birth, etc.), both white females and black females at the lowest income levels are significantly at risk of obesity—the predicted BMIs are not significantly different than 30 at low income levels—while black males with childhood income levels

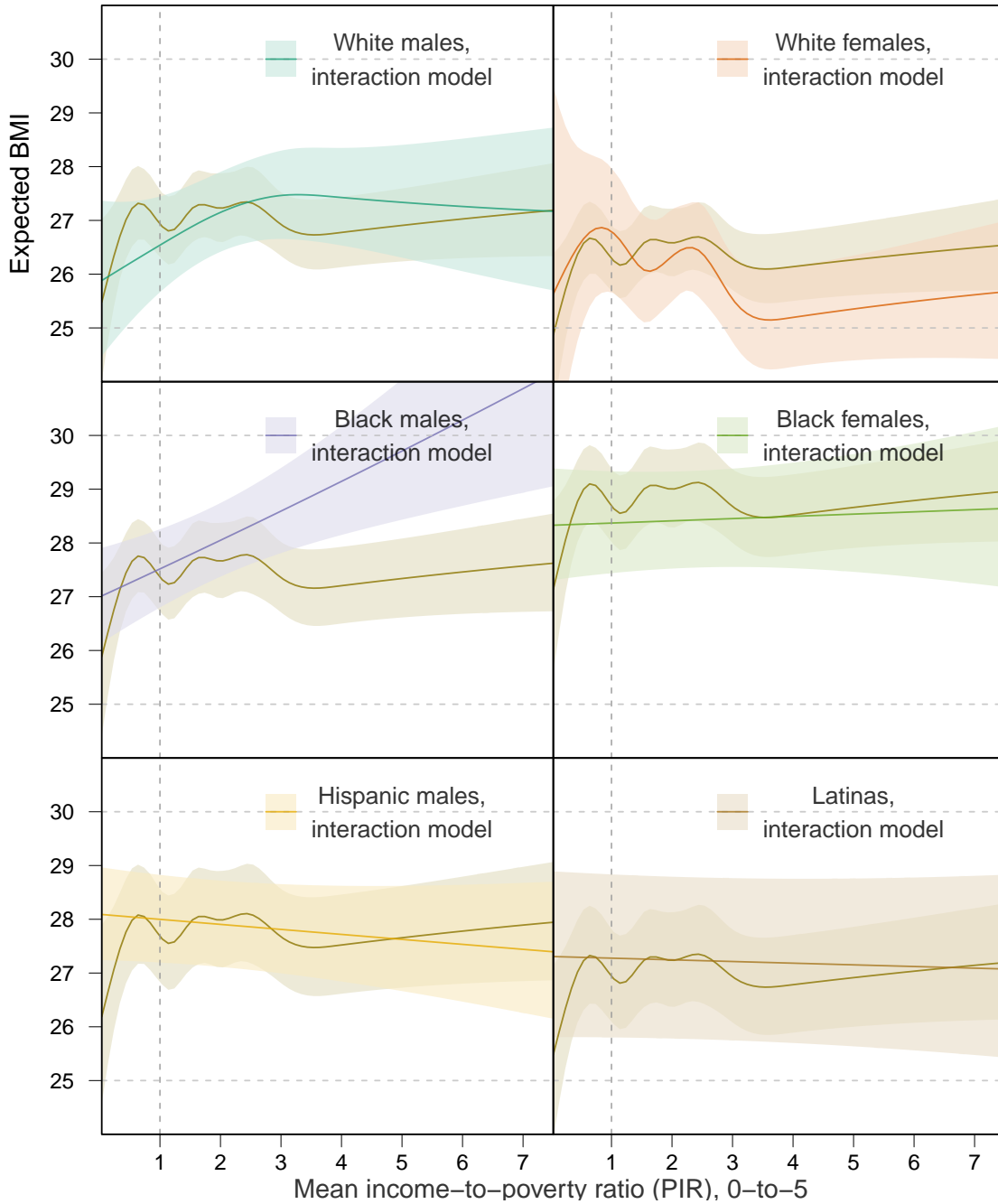
above around 275% of FPL, and particularly above 450%, are also expected to be at risk for having a BMI above 30. White females above around 325% of FPL and Hispanic females above approximately five times the poverty level, *ceteris paribus*, remain most likely to be of normal weight. This analysis provides evidence that additional spending power among those with lower incomes may improve adult health, at least as represented by BMI, only for females, while marginally more childhood income appears to be associated with additional weight for black males.

#### 4.3.2 *The effects of average income within developmental periods*

As noted, many theories imply that the health effects of SES or adversity might be more prominent when they occur in early childhood, which may indicate a more irreversible mechanism than if the key period were during adolescence. In order to have the current exploration of the curvilinear effect of childhood income on later BMI contribute to research on such timing models, I expand the basic model (4.1) and the analyses above to simultaneously fit separate splines for the effect of average income within each of three developmental periods. I estimate the developmental period spline models for the same trimmed sample, eliminating those with the highest and lowest observed values of childhood permanent income. (Doing so creates little in the way of notable change versus the splines estimated on the full sample.) For illustration, I present the expected BMI holding income in the other two periods constant at its sample-wide average, again comparing a sample-wide or average spline effect estimated across all groups with a group-specific spline. Thus, in Figure 4.14 I present the expected BMI for each of our six gender-by-ethnicity groups for the same profile as above over the same range of possible average income during early childhood, holding average incomes during middle childhood and adolescence constant.

As seen in Figure 4.14, even after eliminating the observations with reported average childhood income of \$0 who seem to have driven the “two poverties” effect of overall childhood income above, the overall model finds non-linearity in the effect of marginally more income within low income levels early in life, across all groups. (Note that there remain others with an average net family income of \$0 from the birth year through the year the

Figure 4.14: Marginal effect of mean income from 0 to 5 years of age, trimmed sample



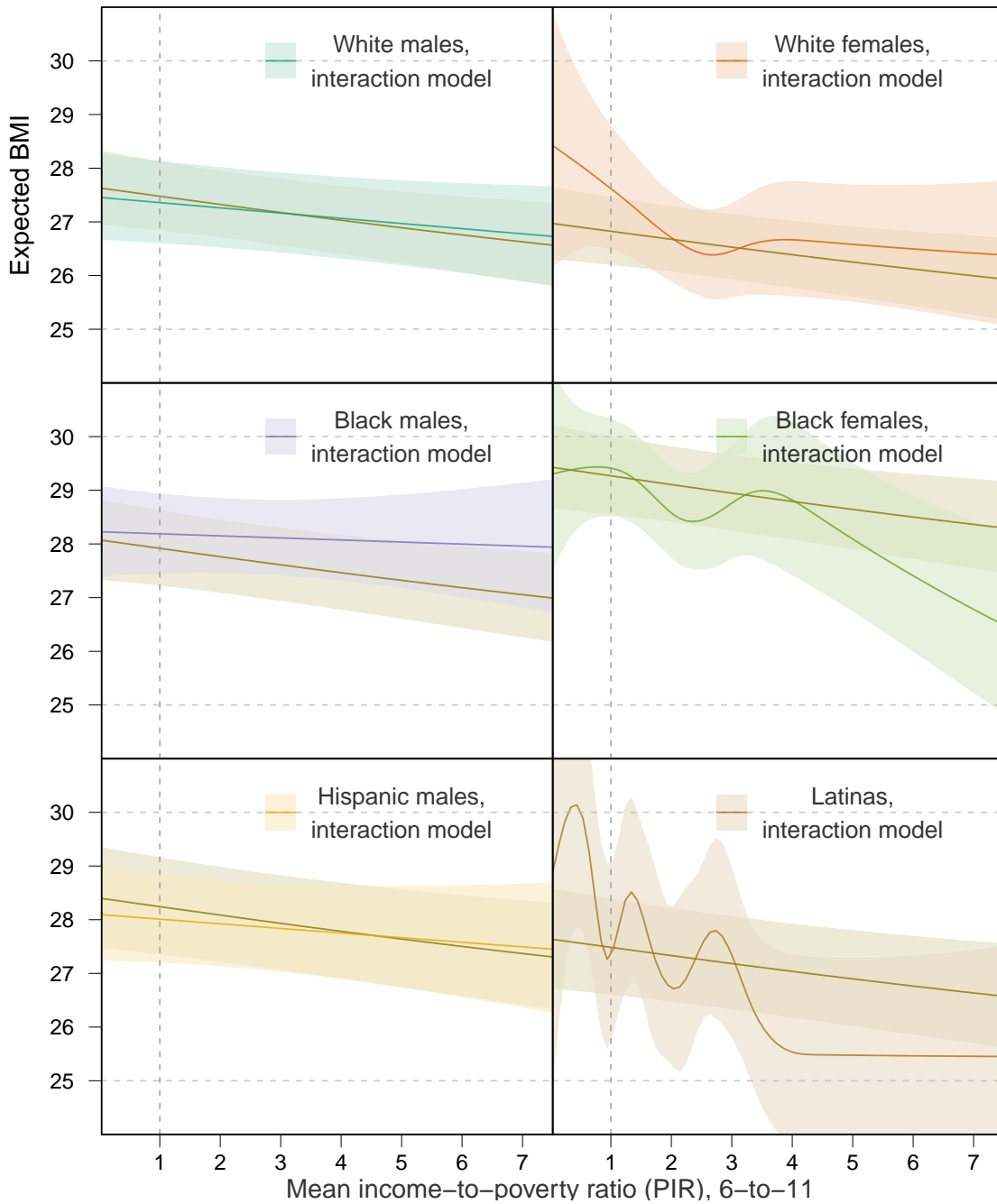
subject turned 5.) The lowest expected BMI is associated with the lowest levels of average income during early childhood. Elevated BMIs are also associated with mean incomes dur-

ing early childhood in the range of around 150% to 280% of FPL in addition to between around 50% and 100% of poverty, although the confidence interval indicates this range of elevated risk could well extend from below 50% to 300% of poverty. Furthermore, higher levels of income, above 350% of FPL, are associated with *higher* adult BMI, although the magnitude of this effect is small, at least up to 750% of poverty, and the confidence interval does not rule out zero effect above around 50% of poverty. There is little evidence that low income during early childhood is associated with elevated adult BMI over the income range considered here, and instead an indication that, across all gender and ethnicity groups, additional family income in this range is generally associated with higher BMIs in adulthood, on the average.

Most of the gender-by-ethnicity groups do not exhibit patterns that radically differ from this overall model. White males contribute to the positive gradient with higher income below around 280% of FPL, and while the point estimates imply a slight downward trend above 300% of FPL, the wide confidence intervals do not rule out a positive effect of more income. Black females and Hispanics exhibit little effect of early childhood income. The main departure appears to be for black males: Controlling for income in other periods (and maternal characteristics, age, year, etc.), black males exhibit a steep positive gradient and an effect of early childhood income quite different than the rest of the sample. For the profile illustrated here (25.5 years of age, etc., and holding income in the other two periods constant at the within-period means), black males with early childhood incomes above 500% of poverty appear to be at high risk of being obese in adulthood.

In contrast to the curvilinearity in the marginal effect of income in early childhood between 50% and 300% of FPL seen overall and among white women, the overall model shows a modest protective effect of income in middle childhood. As seen in Figure 4.15, the confidence intervals for this effect indicate that the overall trend is likely little different from no effect. White females may particularly benefit from additional family income up to around 200% of poverty, but note that the confidence interval for the group-specific spline expands rapidly with average middle childhood income below poverty level. Latinas may

Figure 4.15: Marginal effect of mean income from 6 to 11 years of age, trimmed sample



see little protective effect of more income in middle childhood above approximately 350% of FPL, while black females may have lower adult BMI with middle childhood income above

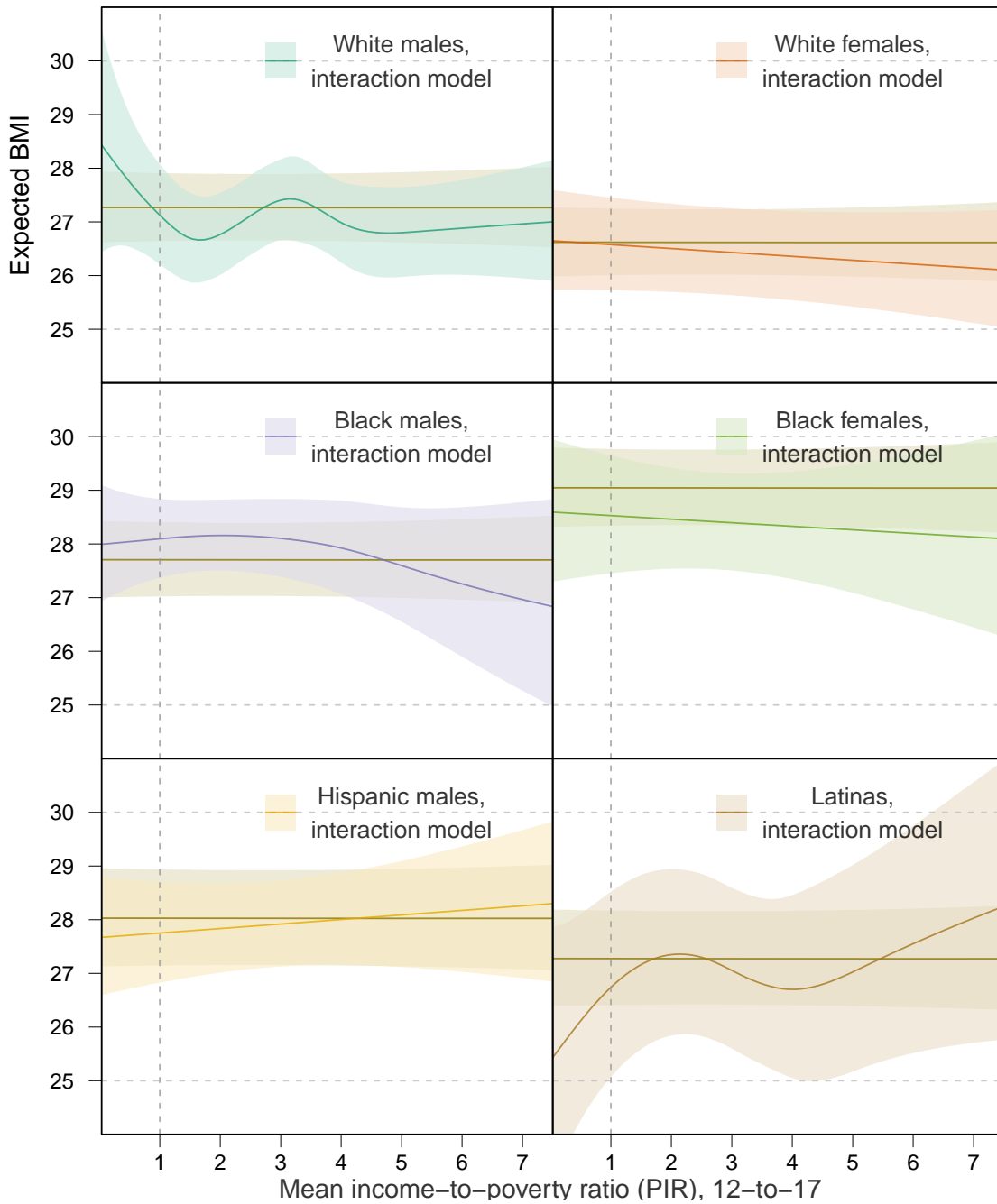
that same level, controlling for income in early childhood and adolescence, but, as for all groups, there is little evidence the group-specific effect is significantly different than the modest (and more precisely estimated) effect of the overall model. Family income in middle childhood appears to have little influence on adult BMI.

Similarly, the overall effect of average income during adolescence is one of no effect on adult BMI. As seen in Figure 4.16, there is some evidence individual groups depart from this overall null effect, but the rather wide confidence intervals imply the true group-specific effect may indeed be no different than 0. White males who experience the lowest levels of family income during adolescence may be more likely than other groups—particularly Latinas—to have higher adult BMI, while relatively higher income in adolescence—above 600% of poverty—may confer a substantially more protective effect on black males. While the confidence interval indicates the slope of income in this range could well be 0, note that the positive effect of income for black males seen in all childhood (see Figure 4.10) and in early childhood (Figure 4.14) is not evident in middle childhood or adolescence.

#### **4.4 Conclusions and implications**

“Obesity researchers increasingly believe that material disadvantages best explain the spread of obesity among poor people” (Ambinder, 2010, p. 76). This is but one example of how poverty or low income has come to be an assumed causal factor in the rise of obesity. Much of the research underlying this conventional wisdom connects current poverty or wealth to child or adult adiposity (e.g., Anderson, Butcher, & Schanzenbach, 2007; Hajat, Kaufman, Rose, Siddiqi, & Thomas, 2010; Kant & Graubard, 2007; Ogden et al., 2010; Storey, Forshee, Weaver, & Sansalone, 2003). A contemporaneous relationship between income and BMI, however, may be due to the BMI causing the income or to the income causing the BMI (or even a circular effect, as described by Kawachi and colleagues [2010]). Both of these causal paths could also be disaggregated into developmental or childhood effects versus more recent, within adulthood effects. Concluding that low childhood income is not a driver of adult adiposity does not inherently conflict with evidence that economic conditions in adulthood do influence BMI. Such a conclusion merely conflicts with conclu-

Figure 4.16: Marginal effect of mean income from 12 to 17 years of age, trimmed sample



sions that childhood is the period in which to concentrate intervention and policy levers to reduce the rise of obesity and with parallel conclusions that the effects of childhood

economic deprivation are irreversible.

In this analysis, I explored in a flexible fashion the influence of childhood income (specifically, family income as a multiple of the appropriate poverty level) on adult BMI, controlling for a rich set of covariates capturing economic and social context as of the subject's birth. The results indicate that overall lower income has little effect on adult BMI: While I reproduce the usual negative gradient, the effect is on the order of less than one BMI point ( $1 \frac{kg}{m^2}$ ) as average income decreases from 7.5 times poverty level to poverty level. This analysis moved beyond this simpler model and explored why childhood income indeed appears to have little effect.

One explanation is that the effect of marginally more income varies by group. The protective effect of more income—the usual negative gradient—is seen only among women and only, at least for white and Hispanic women, in certain ranges (below around 400% of FPL). Overall childhood permanent income has little effect on the adult BMIs of white and Hispanic men. Most striking is that the effect of more income, at least up to 7.5 times poverty level, is positive for black men: Adult African American males who grew up in higher income households have higher BMIs, on the average, than their peers who grew up in lower income households. Thus, the negative gradient appears to apply only to women, and the opposite effect occurs for black males. This is at least a partial explanation for why childhood poverty has essentially no relationship with adult BMI, as low income during childhood appears to be related to lower adiposity among black males and has no effect on the adult BMI of white and Hispanic males.

Second, the timing of income appears to matter. The positive effect of income for black males overall appears to be due solely to the effect of more income during early childhood, here defined as the birth year through the year the subject turned 5. In fact, there is some evidence that higher income in adolescence, at least between 400% and 750% of FPL, is protective for African American males, while middle childhood income has no effect. White males and females also exhibit a positive effect of more income at low levels of early childhood

average income, although for females this appears to peak at just below poverty level. The more precise overall model, which averages the marginal effects of income across groups, finds a general positive trend of more income in early childhood. Higher levels of income during the first six years of life appear to be associated with higher adult BMIs, particularly for black and white males and white females. This may be related to the results of studies that track obesity prevalence from infancy to young adulthood and find that the probability of still being obese in adulthood decreases the earlier the onset of childhood obesity (Daniels, 2006). If higher income children do indeed have lower current BMIs (Anderson et al., 2007; Ogden et al., 2010; Strauss & Pollack, 2001), a substantial proportion—particularly among black males—may gain weight later, weakening the correlation between childhood obesity and adult obesity.

Note that early childhood for this sample occurred largely (but not completely) during the 1980s, or early in the trend in the last 30 years towards broad availability of centrally-produced foods (Cutler, Glaeser, & Shapiro, 2003) and large increases in consumption of added sugars in foods and beverages (Marriott, Cole, & Lee, 2009; Tappy & Le, 2010). That is, during this period more income (particularly more income among those with an average household income of less than 7.5 times poverty level) may well have been associated with increased caloric consumption. Much of the positive effect found here of income in early childhood on later BMI may have occurred during the birth year, as perinatal over-nutrition has been linked to metabolic and physiological changes in the developing child that contribute to later adiposity and related conditions such as diabetes (Cohen et al., 2010; Lawlor & Chaturvedi, 2006). Under-nutrition during pregnancy and low birth weight are also associated with later adiposity (Caprio et al., 2008; Fall, 2011; Yanping et al., 2011).

Only middle childhood exhibits a modest negative gradient overall and particularly among white females at lower levels (below around 250% of poverty), black females at higher levels (above around 4 times poverty), and Latinas at lower levels (below around 350% of FPL). That is, the negative gradient of overall income appears to be limited to females and to income in middle childhood, but, again, the effect is modest at least up to 7.5 times the

poverty level. Higher family income in adolescence appears to have little effect. If low income during middle childhood was associated with consumption of energy-dense foods and thus high caloric intake (Drewnowski, 2007, 2009), this over-nutrition may have contributed to earlier puberty among girls (Vandeloo, Bruckers, & Janssens, 2007) which may in turn contribute to higher adult BMI (Cohen et al., 2010; Laitinen et al., 2001). Alternately, lower income in middle childhood might be associated with living in less advantaged school districts that might be more likely to turn to snack and sugar-sweetened beverage sales to augment limited school budgets (Anderson & Butcher, 2006; French, Story, & Jeffery, 2001; Johnson, Bruemmer, Lund, Evens, & Mar, 2009), but the viability of this explanation relies on this affecting girls more than boys and the availability of such foods in middle and high schools being independent of income.

Contrary to the conventional wisdom, then, low income during childhood does not appear to be strongly related to higher BMIs in adulthood. As noted, this does not preclude a causal effect of income on BMI. Rather, it indicates that focusing on measures that try to counteract low income during childhood—particularly early childhood and adolescence—in the fight against obesity may be misguided to the extent that the forces set in motion by marginally more income during the 80s and 90s are still in place today. While economic deprivation in childhood has varied pernicious effects, the effects on body mass appear to be either mild or moderated by later experiences that may be a better focus of research and policy. Once again, the obesity problem has no simple answer at the public health level. In their review of childhood weight, Strauss and Pollack (2001) concluded

Childhood overweight reflects the convergence of many biological, economic, and social factors. Overweight arises from multiple causes, some as intimate as the family dinner table, others as seductive as television of the latest children's video game. Provision of high-fat meals and snacks in school settings is both a powerful temptation and a clear signal of accepted nutritional norms. . . . No one intervention, by itself, is likely to produce large reductions in the prevalence of obese or overweight children. Like adolescent smoking, teen pregnancy, and youth violence, childhood overweight is prevalent because it arises from deeply

rooted behaviors and from social practices that are hardly confined to children (p. 2848).

Apparently these forces are similarly hardly confined to the poor.

#### **4.5 Next steps**

The focus of the current analysis is on the total effect of childhood income. No attempt was made to investigate the effect—whether mediators or moderators—of variables that come chronologically after childhood. For example, maternal weight status is a powerful predictor of child weight (e.g., Classen, 2010; Daniels, 2006; Laitinen, Power, & Jarvelin, 2001) but is likely endogenous to the family income–child adiposity relationship. Constructs such as family structure, or more directly measuring parental monitoring, may explain the positive effect of income during early childhood on the BMI of African American males. Similarly, this analysis explored only part of the timing of income in creating future health. One way of more fully addressing the potential causal effect of income on BMI would be to extend the current analysis with one or more periods in adulthood (up to some number of years before the BMI is measured), to assess and compare the effects of prior income throughout the life span.

As with any analysis, the procedures employed here would benefit from increased power. The current analysis relies on NLSY data up to the 2010 wave; replication incorporating the 2012 administration of the Child and Young Adult survey might result in more precise estimation with some added sample members. Furthermore, the covariates used here paralleled those of my previous work examining the total effect of child poverty dynamics on adult BMI. A more concise set of controls (only age and year in addition to gender and ethnicity) produced tighter confidence intervals (not shown), so replication of this analysis might benefit from some pruning of the covariate list, but at the loss of accounting for a rich set of factors likely associated with both childhood income and development of adiposity.

Average income, even divided into developmental periods, remains a monolithic measure that may ignore important dynamics, primary among them being the variability of that

income. Two families with average incomes just above poverty level may reflect very different experiences if one family's income remained within a small range of that average and the other's a much broader range. A more unpredictable level of income and thus consumption likely represents more stress and/or more stocking up when possible than with a consistent level of income. The next step for this project is to partially replicate the analysis of Smith and colleagues (Smith, Stoddard, & Barnes, 2009) and examine the effect of income instability net of permanent income in a life course perspective.

## Chapter 5

**PART 3: INCOME INSTABILITY IN CHILDHOOD AND ADULT  
ADIPOSIITY****5.1 Background**

Although there is little doubt that there are genetic influences on obesity, excess body fat is essentially about caloric intake exceeding expenditure over time. As for causing obesity itself, “the genes or gene variants that would support [an obesity genotype] hypothesis have not been identified” (Caprio, et al., 2008, p. 2214), and obesity dynamics have simply occurred too quickly for changes in genetics to be a cause (Bleich, Cutler, Murray, & Adams, 2008), leaving us with a broad and varied literature about potential influences on the proximal causes of diet and exercise. Excess body fat represents a marker of important health behaviors which are independently associated with health outcomes; a risk factor for cardiovascular disease, diabetes, and some types of cancers (Danaei, et al., 2009; Visscher & Seidell, 2001); and a contributor to poor social, psychological, and economic outcomes (e.g., Cawley, 2004; Cutler, Glaeser, & Shapiro, 2003; Daniels, 2006). Given these potential sequelae, it is important to take a rigorous scientific approach to identify what influences should be the target of policy.

Socioeconomic conditions are frequently cited as important risk factors for excess adiposity. Those with lower levels of income and education are more likely to be obese (Caprio et al., 2008; Cutler, Glaeser, & Shapiro, 2003; Kant & Graubard, 2007; Storey, Forshee, Weaver, & Sansalone, 2003). Socioeconomic background—usually measured via indicators of parental occupation (often at birth) and/or education—has been linked to later adiposity (Baltrus, Everson-Rose, Lynch, Raghunathan, & Kaplan, 2007; Hayward & Gorman, 2004; Laitinen, Power, & Jarvelin, 2001; Langenberg, Hardy, Kuh, Brunner, & Wadsworth, 2003; Power et al., 2005; Senese, Almeida, Fath, Smith, & Loucks, 2009). Early poverty or income experiences are associated with BMI in late adolescence (Evans & Kutcher, 2011;

Wells, Evans, Beavis, & Ong, 2010) and young adulthood (Lee, Harris, & Gordon-Larsen, 2009; Ziol-Guest, Duncan, & Kalil, 2009). Some, but not all, causal modeling efforts relying on endogenous income variation have linked lower income to higher weight (Smith, Stoddard, & Barnes, 2009), but this effect may be reversed at the lower end of the income scale (Akee, Simeonova, Copeland, Angold, & Costello, 2010; Schmeiser, 2009). Obesity likely joins other health disparities in both contributing to disadvantage and resulting from disadvantage in a process of cyclical causation (Kawachi, Adler, & Dow, 2010) and compounding disadvantage (Braveman et al., 2011).

Most studies of socioeconomic status (SES) and health outcomes rely on “on relatively crude measures of socioeconomic status, such as manual labor versus nonmanual labor social class as indicated by fathers’ occupational status” (Ziol-Guest et al., 2009, p. 528) that belie the variability individuals and families face over time. Parental occupation or education at birth, while likely correlated with later experiences, would seem to tell us little about the diet and exercise habits engendered in the developing child. Research addressing child poverty often relies on only an indicator of ever being in poverty “at some nonspecific point before age 18 years and most often [assessed] retrospectively in large-scale population-based surveys” (Ziol-Guest et al., p. 528). Poverty experiences, wealth, income, and even parental education all change over time, and “circumstances at the time of measurement [may not] reflect one’s position over a lifetime” (Hayward & Gorman, 2004, p. 88). In this paper, I combine two perspectives on the dynamic experience of childhood economic experiences, one focusing on the timing or chronicity of experiences and another focusing inherently on instability of resources. I address not merely average income or whether the individual was ever poor as a child, but the instability of income in different developmental periods.

### *5.1.1 Stress and adiposity*

Many of the exposures thought to link SES to health outcomes, from relationship and housing instability to inconsistent parenting to poor household maintenance to neighborhood crime, inherently involve change and subsequent psychological and physiological stress. The allostatic load conceptualization, for example, embodies how extended and repeated

stress responses harm physiological systems, including those that regulate metabolism.

The premise is that while relatively short-term fluctuations in levels of physiologic activity are necessary for the body to respond successfully to stimuli (e.g., the fight or flight response in the face of various types of danger), excessive fluctuations either in terms of the extent, duration or frequency (e.g., responding to perceived danger' everywhere) can result in wear and tear on the body's regulatory systems (Seeman, Epel, Gruenewald, Karlamangla, & McEwen, 2010, p. 226).

Yet most studies of SES-related factors and health outcomes have focused on states rather than changes in SES. "Less discussed (or tested) is the possibility that multiple changes in SES (whether upward or downward), particularly if they are drastic differences, throughout the life course may negatively impact future health outcomes by creating unstable and unpredictable environments" (Cohen et al., 2010, pp. 47–48). One can imagine that a childhood at a constant level of poverty might be relatively less stressful and have fewer negative long-term effects than one characterized by wide fluctuations in the supply of nutrition, housing quality, attentive and authoritative parenting, etc. Insecurity—particularly the subjective experience of the probability of a loss and resulting (relative) famine—represents a stressor that may result in weight gain in adults, the argument essentially connecting economic circumstances to psychological and neuroendocrine systems connected to appetite, dietary choices, metabolism, and weight gain. Research findings supporting the negative effects of childhood SES status might actually be measuring the effects of attendant instability. We know, for example, that lower income families experience higher volatility of income over time (Gundersen & Ziliak, 2003). Furthermore, "even if many people exit poverty each year, they [remain] at substantial risk of reentry" (Jantti, 2009, p. 196).

Instability of resources itself may thus capture much of the apparent relationship between low income and obesity. Smith and colleagues (2009) investigated the effect on obesity of recent individual-level economic security in males net of income levels. The analysis found a significant predictive relationship for three of the four insecurity measures tested

(except probability of being under the poverty line): “(1) a 1 [percentage point] increase in the probability of becoming unemployed causes an increase in body weight of over half a pound, (2) for each additional year in which real income drops by at least 50%, body weight increases by 5 pounds, [and] (3) a decrease in  $R^2$  (a measure of income stability) by 0.1 units corresponds to an increase in body weight of 2.4 pounds” (p. 12). Cross-national analysis has similarly connected nationwide measures of economic insecurity to obesity levels (Offer, Pechey, & Ulijaszek, 2010). These investigations of income insecurity address one of the unanswered questions laid out by Kawachi and colleagues (2010):

Distinguishing the effects of temporary income (income shocks) from the effects of permanent income. Income shocks may be associated with more harmful behaviors in the short term (e.g., cigarettes and booze become more affordable), whereas increases in permanent income may improve a person’s prospects for the future and lead to increased incentives to invest in their longevity (p. 65).

Financial stress may also be defined as being in debt or having other current financial difficulties. Research shows, for example, that having credit card debt is predictive of being overweight or obese for men and women, while women with trouble paying bills are more likely to be at least overweight and more likely to be obese (interestingly, men with bill trouble were less likely to be overweight; Averett & Smith, 2012). There is thus some evidence that higher levels of economic insecurity during adulthood are associated with obesity.

### *5.1.2 Potential mechanisms*

Chronic stress chronically activates stress-related neurochemical pathways, leading to dysregulation of important physiological systems (Seeman et al., 2010) and enhancing the reward inherent in food (Huang, Drewnowski, Kumanyika, & Glass, 2009). The concepts of “comfort food” and “self-medication” via food intake refer to using food as a coping strategy (e.g., Nguyen-Rodriguez, Unger, & Spruijt-Metz, 2009; Offer et al., 2010; Olson, Bove, & Miller, 2007; Smith et al., 2009). The relationship between stress and weight gain

appears to be moderated by tendency towards such emotional eating (O'Connor, Jones, Conner, McMillan, & Ferguson, 2008), perhaps in the perceived absence of other stress reduction techniques. Indeed, research shows that it is people already in the upper part of the BMI distribution who tend to gain weight with stress, while the thin tend to lose weight (Kivimaki et al., 2006), and animal models show that stress induces weight gain only in the presence of high calorie foods (Kuo et al., 2008). Excess weight itself may directly or indirectly cause stress and contribute to further overeating or under-exercising.

Numerous authors have written about the evolutionary predisposition of humans to eat substantially when food is available, storing energy for times of famine (e.g., Ambinder, 2010; Pollan, 2006; Popkin, 2009; Ulijaszek, 2007, 2008).

The reason humans and other animals evolved the ability to store body fat is presumably because it was necessary to survive periodic food shortages. The evidence for this is surprisingly strong. It has been demonstrated again and again, for instance, that animals in natural environments face very real periodic starvation risk, and that such risk is a strong predictor of fattening behavior (Smith et al., 2009, p. 1).

The effect in modern humans is not yet clear. While many but not all cross-sectional studies of food insecurity or insufficiency find a relationship with obesity (Dinour, Bergen, & Yeh, 2007; Larson & Story, 2011), a longitudinal study of food insecurity (using the US Department of Agriculture definition) and adiposity found no effect of baseline food insecurity on weight gain (Jones & Frongillo, 2007), which may be due to weakness of this retrospective, self-report measure. A relationship between food availability and food intake has been found, however, in response to the monthly cycle of food stamps benefit distributions (as reviewed in Dinour et al., 2007; Rosenkranz & Dzewaltowski, 2008; Smith et al., 2009; Zagorsky & Smith, 2009). Lower income families may face frequent diet changes and constriction “in the week or two before the next check arrives” (Engler-Stringer, 2010, p. 222). “Studies indicate that bingeing often follows periods of food deprivation... and that binge eating is positively associated with obesity because it can alter metabolism

in ways that promote fat accumulation” (Zagorsky & Smith, p. 248). Thus, whether due to a propensity for comfort seeking in food (emotional eating), chronic activation of neuroendocrine systems, or as an evolutionary response to perceived risk of famine, humans may respond to economic stress by overeating.

Developmentally, excess calories in utero and early exposure to energy-dense foods appear to result in biological changes that affect future diet preferences as well as how that diet is metabolized. Paradoxically, low weight at birth and childhood malnutrition may have similar effects. The biomedical literature discusses the “programming” effects of both over- or under-nutrition in utero and in the early years of childhood, which are thought to operate via epigenetic modifications, metabolic or endocrine changes, insulin resistance or glucose tolerance, and other mechanisms that contribute to future weight and health (Fall, 2011; Li, Goran, Kaur, Nollen, & Ahluwalia, 2007; Martin-Gronert & Ozanne, 2010; Tarry-Adkins & Ozanne, 2011). Studies have shown, for example, that adults exposed in utero or in early childhood to famine conditions are more likely to be obese or be diagnosed with the closely related metabolic syndrome (at least three of elevated triglyceride levels, elevated glucose levels, lower high-density lipoprotein cholesterol levels, high waist circumference, or high blood pressure; Fall, 2011; Olson et al., 2007; Yanping et al., 2011).

Olson and colleagues (2007) have speculated that the relationship between food insecurity and obesity is due to “the experience of poverty-associated food insecurity in early childhood and the eating practices and attitudes resulting from food insecurity,” including emotional eating (p. 199). Their qualitative data revealed ways in which childhood food deprivation might affect future diet choices: Some of their mothers developed an emotional attachment to having food available for their current families, resulting in stocking up; for some, “the early experiences resulted in the removing of economical and nutritious foods from women’s own and their families’ diets” (p. 203); and for some, the emotion attached to enjoying food whenever it was available reinforced and heightened the reward associated with eating. These effects can result in obesity via the Costco effect—the effect that food salience and large package size have on consumption (French et al., 2001; Wansink, 2004)—of stocking

up, rejecting the economical but still somewhat nutritious choices of our parents in favor of today's energy-dense cheap foods, and turning to food for comfort. Furthermore, the poor mothers in Olson and colleagues' investigation poignantly illustrated the binge-promoting effects of periodic bounty:

Immense excitement and pleasure sometimes accompanied the influx of food into these food-insecure households following the arrival of food stamps, a paycheck, or a gift of food. Both adults and children appeared to increase their intake of food once it became available again, increasing their consumption of preferred foods in particular, which had been especially missed. . . . These findings indicate that periods of overeating by both children and adults sometimes accompanied the influx of food into food-insecure households (p. 204).

### 5.1.3 *Modeling the effects of childhood economic conditions on adult adiposity*

There is thus beginning evidence that a history of instability of economic resources may drive part of the usual SES-obesity relationship. Most investigations of the potential causal effect of SES factors, however, rely on simple measures that do not capture the dynamics of SES-related experiences. Beyond whether childhood economic conditions affect adult adiposity, a key set of questions revolves around how much exposure and when is necessary. The answers to these questions will offer much insight as to potential mechanisms. Many discussions of the SES-obesity relationship, or the broader relationship between SES and health, do not move beyond the simple consideration of *any* experience of deprivation or some indicator of “average” SES that stands for all childhood experiences.

The developmental health disparities literature often relies on two main models of how childhood SES might affect future health. The first is a *timing* or developmental period model, which focuses on the effects of SES-related factors—e.g., average income or at least one experience of poverty—during sensitive or critical periods during the child's development (Cohen et al., 2010). In this model, the effects of exposures (usually states or events) during these critical periods are thought to be irreversible, and thus independent of the effects of

later, particularly adult, SES. Conroy and colleagues also call such a conceptualization the “latency” model, “highlighting that the health effects of these critical periods may not be recognized until much later” (Conroy et al., 2010, p. 155). The fetal origins or fetal programming hypothesis is a variant of the critical periods model, focusing on the prenatal through immediate post-partum periods. Ziol-Guest, Duncan, and Kalil (2009) tested the effects of income in a single prenatal year combined with the year of the subject’s birth, early childhood (ages 1 to 5), and later childhood (ages 6 to 15). After finding that more income (at low levels) in the earliest period was associated with adult BMI, overweight prevalence, and obesity prevalence, but income in the other two periods was not, the authors concluded their results were “consistent with the hypothesis that fetal programming induced by early stimulants and insults has long-lasting implications for physiology and disease risk” (p. 531). By not controlling for the mother’s prior experiences, however, this conclusion may have been an artifact of correlation between the mother’s prior experience and that in the prenatal and birth years. If so, the effects may have been less physiological than psychological—not so much about epigenetic programming and endocrine development itself but more about the mother’s nutrition and exercise habits developed in reaction to economic experiences just before the child’s birth. For simplicity I label as timing models specifications that estimate the effects of dichotomous indicators of whether an event ever occurred within a developmental period, regardless of how often.

In contrast, another main approach is to ignore timing and focus on the duration and extent, or dosage, of deprivation. This conceptualization is called the *accumulation* (Cohen et al., 2010) or cumulative effects model (Conroy et al., 2010). This model is also known as the “pathways” model due to the cumulative nature of multiple exposures in affecting health, often creating a correlated if not causal chain of events, as when maternal smoking results in low birth weight results in neurochemical changes that promote affinity for sugary foods (in the context of high availability). Allostatic load is another manifestation of hypotheses focused on the cumulative effects of exposures on later health functioning (Seeman et al., 2010). The work by Wells and colleagues (Wells et al., 2010) tested the accumulation model by including a variable that measured the proportion of time (in 6-month intervals) between

birth and age 9 that the subject lived in poverty.

The current analysis effectively modifies and extends analysis of the dual income effect—separating permanent income from (fear of) income shocks—of Smith and colleagues (Smith et al., 2009) in a developmental context, with a focus on childhood dynamics of instability. I apply the timing and accumulation models and extend them by examining a hybrid model of accumulation within periods, to explore the insights gained from considering dynamic experiences versus a crude measure of ever experiencing some form of income instability. I also extend these models by incorporating maternal instability before the child's birth to test the effect of childhood instability over and above that already experienced, and perhaps incorporated into diet and exercise practices, by the mother.

## **5.2 Data and methodology**

Since 1979, the National Longitudinal Survey of Youth has been following a representative cohort, originally aged 14 to 22, annually through 1994 and biennially to date. The NLSY79 Children and Young Adults (CYA) study, started 1986, follows children of mothers in NLSY79, allowing for linking mothers and offspring, many of whom are now adults. The NLSY is commonly used to examine income and related phenomena as well as health indicators, and provides a rich dataset for linking childhood and pre-birth conditions to second generation outcomes. Among the variables created by the managers of the study are a Total Net Family Income variable, combining income across all sources from all co-resident family members related by birth or marriage (but excluding unmarried partners), and a Poverty Status indicator that compares the previous year's family income to federal poverty thresholds based on family size and location. As family income includes the value of income supports such as food stamps and EITC, this means the NLSY measure assesses a form of adjusted income poverty (it does not adjust for taxes paid) and thus categorizes fewer families as poor than the poverty measure produced by the US Census Bureau.

Reported incomes were inflated to 2010 dollars using the Consumer Price Index and normalized to account for family needs by dividing by the square root of family size, a

common choice for adjusting income levels across families of different sizes (Coulter, Cowell, & Jenkins, 1992; Cowell & Mercader-Prats, 1999; Iceland, 2003). To calculate childhood and pre-birth income instability, the mother's reported family income and poverty status were translated from calendar-year-relevant to birth-year-relevant data (e.g., translating mother's income in 1994 to income in the year the child turned 15).

Similar to Smith and colleagues (2009), I examine the effects of three distinct measures of income instability. First, one may conceptualize income volatility as variation from the time trend of income. Specifically, for each individual I regress per capita household income on a year indicator as follows, separately for each relevant time period:

$$Y_{it} = \alpha_i + \beta_i t + \varepsilon_i \quad (5.1)$$

where  $i$  indicates individuals and  $t$  a simple count of years. Smith and colleagues used the  $R^2$  goodness of fit measure from each individual's regression as an instability measure along with the  $\beta_i$  to indicate trend and direction of income over time. I use  $1 - R^2$  (and multiply by 100) so that higher values indicate more instability. Note that this first instability measure captures the accumulation of instability over a whole period, and cannot be used in models requiring a dichotomous indicator of any instability within the period.

Our potential mechanisms for a relationship between instability of material resources and obesity largely are concerned with the effects of risk of scarcity. It may be only *downward* volatility that is associated with future obesity, whether via adoption of energy-dense foods, emotional eating, or other changes to intake patterns, or changes in expenditure. Large decreases in income may be more important than the overall level of instability, which may include increases. (I leave aside for now the possibility that a pattern of early instability, cementing habits of increased intake, is most predictive of obesity when later income increases allow for purchasing greater quantities of energy-dense foods.) I use the rate of large income drops, with each year's income judged against the prior available income observation. Smith and colleagues used a seemingly large threshold of 50% drops in income, and reported that using 10% decreases produced similar but somewhat attenuated results. I use 25% for income decreases (but also test 10% and 50% drops for sensitivity).

The third instability measure attempts to capture risk of an income decline that moves the family into poverty. Poverty instability is measured as the proportion of years within the period the family was at risk of falling into poverty (i.e. was not in poverty the previous year) and did indeed become poor, using the NLSY poverty indicator. If during the period the observed data indicate never *becoming* poor—whether because the collected poverty indicators indicate always being poor, never being poor, or leaving but not re-entering poverty—the poverty risk is set to 0. In contrast, if the observations within the period indicate returning to poverty immediately after every year in which the family left poverty, the value for the poverty instability measure will be 1. To illustrate, in each of the examples in Table 5.1, poverty transitions are shown over a five year period with “1” indicating a year when the family is “poor” and “0” indicating “not poor”.

Table 5.1: Poverty entry risk examples

Example	Year					Probability of poverty entry
	1	2	3	4	5	
1	1	0	1	0	1	This person had 2 years (2 & 4) out of poverty, and thus 2 opportunities to become poor. Both non-poor years were followed by being in poverty, so the probability of entering poverty is $\frac{2}{2} = 1$ .
2	0	1	1	1	1	This person fell into poverty after the only year observed not in poverty, so the probability of entering poverty is $\frac{1}{1} = 1$ .
3	0	0	0	1	1	This person had 3 observed non-poor years, and fell into poverty after 1 of them. Probability of poverty entry is $\frac{1}{3}$ .
4	0	0	0	0	0	This person had 4 opportunities to follow a non-poor year with a year in poverty, so the probability of entering is $\frac{0}{4} = 0$ .
5	1	1	1	1	1	This person was not observed entering poverty, so the entry probability is set to 0.

The three measures capture different aspects of instability. A relatively linear downward trend in income may result in a high value for the second instability measure and a low value for the Instability 1 measure, but represent only a single crossing of the poverty threshold out of multiple opportunities to do so for the Instability 3 measure—which could be represented by Examples 2 through 4 in Table 5.1. Example 1, in contrast, might represent high volatility around a time trend and two large income declines—i.e. high on Instability 1 and Instability 3, moderately high on Instability 2—or could represent very small changes in income that just happen to move the family above and below poverty level—i.e. close to 0 on Instability 1 with 0 large income drops. Table 5.2 summarizes these different proposed proxies for resource instability.

Table 5.2: Summary of instability measures and dynamics models

	Income Instability 1	Instability 2: Income drops	Instability 3: Poverty instability
Dichotomous model		Any 25% income decreases, pre-birth & childhood	Any entry into poverty, pre-birth & childhood
Timing model		Any 25% income decreases, pre-birth & 3 developmental periods	Any entry into poverty, pre-birth & 3 developmental periods
Accumulation model	$1 - R^2$ , pre-birth & childhood	Rate of 25% income decreases, pre-birth & childhood	Probability of poverty entry, pre-birth & childhood
Hybrid model	$1 - R^2$ , pre-birth & 3 developmental periods	Rate of 25% income decreases, pre-birth & 3 developmental periods	Probability of poverty entry, pre-birth & 3 developmental periods

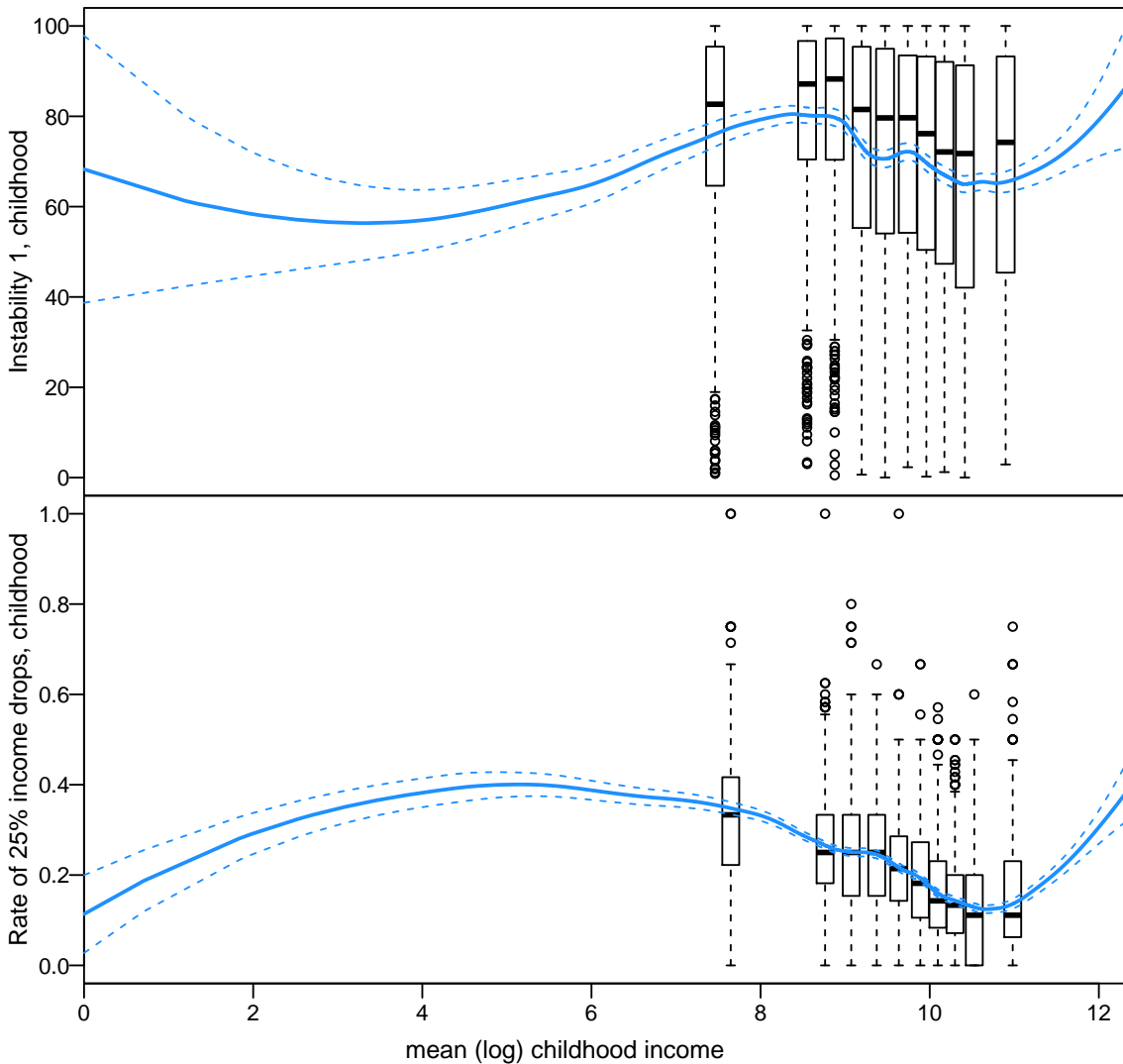
As noted, there is some evidence that income is more volatile among those at the lower end of the income distribution (Gunderson & Ziliak, 2003). This likely depends upon how volatility is measured. Among the proposed three measures of income instability here, poverty entry rate (Instability 3) will obviously be quite high among those adults with the lowest average childhood income, and 0 for most subjects with above average childhood permanent income. Figure 5.1 shows the distribution of the  $R^2$ -based Income Instability 1 measure and of the rate of 25% income decreases (Instability 2), both over all of childhood, via boxplots (marking the group median, interquartile range, etc.) within each decile of permanent childhood income (plotted at the decile mean) and via a continuous loess smooth approximating the local mean (with 95% confidence interval for the loess fit). The rate of income drops, but not the Instability 1 measure, does appear to be highest at the low end of the income distribution, although within the first decile (which is quite large in range) the loess fit indicates the very lowest observed mean childhood income values represent childhoods with relatively few income declines. For both measures, there is some evidence of a marginally lower level of observed instability as one moves from the second through the ninth decile. Overall, however, there is much overlap in the distribution of instability (and little correlation between instability and permanent childhood income), at least measured over all of childhood.

These indicators of income instability are entered into the following basic model,

$$\ln BMI_i = \beta_0 + \beta_1 PI_i + \beta_2 CI_i + \beta_3 \bar{Y}_i + \beta_4 P_i + \beta_5 C_i + \beta_6 T_i + \varepsilon_i \quad (5.2)$$

where  $PI$  and  $CI$  are the indicators of parental (up to 10 years before the year of the child's birth) and childhood (year of the child's birth through the year the child turned 17) instability,  $\bar{Y}$  is permanent childhood income,  $P$  and  $C$  represent vectors of parental and child covariates,  $T$  is a linear time trend capturing secular increases in BMI, and  $\varepsilon$  is the usual error term. For the developmental models—the timing and hybrid models—the childhood instability term is replaced by three terms representing early childhood (0 through 5), middle childhood (6 to 11), and adolescence (12 to 17). Note that the Instability 1 measure includes the relevant  $\hat{\beta}_i$  for each period from (5.1) as additional controls, such that

Figure 5.1: Childhood instability over income distribution: Boxplots by income decile and less smooth (with 95% confidence interval)



the hybrid model assessing the effects of the regression-based volatility measure becomes

$$\ln BMI_i = \beta_0 + \beta_1(1 - R_{pre,i}^2) + \sum_{a=1}^3 \gamma_a(1 - R_{ai}^2) + \lambda_{pre} \hat{\beta}_{pre,i} + \sum_{a=1}^3 \lambda_a \hat{\beta}_{ai} + \beta_3 \bar{Y}_i + \beta_4 P_i + \beta_5 C_i + \beta_6 T_i + \varepsilon_i \tag{5.3}$$

where *pre* and *a* indicate the pre-birth and three developmental periods, respectively.

The outcome in the above models is the natural log of the latest observed BMI. Exploration of potential non-linearity in the relationship between various key predictors and BMI, in the context of controls, indicated that this provided a better fit than a linear BMI specification. This transformation furthermore captures percentage growth and fits with the distributional dynamics of the rise of excess adiposity in the US: The distribution of BMI has been shifting upwards and, more dramatically, becoming more and more positively skewed (Anderson & Butcher, 2006; Flegal & Troiano, 2000). This skewing has resulted in greater growth in the probability of being obese conditional on being overweight than in the prevalence of overweight (including obesity) itself (Anderson & Butcher, 2006; Cook & Daponte, 2008). Before logging, the observed BMIs were Winsorized to the 1<sup>st</sup> and 99<sup>th</sup> percentiles to reduce the effects of outliers and influential cases.

The covariates in  $P$  and  $C$  above were selected carefully to account for potential factors that might influence both income and adiposity while allowing for estimating the total effect of childhood income instability, net of childhood average income. Unlike Smith and colleagues (2009), I do not include a baseline BMI. To further reduce endogeneity, the controls selected are either invariant (the interaction of child gender and ethnicity, mother's aptitude<sup>1</sup>), occurred largely before even the pre-birth period (education of mother's parents and oldest sibling, if any; mother's immigrant status and whether a foreign language was spoken at home during her childhood; whether she lived in the South or in a rural area at age 14; and the religion in which she was raised), or are otherwise plausibly exogenous (subject's age when the last BMI was observed).

As is common with studies considering income, I use a log transformation of each year's normalized income (adding \$1 to avoid taking the log of 0). This helps reduce heteroskedasticity and is also consistent with the individual's focus on relative change—e.g., an extra \$1000 means more to someone making \$10,000 than someone making \$100,000—and with the idea that growth in income is often experienced in percent terms (e.g., raises or interest). The permanent income control  $\bar{Y}$  in the models above is thus the average of the logged

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<sup>1</sup>Measured via the Armed Forces Qualifying Test (AFQT), administered as part of NLSY in 1980.

income observations over the subject's childhood. The income drops rate comprising the Instability 2 measure therefore counts decreases of greater than .288 in logged income<sup>2</sup>.

As with any survey, missing data pose a potential problem. Before transforming the data in reference to the child's birth year, the control variables and base variables upon which they are built were imputed using a multivariate normal regression framework (implemented using the R package *Amelia* [Honaker, King, & Blackwell, 2011]). The imputed covariates were indicators of any relatives (mother's mother, father, or oldest sibling) completing high school and completing any post-secondary education (as of 1979), whether the mother spoke a foreign language at home growing up, whether she lived in the South and whether she lived in a rural area at age 14, the religion in which she was raised, and her aptitude test score. I did not impute income and poverty data so as to not introduce stability into the economic histories of the subjects.

After extensive cleaning of height and weight data, there are 5046 young adults with at least two years of poverty indicators for both the pre-birth and childhood periods. The distribution of their BMIs (before Winsorization) and covariates describing the subjects and their mothers are presented in Tables 5.3 and 5.4, respectively. Descriptive statistics for permanent income and the instability measures appear in Tables 5.5 through 5.7. The data requirements for the different instability definitions mean substantial and varying portions of the sample are missing data. For example, calculating an  $R^2$  for the first instability measure requires three income observations in a period, while estimating a rate of income drops requires a minimum of two<sup>3</sup>. Thus, 2386 are missing the Instability 1 measure for adolescence (Table 5.5) compared to 1111 for the Instability 2 measure (Table 5.6). Higher missingness for income drops versus poverty entries is due in part to mother interviews having the poverty indicator but not all of the income components required to calculate

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<sup>2</sup>A P% change in  $Y$  results in a  $\ln(1 + \frac{P}{100})$  change in  $\ln(Y)$ .  $\ln(.75) = -0.287682072$ .

<sup>3</sup>Income drops and poverty entries *could* be counted if there was only one observation in the period, against an observation before the period began. Doing so, however, could result in a rate of 1. To avoid having a subject with only one observation in a period getting a rate that appears to make that period twice as volatile as another subject with a rate of  $1/2$ , a minimum of two observations within the period were required.

total family income.

Table 5.3: Descriptive statistics for subject characteristics

	Mean	SD	Min	1st Qu	Med	3rd Qu	Max	NA
Adult BMI	27.33	6.38	12.40	22.86	25.82	30.41	64.55	0
log of BMI	3.28	0.22	2.52	3.13	3.25	3.41	4.17	0
Age BMI observed	24.27	3.28	19.00	21.00	24.00	27.00	30.00	0
Year BMI obs.	2009.63	1.19	2000	2010	2010	2010	2010	0
Female	0.49	0.50	0.00	0.00	0.00	1.00	1.00	0
Black	0.32	0.47	0.00	0.00	0.00	1.00	1.00	0
Hispanic	0.23	0.42	0.00	0.00	0.00	0.00	1.00	0
Asian/PI	0.00	0.05	0.00	0.00	0.00	0.00	1.00	0
Birth year	1985.35	3.29	1980	1983	1985	1988	1991	0

*Note:* SD is standard deviation, Min is minimum value observed, Qu is quartile, Med is median, Max is maximum value observed, and NA indicates number of observations missing data.

Of the 5046 possible sample members, 3787 are members of sibling groups of size ranging from two to seven. To account for the correlation of errors among family members, I estimate all models with heteroskedasticity-consistent standard errors, clustering within families to account for both a family-specific correlated error component and the usual idiosyncratic error.

The relationship between background material resources and future BMI may be due to factors predictive of both income and BMI. While some of these factors are entered as controls, such as familial education history, unobserved factors may create biased ordinary least squares (OLS) estimates of the parameters of interest. These might include other parent characteristics such as mental illness, physical maladies, or coping skills that are

Table 5.4: Descriptive statistics for maternal background characteristics

	Mean	SD	Min	1st Qu	Med	3rd Qu	Max	NA
(i) Any relative completed HS	0.77	0.42	0.00	1.00	1.00	1.00	1.00	0
(i) Any relative some post-HS	0.40	0.49	0.00	0.00	0.00	1.00	1.00	0
Mom foreign born	0.08	0.27	0.00	0.00	0.00	0.00	1.00	0
(i) Foreign language at home in childhood	0.26	0.44	0.00	0.00	0.00	1.00	1.00	0
(i) Mom's AFQT %ile	35.48	26.75	-19.40	12.63	29.80	53.73	100.00	0
(i) Mom resided in South at 14	0.38	0.48	0.00	0.00	0.00	1.00	1.00	0
(i) Mom lived in rural area at 14	0.20	0.40	0.00	0.00	0.00	0.00	1.00	0
(i) Mom raised in other religion	0.11	0.31	0.00	0.00	0.00	0.00	1.00	0
(i) Mom Jewish	0.00	0.06	0.00	0.00	0.00	0.00	1.00	0
(i) Mom Catholic	0.36	0.48	0.00	0.00	0.00	1.00	1.00	0
(i) Mom Protestant	0.50	0.50	0.00	0.00	0.00	1.00	1.00	0
(i) Mom raised with no religion	0.03	0.18	0.00	0.00	0.00	0.00	1.00	0

*Note:* SD is standard deviation, Min is minimum value observed, Qu is quartile, Med is median, Max is maximum value observed, and NA indicates number of observations missing data. (i) refers to average of 5 imputed values for each case. %ile is percentile.

predictive of both income instability (via job loss, disability, etc.) and child BMI (via modeling of diet and exercise or inherited traits). To test whether the OLS estimates

that follow are robust to this potential endogeneity, I conduct analyses controlling for such family-level effects using a sibling or family fixed effect (FE), essentially including a separate indicator for each mother to account for time-invariant unexplained heterogeneity. The analysis for all models compare the estimates from the more efficient but biased (clustered) OLS and the FE estimation.

The FE model parameters are estimated among sibling groups, creating a smaller sample size due to the exclusion of singletons. In addition, each model is identified to the extent that characteristics differ among siblings, resulting in further loss of efficiency due to the underlying differencing of observations from the family mean for each variable and accompanying loss of degrees of freedom (i.e. one per family). Since non-twins are born some time apart, the pre-birth and developmental periods may differ between siblings, and thus the economic circumstances for the relevant period may differ. Siblings that are identical on these variables do not contribute to the estimation of the effects of the instability measures on adult BMI.

Table 5.5: Descriptive statistics for income and Instability 1 measures

	Mean	SD	Min	1st Qu	Median	3rd Qu	Max	NA
childhood mean log family income	9.69	1.05	0.00	9.14	9.87	10.37	12.35	0
1 - $R^2$ , pre-birth	63.35	30.14	0.00	38.66	68.70	91.90	100.00	743
beta, pre-birth	-0.01	0.39	-5.31	-0.08	0.01	0.11	4.36	743
1 - $R^2$ , childhood	71.25	26.67	0.02	53.26	79.22	94.58	100.00	54
beta, childhood	0.01	0.16	-1.41	-0.02	0.02	0.05	4.99	49
1 - $R^2$ , 0 to 5	60.63	30.95	0.00	34.04	64.79	90.24	100.00	377
beta, 0 to 5	0.03	0.35	-5.01	-0.05	0.02	0.11	4.99	372
1 - $R^2$ , 6 to 11	53.03	33.38	0.00	22.56	53.73	85.87	100.00	1371
beta, 6 to 11	0.01	0.36	-5.21	-0.06	0.02	0.09	3.72	1363
1 - $R^2$ , 12 to 17	42.43	34.09	0.00	11.49	33.22	73.53	100.00	2386
beta, 12 to 17	0.03	0.42	-2.59	-0.04	0.02	0.09	3.04	2377

*Note:* SD is standard deviation, Min is minimum value observed, Qu is quartile, Max is maximum value observed, and NA indicates number of observations missing data.

Table 5.6: Descriptive statistics for Instability 2 measures

	Mean	SD	Min	1st Qu	Median	3rd Qu	Max	NA
Any 25% income drops, pre-birth	0.79	0.41	0.00	1.00	1.00	1.00	1.00	739
Proportion of pre-birth with 25% drops	0.25	0.19	0.00	0.11	0.25	0.38	1.00	739
Any 25% income drops, childhood	0.88	0.33	0.00	1.00	1.00	1.00	1.00	6
Proportion of childhood with 25% drops	0.20	0.14	0.00	0.10	0.20	0.29	1.00	6
Any 25% income drops, 0 to 5	0.68	0.47	0.00	0.00	1.00	1.00	1.00	137
Proportion of 0 to 5 with 25% drops	0.21	0.19	0.00	0.00	0.20	0.33	1.00	137
Any 25% income drops, 6 to 11	0.52	0.50	0.00	0.00	1.00	1.00	1.00	587
Proportion of 6 to 11 with 25% drops	0.19	0.21	0.00	0.00	0.17	0.33	1.00	587
Any 25% income drops, 12 to 17	0.45	0.50	0.00	0.00	0.00	1.00	1.00	1111
Proportion of 12 to 17 with 25% drops	0.20	0.24	0.00	0.00	0.00	0.33	1.00	1111

*Note:* SD is standard deviation, Min is minimum value observed, Qu is quartile, Max is maximum value observed, and NA indicates number of observations missing data.

Table 5.7: Descriptive statistics Instability 3 measures

	Mean	SD	Min	1st Qu	Median	3rd Qu	Max	NA
Any poverty entries, pre-birth	0.40	0.49	0.00	0.00	0.00	1.00	1.00	0
Probability of poverty entry, pre-birth	0.20	0.32	0.00	0.00	0.00	0.33	1.00	0
Any poverty entries, childhood	0.49	0.50	0.00	0.00	0.00	1.00	1.00	0
Rate of childhood poverty entries	0.20	0.31	0.00	0.00	0.00	0.25	1.00	0
Any poverty entries, 0 to 5	0.33	0.47	0.00	0.00	0.00	1.00	1.00	114
Probability of poverty entry, 0 to 5	0.19	0.33	0.00	0.00	0.00	0.25	1.00	114
Any poverty entries, 6 to 11	0.23	0.42	0.00	0.00	0.00	0.00	1.00	586
Probability of poverty entry, 6 to 11	0.15	0.31	0.00	0.00	0.00	0.00	1.00	586
Any poverty entries 12 to 17	0.17	0.37	0.00	0.00	0.00	0.00	1.00	1111
Probability of poverty entry, 12 to 17	0.12	0.29	0.00	0.00	0.00	0.00	1.00	1111

*Note:* SD is standard deviation, Min is minimum value observed, Qu is quartile, Max is maximum value observed, and NA indicates number of observations missing data.

### 5.3 OLS results

#### 5.3.1 Any instability, regardless of intensity

The simplest model, the dichotomous model, is similar to an inquiry that tests whether ever falling into poverty is associated with some outcome. Here, I separate out the pre-birth period from childhood to test whether childhood experiences have an effect net of the mother's prior experience, or whether the mother's own experience of instability might be what drives the underlying mechanisms connecting economic experiences to adiposity. The timing model breaks childhood up into three developmental periods, still with a predictor capturing the mother's pre-birth experiences. Again, in these models addressing the effect of *any* instability, we are not testing the continuous instability measures in Table 5.2 but only the dichotomous indicators of the presence of any instability—ever experienced a 25% income decline from the prior available year or ever became poor.

In Table 5.8 I present the results for OLS estimation of the simplest model, for each of the two instability indicators in the pre-birth and all childhood periods. In the top panel of the table, I also include the effect of mean (logged) income to show the influence of permanent childhood income net of the instability effects. Table 5.8 shows that, in this sample, there was no significant effect of falling into poverty or of experiencing an income decline in either period, net of experiences in the other period and permanent income (and other covariates). At least one entry into poverty during childhood approaches significance (at the  $\alpha = .05$  level), implying an adult BMI about 1.2% higher, *ceteris paribus*. Childhood income was not significantly related to adult BMI net of ever being in poverty and ever experiencing an income drop (regardless of the definition of income drop used; not shown).

In the bottom panel of the table (and others to follow), I present model information for each definition. The Bayesian Information Criterion fit measures are less comparable here due to the varying sample sizes. The F and BIC change for the instability measures represent two ways of judging the additional predictive power of the instability terms when added to a base model including permanent income, mother's educational background, etc.

Table 5.8: Effects of any instability, pre-birth versus childhood, on adult (log) BMI

Instability definition:	25% income drops	Poverty entries
Any in pre-birth period	0.006 (0.008)	0.009 (0.007)
Any in childhood	-0.006 (0.010)	0.012 (0.007)
Mean (log) income	-0.003 (0.004)	0.000 (0.004)
$R^2$	0.099	0.115
BIC	-257.067	-422.680
F for instability terms	371.144*	3.254*
BIC change for instability terms	89.673	10.517
N	4306	5046

*Note:* \*  $p < .05$ . Cluster-robust, multiple-imputation-corrected standard errors in parentheses. All models control for age, year, demographics, mother's background characteristics, etc. See text for full covariate list.

Here we see disagreement between the F-test approach and the BIC approach, the former judging the dichotomous instability measures in the two periods to significantly improve prediction of adult BMI, the latter judging any improved fit to not be worth the additional degrees of freedom (the BIC change is positive).

In Table 5.9, the childhood period is broken out into our three developmental periods. The effect of having an income drop during middle childhood approaches significance, but the effect is in the opposite direction as hypothesized, indicating a protective effect in terms of lower adult BMI among those who experience at least one 25% income drop during the years they turn 6 to 11 years of age. (This effect reached significance if an income decline is defined as a 50% drop in income.) Having three indicators instead of the single all-childhood predictor appears to slightly improve model fit, albeit in a slightly smaller sample, but as a group the four indicators of ever entering poverty do not appear to add predictive power beyond permanent income and other covariates.

Table 5.9: Effects of any instability, pre-birth and developmental period, on adult (log) BMI

Instability definition:	25% income drops	Poverty entries
Any in pre-birth period	0.009 (0.01)	0.012 (0.008)
Any in 0–5	0.012 (0.008)	-0.001 (0.008)
Any in 6–11	-0.015 (0.008)	0.006 (0.009)
Any in 12–17	-0.006 (0.008)	0.010 (0.010)
Mean (log) income	-0.006 (0.005)	-0.002 (0.005)
$R^2$	0.111	0.124
BIC	-162.771	-272.672
F for instability terms	143.314*	1.072
BIC change for instability terms	73.783	28.454
N	3096	3614

*Note:* \*  $p < .05$ . Cluster-robust, multiple-imputation-corrected standard errors in parentheses. All models control for age, year, demographics, mother’s background characteristics, etc. See text for full covariate list.

This estimation includes subjects with two pre-birth observations up to 10 pre-birth observations, but the latter would obviously have more of a chance to be observed having an income drop or falling into poverty. These different risk sets raise the concern that those with fewer pre-birth income or poverty measures are systematically different than those with more. For example, delayed childbirth is likely related to not being poor as of the year of the subject’s birth, but also creates more possible chances to be observed in poverty previously. Assuming their mothers responded to the survey every year, a child born in 1981 has two possible pre-birth observations, while a child born in 1989 has 10 possible. On the other hand, if there is something systematically related to economic experiences and an eventual child’s adiposity which also influences nonresponse, then there may be important differences between two subjects born in 1989 with two (or three) and 10 (or nine) pre-birth observations that either attenuate or inflate the estimated effect of pre-birth experiences.

In Tables 5.10 and 5.11, I restrict the sample to only those with at least five pre-birth observations (still over 10 possible years) and define the presence of either an income drop or a poverty entry only during the first five pre-birth years available. By accounting for potential omitted variable bias due to the mother's age at birth or other factors related to the number of pre-birth observations with this restricted sample and definition, we see that some of the estimated effects change substantially. The effect of any 25% income drop during childhood, for example, decreases by almost 86%, while the point estimate for any poverty entry during early childhood increases by a factor of 6. No indicator of instability significantly predicts later BMI net of the others, mean childhood income, and other covariates, although early childhood income instability approaches significance (and achieves it using the larger 50% income decline threshold; the negative effect of any income drop in adolescence becomes significant using the smallest threshold of a 10% decline). The BIC indicates adding pre-birth and childhood income drops (by all definitions tested) to the base model with income and other covariates creates improved fit in this smaller sample with (presumably) more homogeneous maternal age at birth.

### 5.3.2 *Intensity of instability*

The effects of frequently falling into poverty or multiple substantial year-over-year income declines may be quite different than experiencing a single such event. With the accumulation and hybrid models in Tables 5.12 and 5.13 we are examining the effects of the intensity of instability by each operational definition rather than simply whether some form of change occurred at least once in the time period specified. These models are able to use the Instability 1 measure ( $1 - R^2$  for income regressed on time, as a percent) along with the rate of income declines and poverty entry probability. In the simpler of the two (the accumulation models in Table 5.12), more pre-birth income instability, as measured by the Instability 1 measure, is associated with higher adult BMI, net of instability and income during childhood as well as the other covariates. The effect is precisely estimated but small (0.000239), implying a 10 percentage point increase in pre-birth income volatility as measured by the  $R^2$ —having the time trend explain 10 percentage points less of the variation in the mother's

Table 5.10: Effects of any instability on adult (log) BMI, with restricted pre-birth period

Instability definition:	25% income drops	Poverty entries
Any in pre-birth period	0.009 (0.009)	0.009 (0.008)
Any in childhood	-0.001 (0.011)	0.009 (0.008)
mean (log) income	-0.003 (0.005)	0.000 (0.004)
$R^2$	0.101	0.096
BIC	-91.969	-167.966
F for instability terms	541.083*	1.734
BIC change for instability terms	-1.309	12.845
N	2554	3523

*Note:* \*  $p < .05$ . Pre-birth period here refers to 5 observations prior to subject's birth, only among those with at least 5 pre-birth surveys. Cluster-robust, multiple-imputation-corrected standard errors in parentheses. All models control for age, year, demographics, mother's background characteristics, etc. See text for full covariate list.

family income—is associated with an adult BMI just over 0.2% higher. In contrast, Smith and colleagues (2009) found the same change in income instability over the prior 12 years among adult males was associated with an additional 2.4 pounds in weight. For a baseline of 5 feet 9 inches and 150 pounds (BMI = 22.15), the result of Smith et al. implies a new BMI of 22.50 or 1.6% higher. If the Smith et al. baseline male was 250 pounds, this 10 percentage point decrease in income predictability implies a change of less than 1% (37.27 versus 36.91), but still notably higher than the current 0.2%. Maximizing the possible range of this predictor produces the following hypothetical: Comparing someone whose pre-birth income is completely random and not at all explained by the time trend (Instability 1 = 100) to someone whose time trend perfectly accounts for variation in income during this period (Instability 1 = 0) results in an adult BMI almost 2.4% higher.

Table 5.11: Effects of any instability on adult (log) BMI, with restricted pre-birth period

Instability definition:	25% income drops	Poverty entries
Any in pre-birth period	0.014 (0.010)	0.014 (0.010)
Any in 0–5	0.017 (0.009)	-0.008 (0.010)
Any in 6–11	-0.002 (0.009)	0.008 (0.011)
Any in 12–17	-0.005 (0.010)	0.012 (0.013)
Mean (log) income	-0.004 (0.006)	-0.003 (0.005)
$R^2$	0.119	0.108
BIC	-52.004	-95.043
F for instability terms	196.149*	1.129
BIC change for instability terms	2.428	26.812
N	1893	2548

*Note:* \*  $p < .05$ . Pre-birth period here refers to 5 observations prior to subject’s birth, only among those with at least 5 pre-birth surveys. Cluster-robust, multiple-imputation-corrected standard errors in parentheses. All models control for age, year, demographics, mother’s background characteristics, etc. See text for full covariate list.

In the accumulation model, the rates of neither 25% income drops nor of falling below the poverty threshold if out of poverty the prior year significantly predicts future BMI. The rate of large (50%) income drops during childhood approached significance and implies a protective effect of downward income instability. The null effect of the probability of poverty entry and of the rate of income drops (by all definitions tested) remains in the more flexible hybrid model in Table 5.13, breaking childhood into three periods. Collinearity may have contributed to less precise estimates of the effect of poverty entry probability, which correlates across periods at 0.12 to 0.21. Collinearity is likely not an issue for the rate of income drops, as no inter-period correlation exceeds 0.092.

Table 5.12: Effects of instability accumulation, pre-birth versus childhood, on adult (log) BMI

Instability definition:	$1 - R^2$	25% income drops	Probability of poverty entry
Pre-birth period	0.0002 (0.0001)*	0.007 (0.019)	0.014 (0.011)
Childhood	-0.0001 (0.0001)	-0.041 (0.027)	0.019 (0.012)
Mean (log) income	-0.006 (0.004)	-0.005 (0.004)	0.001 (0.004)
$R^2$	0.102	0.100	0.115
BIC	-250.833	-259.151	-422.051
F for instability terms	199.578*	372.117*	2.698
BIC change for instability terms	91.081	88.006	11.635
N	4261	4306	5046

*Note:* \*  $p < .05$ . Cluster-robust, multiple-imputation-corrected standard errors in parentheses. All models control for age, year, demographics, mother's background characteristics, etc. See text for full covariate list.

The non-significant effect of childhood income instability around the time trend of income (Instability 1) in Table 5.12 may have been due to differential effects of income instability depending upon when it occurs: More instability during early childhood appears to be positively associated with later BMI while instability during middle childhood appears to be negatively associated. The effects are again precisely estimated (inter-period correlations of the Instability 1 measure are all  $< 0.04$ ) but small (0.000381 and -0.000315). The higher  $R^2$  model fit result for the Instability 1 measure is also due to the presence of the four terms capturing the income trend in the four periods (all of which were non-significantly positive in effect, illustrated in Figure 5.2 in the next section).

Table 5.13: Effects of instability accumulation, pre-birth and developmental period, on adult (log) BMI

Instability definition:	$1 - R^2$	25% income drops	Probability of poverty entry
Pre-birth period	-0.0001 (0.0002)	0.019 (0.022)	0.020 (0.014)
0–5	0.0004 (0.0002)*	0.018 (0.022)	0.004 (0.013)
6–11	-0.0003 (0.0001)*	-0.022 (0.019)	0.000 (0.013)
12–17	-0.0001 (0.0001)	-0.009 (0.017)	0.015 (0.014)
Mean (log) income	-0.004 (0.007)	-0.005 (0.005)	-0.002 (0.005)
$R^2$	0.136	0.110	0.124
BIC	-44.353	-158.235	-273.187
F for instability terms	241.664*	141.980*	1.200
BIC change for instability terms	32.121	78.319	27.938
N	1785	3096	3614

*Note:* \*  $p < .05$ . Cluster-robust, multiple-imputation-corrected standard errors in parentheses. All models control for age, year, demographics, mother’s background characteristics, etc. See text for full covariate list.

### 5.3.3 Summary of OLS results

This analysis tests the effect of childhood income instability measured in different ways and at different times on adult BMI. To get a sense of the pattern of results over the multiple definitions, including for potential alternate definitions of income drops, in Table 5.14 I illustrate the signs and strengths of the estimated effects. The direction of effects for models with pre-birth and childhood measures appear in the top panel of Table 5.14, while the models that break out childhood into developmental periods appear in the bottom panel.

Table 5.14: Summary of effects of income instability on adult BMI

	Instability 1	10% drops		25% drops		50% drops		Poverty entries	
		Any	Rate	Any	Rate	Any	Rate	Any	Rate
Pre-birth	▲	△	△	△	△	△	▽	△	△
Childhood	▽	▽	▽	▽	▽	▽	▼	▲◦	△
Pre-birth	▽	△	△	△	△	△	▽	△	△
0-5	▲	△	△	△†	△	△‡	△	▽	△
6-11	▼	▽	▽	▼◦	▽	▼◦	▽	△	△
12-17	▽	▽‡	▽	▽	▽	▽	▽	△	△

*Note:* ▲ indicates significant positive and ▼ significant negative relationship with adult BMI, both  $p < .05$ . ▲ indicates marginally significantly positive and ▼ marginally significant negative relationship,  $p < .10$ . △ and ▽ indicate non-significant positive and negative relationship. ◦ indicates formerly significant relationship becomes non-significant ( $p \geq .10$ ) with restricted sample and pre-birth definition as in Tables 5.10 and 5.11; † and ‡ indicate relationship becomes significant at  $p < .10$  and  $p < .05$ , respectively. All models control for mean childhood income, age, year, demographics, mother's background characteristics, etc. See text for full covariate list. Models with Instability 1 measure also control for within-period time trends of family income.

Sensitivity testing with a restricted sample of those with at least five pre-birth income observations, designed to counteract possible omitted variable bias from factors related to the number of pre-birth observations available, never changed the significance of pre-birth indicators (any income drop or any poverty entry) but did affect the significance of indicators during childhood. Poverty entry is at least marginally significant only once, but this possible effect of ever entering poverty disappears in the restricted sample. The probability of entering poverty is always positively related to adult BMI, implying past risk of poverty may affect diet and exercise, but this effect is never even marginally significant net

of mean income and other factors. As for purely downward instability, our main definition for income drops was marginally significant once in the main sample (any 25% drops in middle childhood). Sensitivity testing with smaller and larger income declines found that the resulting effect estimates were almost always in the same direction (except for the pre-birth rate of 50% income drops) and sometimes larger. The restricted sample sensitivity testing found that protective relationships for the presence of a 25% or 50% income drop in middle childhood disappeared, but the positive effect on later BMI of such a decline in early childhood became at least moderately significant. The frequency of income declines was marginally significant only once out of 18 chances, and in the opposite direction as hypothesized, implying the presence of any significant year-over-year income decline is a stronger factor in the diet and exercise patterns that result in BMI than the accumulation of such declines. The strongest measure appears to be the Instability 1 indicator, based on the within-individual and -period regression of family income on time, which is significant in three out of six estimates.

Instability during the pre-birth period is almost always positively if weakly associated with higher adult BMI. There are three notable exceptions to this. First, pre-birth instability as measured by the rate of larger (50%) income drops is negatively but non-significantly related to adult BMI across both the accumulation and hybrid models. The  $R^2$ -based Instability 1 measure becomes negative but non-significant in the more complex hybrid model, which includes fewer sample members due to higher data requirements from dividing childhood into three periods. This might imply that some of the apparent effect of pre-birth instability is due to correlation between instability in this period and instability in the earliest years of the child's life. The correlations among the Instability 1 measures are quite low (on the order of 0.02 or less), and (as seen in Figure 5.2 below) the positive effect of pre-birth income instability appears to be driven by subjects who do not have at least three income observations within each developmental period and thus do not contribute to estimation of the four-period hybrid model.

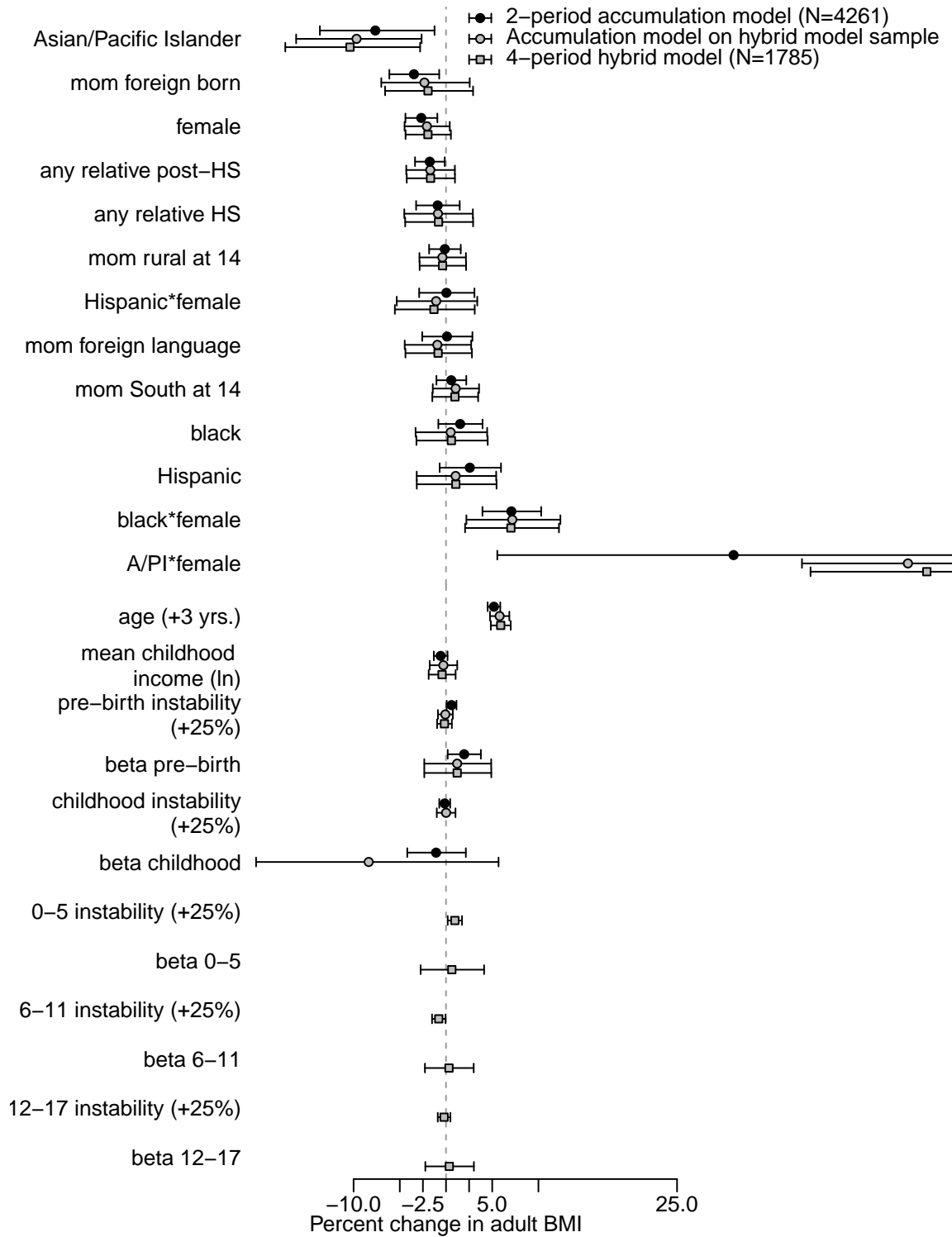
The benefit of the latter model, of course, is that it offers evidence that instability during early childhood and middle childhood have opposing effects. As the bottom panel of Table 5.14 shows, this pattern of opposing effects of instability in early childhood versus later childhood is fairly stable if weakly estimated. Instability in early childhood is almost always positively related to adult BMI, except for any poverty entry, while instability in middle childhood and adolescence is almost always related to a lower adult BMI, except for poverty entries. This apparent importance of developmental period may be part of why the two-period models found only two effects across all of childhood to be even marginally significant. Any poverty entry shows the opposite pattern, with a non-significant negative effect in early childhood and positive effects in the later two periods. Again, the probability of poverty entry exhibits a different relationship with adult BMI than the other continuous measures. This “rate” captures a different phenomenon than income drops or the  $R^2$ -based measure, in that the measure could be maximized via a single decline in income (e.g., starting out above poverty and immediately becoming and remaining poor) or by dropping back below poverty level immediately after each year out of poverty.

If the Instability 1 measure is the strongest of the three proxies for developmental income instability, further investigation of the size of the effect of instability reveals the weakness of any relationship with adult body mass. In the top half of Figure 5.2, I present the estimated percent difference in adult BMI for those with versus those without dichotomous traits controlled for in the analysis (i.e. coefficients presented as percent change in adult BMI). These effect sizes, with 95% confidence intervals, are presented for the two-period accumulation model and the four-period hybrid model as above. In addition, as a sensitivity check, I present results for the two-period model estimated on the smaller four-period sample (i.e. those individuals with three or more income observations in each of the four developmental periods). The dichotomous covariate effects are presented in order of the point estimate for the full accumulation model estimation. In the bottom half of the figure, I present similar estimates for continuous covariates age and mean (logged) childhood income and then for the instability and accompanying within-period time trend of income (the  $\hat{\beta}$  from (5.1)) for each period examined. To ease comparison, the effect associated with age is presented

as the average percent change in adult BMI associated with an additional three years in age, while the effects associated with the instability measure are estimated as the percent change associated with an additional 25% of the variation in income not explained by the time trend (i.e. a 25 point change instead of a 1 point change). As seen in Table 5.5, this represents just below one standard deviation of difference for each period's Instability 1 measure among all those with a value for that period, and (as seen in Table 5.3) just below a one standard deviation increase in age.

Figure 5.2 illustrates that some results across the three estimations are fairly consistent. Asians and Pacific Islanders as a group have a lower BMI than whites, although the relatively few Asian/Pacific Islander females in the sample have a significantly higher BMI, while black females have a higher BMI than black males or white females (i.e. the black female interaction effect is significantly greater than 0). An additional three years of age is consistently associated with a BMI 5.2 to 5.9% higher. Some differences are seen between the larger sample with complete information on pre-birth and childhood instability—the 4261 involved in the full accumulation model estimation—and the smaller sample on which the four-period hybrid model is estimated: The elasticity of adult BMI with respect to mean childhood income is significantly negative in the full accumulation model but moves closer to 0 (and, of course, has a broader confidence interval) in the smaller sample. As noted above, the point estimate for pre-birth instability also changes signs in the smaller sample. The confidence intervals for this and the other three instability effects indicate that all are precisely estimated across all three estimations presented, and all are quite small. Even when expanded to illustrate the effect of a movement of one-quarter of the possible range of income instability, and even when statistically significant, the size of the effect of more income instability, net of average income and the other covariates, pales in comparison to three more years of age or being a black female versus a white female or black male.

Figure 5.2: Discrete effects of income instability and covariates on adult BMI (with confidence intervals)



#### 5.4 *Fixed effects model results*

With the fixed effects models, we are estimating the effects of predictors of interest over and above common factors that may include important genetic, cultural, and psychological influences on weight within the family. If the estimated coefficients remain consistent, this indicates the influence of income volatility may be independent of these family factors. If the estimates change substantially, this indicates likely correlation between the family factors and the instability. As noted, fixed effects estimation of the instability parameters requires within-family variation on income instability or poverty entries, which is more likely with rates than with dichotomies and more likely with smaller time periods which may overlap little among siblings. Thus, as seen in Tables 5.15 and 5.16, the precision of our estimates is lower than in the OLS estimates in part because, for example, only 140 families have at least one sibling who differs from the other(s) on the presence of a 25% income drop during childhood, and only 180 families differ on the mother entering poverty some time during the child's adolescence. This loss of power is somewhat counteracted by the fact that the models are inherently more parsimonious due to the dropping of any covariate—now absorbed by the family fixed effect—constant among sibling groups, including family educational background, mother's immigrant status, etc. While none of the instability indicators in the basic two-period model are significant, note that, despite the lower precision the estimated effect of permanent income net of the presence of at least one income decline (by each definition) is significant, and the income effect net of poverty entry approached significance. Both are in the opposite direction of the conventional wisdom, and imply a higher BMI with higher childhood income controlling for the presence of an income decline or a poverty entry in both the pre-birth and childhood periods.

Any income drop during adolescence, net of permanent income and these time-invariant family factors, appears to be negatively related to later BMI, and an income drop during middle childhood is marginally significantly related to a lower adult BMI as well. The direction of these effects is consistent with the OLS models, and the estimated sizes of the effects grow substantially when accounting for unobserved family level heterogeneity, with

Table 5.15: Fixed effects estimates of influence of any instability, pre-birth versus childhood, on adult (log) BMI

Instability definition:	25% income drops	Poverty entries
Any in pre-birth period	0.014 (0.016)	-0.013 (0.015)
Any in childhood	0.011 (0.018)	-0.006 (0.016)
Mean (log) income	0.021 (0.010)*	0.016 (0.010)
$R^2$	0.109	0.110
BIC	-278.475	-366.544
F for instability terms	502.157*	2.418
BIC change for instability terms	16.501	11.634
N	2980	3787
Families	1276	1580
Differ on pre-birth	251	355
Differ on childhood	140	255

*Note:* \*  $p < .05$ . Cluster-robust, multiple-imputation-corrected standard errors in parentheses. All models control for age, year, gender, and race/ethnicity.

the presence of an income decline in adolescence becoming a significant predictor of adult BMI. The size and significance of these effects are not, however, consistent across the three definitions of an income drop. The significant positive effect of the presence of an income drop in early childhood hinted at by the restricted sample sensitivity testing in Tables 5.11 and 5.14 is not apparent when controlling for unobserved family-level heterogeneity, which would include the mother's age at first birth (note that any mother consistently reporting zero family income would likely not have her children included in the FE models due to lack of variation in the relevant factors). Only the estimate of the effect of a 25% income decline in early childhood (and, not shown, of a 50% income drop in middle childhood) is consistent in size between the OLS and FE models, changing less than 5%. The effect of permanent income noted in the results in Table 5.15 is reduced to zero when accounting for

Table 5.16: Fixed effects estimates of influence of any instability, pre-birth and developmental period, on adult (log) BMI

Instability definition:	25% income drops	Poverty entries
Any in pre-birth period	0.006 (0.019)	-0.003 (0.020)
Any in 0–5	0.012 (0.014)	0.001 (0.018)
Any in 6–11	-0.032 (0.017)	-0.001 (0.020)
Any in 12–17	-0.031 (0.015)*	-0.023 (0.022)
Mean (log) income	0.000 (0.019)	0.000 (0.019)
$R^2$	0.128	0.115
BIC	-200.636	-230.129
F for instability	166.875*	0.837
BIC change for instability terms	-2.236	27.977
N	2019	2524
Families	878	1077
Differ on pre-birth	184	223
Differ 0–5	288	285
Differ 6–11	306	230
Differ 12–17	269	180

*Note:* \*  $p < .05$ . Cluster-robust, multiple-imputation-corrected standard errors in parentheses. All models control for age, year, gender, and race/ethnicity.

income declines or poverty entries in the three developmental periods.

As seen in Tables 5.17 and 5.18, substantially more families differ on the instability intensity measures across the different time periods. In the simpler of the two specifications, combining all of childhood, no instability measure is even moderately related to adult BMI net of permanent income, the other covariates, and the fixed family factors accounted for by the sibling fixed effects. Similar to Tables 5.15 and 5.16, the significant—and, again,

positive—effect of childhood permanent income disappears (although net of the Instability 1 measures the estimated effect actually doubles in size) when we move from the two-period to the four-period specification. The previous significant positive effect of pre-birth volatility as measured by the Instability 1 measure is approximately one-quarter smaller in size (.000177 versus .000239) and non-significant, even at  $\alpha = .10$ , in the FE analysis. In the more flexible hybrid model, the signs of the Instability 1 parameters during early and middle childhood are again in the opposite direction, but are not significant (even at the .10 level). The latter point estimate declines by only around 6% (from .000315 to .000296), indicating the effect is fairly consistent between the OLS and FE models, but its significance is diminished by the lower power of the latter.

Table 5.17: Fixed effects estimates of influence of instability accumulation, pre-birth versus childhood, on adult (log) BMI

Instability definition:	$1 - R^2$	25% income drops	Probability of poverty entry
Pre-birth period	0.0002 (0.0002)	0.035 (0.037)	0.003 (0.022)
Childhood	0.0002 (0.0002)	0.100 (0.066)	0.002 (0.025)
Mean (log) income	0.025 (0.012)*	0.026 (0.011)*	0.015 (0.009)
$R^2$	0.108	0.109	0.109
BIC	-258.604	-280.111	-365.017
F for instability terms	253.060*	501.527*	0.020
BIC change for instability terms	35.419	17.445	16.439
N	2949	2980	3787
Families	1265	1276	1580
Differ on pre-birth	1265	1129	730
Differ on childhood	1265	1167	844

*Note:* \*  $p < .05$ . Cluster-robust, multiple-imputation-corrected standard errors in parentheses. All models control for age, year, gender, and race/ethnicity.

Table 5.18: Fixed effects estimates of influence of instability accumulation, pre-birth and developmental period, on adult (log) BMI

Instability definition:	$1 - R^2$	25% income drops	Probability of poverty entry
Pre-birth period	0.0000 (0.0003)	0.039 (0.051)	-0.008 (0.030)
0–5	0.0002 (0.0002)	0.063 (0.055)	-0.008 (0.024)
6–11	-0.0003 (0.0002)	0.030 (0.046)	-0.010 (0.027)
12–17	0.0001 (0.0002)	-0.011 (0.034)	-0.028 (0.027)
Mean (log) income	0.050 (0.035)	0.012 (0.019)	0.000 (0.019)
$R^2$	0.134	0.122	0.115
BIC	-56.302	-186.976	-229.372
F for instability terms	214.492*	162.363*	0.648
BIC change for instability terms	30.960	11.424	28.734
N	1068	2019	2524
Families	467	878	1077
Differ on pre-birth	467	781	482
Differ 0–5	467	707	467
Differ 6–11	467	570	467
Differ 12–17	467	510	467

*Note:* \*  $p < .05$ . Cluster-robust, multiple-imputation-corrected standard errors in parentheses. All models control for age, year, gender, and race/ethnicity.

To summarize the estimated effects across operational definitions and models, in the top panel of Table 5.19 we see that none of the instability parameters, including for the smaller and larger income decline definitions tested for sensitivity, are significant at the .10 level in the simpler two-period models. In fact, all four effects significant at the .10 level (out of 54 chances) are for income declines in middle childhood and adolescence.

(While each significance test is not an independent event—a handful of instability measure intercorrelations are above 0.2—this certainly does not appear to be better than chance.) Accounting for fixed family-level factors related to both income and adiposity, only the presence or rate of income declines, particularly during adolescence, are associated with later BMI, and whichever way the instability is measured, the effect is negative.

Table 5.19: Summary of fixed effects estimates of income instability predicting adult BMI

	Instability 1	10% drops		25% drops		50% drops		Poverty entries	
		Any	Rate	Any	Rate	Any	Rate	Any	Rate
Pre-birth	△	△	△	△	△	△	△	▽	△
Childhood	△	▽	△	△	△	△	▽	▽	△
Pre-birth	▽	△	△	△	△	△	▽	▽	▽
0-5	△	△	△	△	△	△	▽	△	▽
6-11	▽	▽	△	▼	△	▽	▽	▽	▽
12-17	△	▼	▽	▼	▽	▽	▼	▽	▽

*Note:* ▼ significant negative relationship with adult BMI,  $p < .05$ , and ▼ marginally significant,  $p < .10$ . △ and ▽ indicate non-significant positive and negative relationship. All models control for mean childhood income, age, year, gender, and race/ethnicity. Models with Instability 1 measure also control for within-period time trends of family income.

Comparing the FE results to the OLS results summarized in Table 5.14 (page 170), the FE results are less consistent across definitions and models than the OLS results. For example, the effect of the Instability 1 measure during adolescence is positive but not significant, while every other estimated sign for instability in adolescence is negative. In the OLS models, all but the effects of poverty entries, both the dichotomous indicator and the rate, during adolescence were negatively related to later BMI. While the probability of poverty entry always had a positive if non-significant effect in the OLS models, in the FE hybrid model the estimated effect is always negative, the reverse of the OLS model. Pre-birth instability

and instability during early childhood are less consistently associated with higher BMI. As most of the effects are not estimated to be significantly different from zero, even at the .10 level, this variation likely reflects random fluctuation of weak effects. More notably, all five of the effects estimated to be at least marginally positively related to adult BMI at least once in the OLS models, including all sensitivity analyses, become non-significant in the FE models. Of the five at least moderately significant negative effects in the OLS models, three (the rate of 50% drops in all childhood, Instability 1 in middle childhood, and any 50% drops in middle childhood) disappear in the FE models.

### **5.5 Summary and implications**

While conventional wisdom holds that poor economic conditions contribute to obesity, the evidence for this is mixed. In the usual direction, studies have found child poverty to be associated with later obesity among young women (Lee et al., 2009) and the proportion of childhood spent in poverty to be predictive of childhood obesity risk (Liu, 2007) and BMI growth from middle childhood through adolescence (Wells et al., 2010). Higher income at low levels of income during the prenatal and birth years has been negatively associated with adult risk for overweight and obesity and with mean BMI and the 25<sup>th</sup>, 50<sup>th</sup>, and 75<sup>th</sup> percentiles of BMI (Ziol-Guest et al., 2009). In the other direction, recent evidence links additional exogenous income to higher BMI among less advantaged Native American youth (Akee et al., 2010), and when Liu (2009) added additional demographic and SES controls as well as the mother's weight status, the positive effect of proportion of childhood poor became negative. An analysis of multiple measures of SES in adolescence and young adulthood utilized Latent Class Analysis to identify five different patterns of SES change. All four groups with 'low' or 'middle' teen-age and/or current situations were at higher risk of obesity than those fortunate to have enjoyed high SES in both periods. This included adults whose current SES was relatively higher than their adolescent SES (Scharoun-Lee et al., 2011). Similarly, even cross-sectional data tends to show that risk of obesity does not decline until well above poverty level (e.g., Anderson, Butcher, & Schanzenbach, 2007; Kant & Graubard, 2007; Komlos & Brabec, 2010).

People will experience and react to a given level of income in different ways, depending upon family and cultural norms, the availability of consumption-smoothing resources such as wealth or family support, community resiliency factors, and so on. Individual differences in stress perception and coping skills will further moderate the health effects of what appear to be otherwise equivalently stressful situations (Seeman et al., 2010). While much attention has been paid to the health effects—obesity and otherwise—of low income, relatively little has been given to the potentially important effects of income shocks over and above permanent income levels (Kawachi et al., 2010). Lower income families experience more income volatility (Gundersen & Ziliak, 2003). Such volatility may be a mechanism connecting low income to adverse health effects.

The initial research results supporting the potential effect of economic insecurity on adiposity have come from three very different sources. Offer and colleagues (2010) provide a cross-national perspective linking a country's level of economic insecurity to its obesity prevalence. Developmental evidence comes from the qualitative research of Olson and associates (2007), who describe the effects of occasional bounty, as when the food stamps arrive, as well as how mothers change their purchasing and food preparation customs in response to their past experiences of deprivation. The strongest evidence comes from Smith et al. (2009), who link plausibly exogenous probability of job loss, rate of recent income declines, and a measure of income volatility over the prior 12 years to adult male weight gain.

There is much evidence that a substantial portion of recent increases in adiposity have come among women (Koch & Wilson, 2013), with women essentially passing their formerly heavier male counterparts since the mid 1990s (Cook & Daponte, 2008), and that SES-related disparities in obesity are larger among women (Chang, Hillier, & Mehta, 2009; Lee, 2011). This analysis combined the work of Olson et al. (2007) and Smith and colleagues (2009), simplifying and extending the latter to a developmental context to examine the effects of income instability during childhood and that experienced by the mother prior to the birth of now-adult men and women. The aim was to tease out the effects of resource volatility, which may have stress-related physiological and psychological effects or promote

lasting changes in diet and exercise habits, from the effects of permanent childhood income.

The analysis initially found inconsistent support for a positive effect on adult BMI of income instability experienced by the mother in the period (up to 10 years in length) prior to the subject's birth, and of instability experienced during the early childhood period (the birth year through the year the subject turned 5). There was as much support, however, for a negative effect on later BMI of instability, particularly instability experienced in middle childhood and adolescence, and all even modestly significant positive effects disappeared when accounting for fixed family-level factors that might be related to both income experiences and diet and exercise. The most consistent evidence appears to be for a protective effect of income instability during adolescence. For example, fixed effects modeling found that adults who experienced at least one 25% decline in normalized family income during adolescence had an average BMI just over 3% lower than adults who did not, net of permanent income, income drops in other periods, and other influences. This appears to suggest reactions to income declines might include adaptive measures that serve to assuage budgetary concerns and affect body mass, as in reducing automobile use or food consumption. Note that the latter suggests reducing consumption of favored foods rather than substituting cheaper but higher calorie items. For some, it may be that the income changes themselves represent a parent transitioning to home labor allowing for increased monitoring of the adolescent's diet and exercise.

Although the discussion has largely focused on the effects of instability of economic resources, the investigation is ultimately about separating the effects of volatility or income shocks from that of permanent income. In the OLS models, the effect of permanent childhood income, net of instability and other covariates, was always negative or 0.000, but always very small and never significant. Accounting for unobserved heterogeneity via family fixed effects identified a very different effect of permanent income. Although the coefficient was often zero in four-period models, when it was not, the estimated effect of permanent income was 10 times the size of the OLS estimate and often significant in two-period models. In all cases (except net of the rate of 50% income drops in four periods), average childhood

income was *positively* related to adult BMI net of income instability and family factors, the opposite from the conventional wisdom regarding income and adiposity.

There appears to be no consistent evidence that income instability in or immediately before childhood is associated with higher BMI in adulthood. This finding does not directly conflict with those of Offer and associates (2010), Smith et al. (2009), or even Olson and colleagues (2007). Income instability likely produces adaptations in shopping and dietary patterns, and may well induce weight gain in the short run. In taking a longer view, this analysis adapted developmental timing models of the effects of childhood economic circumstances on adult health, which hold that experiences in certain critical periods may have irreversible long-term impacts on health (Conroy et al., 2010). Given the multiply determined nature of weight, and the myriad messages and resources available regarding healthy eating and exercise, it should perhaps come as little surprise that no such triggering event is clear for body mass. More recent experiences—as in the sample of Smith and colleagues—appear to be more important than childhood economic security in driving the diet and exercise habits that ultimately produce obesity.

## Chapter 6

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