

Age, Socioeconomic Status and Obesity Growth

Charles L. Baum II
Middle Tennessee State University
cbaum@mtsu.edu
(615) 898-2527

and

Christopher J. Ruhm
University of North Carolina at Greensboro and NBER
chrisruhm@uncg.edu
(336) 334-5148

March 2007

Abstract

The rapid growth in obesity represents a major public concern. Although body weight tends to increase with age, the evolution of obesity over the lifecycle is not well understood. We use longitudinal data from the National Longitudinal Survey of Youth to examine how body weight changes with age for a cohort moving into and through early adulthood. We further investigate how the age-obesity gradient differs with socioeconomic status (SES) and begin to examine channels for these SES disparities. Our analysis obtains three main findings. First, weight rises with age but is inversely related to SES at given ages. Second, the SES-obesity gradient widens over the lifecycle, which is a result consistent with research examining other health outcomes such as overall health status or specific medical conditions. Third, a substantial portion of the SES “effect” is transmitted through race/ethnicity and the translation of advantaged family backgrounds during childhood to higher levels of subsequent education. Conversely, little of the SES difference appears to be propagated through family income, marital status, number of children, or a limited set of health behaviors we are able to account for. However, approximately half of the SES-weight correlation persists after the inclusion of controls, illustrating the need for further study of the mechanisms for the gradient.

Age, Socioeconomic Status and Obesity Growth

A positive relationship between socioeconomic status (SES) and health has been widely documented (Marmot et al., 1991; Smith, 2004). The sources of these disparities are not well understood. Medical researchers and epidemiologists tend to emphasize the causal effects of SES, whereas economists frequently focus on how health influences SES or the role of additional factors (such as discount rates or genetics) that may be correlated with both. In an effort to identify the causal impact of SES, some researchers have recently focused on SES-health disparities early in life and on the evolution of these gradients as age increases. A primary advantage of this approach is that health status is unlikely to significantly affect the SES of youths, since the latter is largely determined by the economic situation of the child's parents. Most such investigations suggest that SES-health gradients become more pronounced with age, through at least early adulthood.

The current analysis contributes to this line of study by examining how body weight and obesity evolve during the transition from late adolescence through the middle adult years. The focus on weight is useful for several reasons. First, obesity is an important risk factor for premature death (Allison et al., 1999; Fontaine et al., 2003; Flegal et al., 2005) and health problems like diabetes, gallbladder disease, coronary heart disease, high cholesterol, hypertension and asthma (Must et al., 1999; Mokdad et al., 2001; McTigue et al., 2006). Excess weight reduces the quality of life, raises medical expenditures, places stress on the health care system and results in productivity losses due to disability, illness and premature mortality (Quesenberry et al., 1998; Finkelstein et al., 2003; Andreyeva et al., 2004). A second advantage is that changes in body weight are easily observable, whereas many health indicators (such as self-reported overall health

status or specific medical conditions) are likely to be measured with greater error or require interaction with the medical system for diagnosis.¹ Third, obesity represents a rapidly increasing health risk. Using conventional definitions, 31 percent of 18-74 year olds were obese in 1999-2004, compared to just 14 percent in 1976-1980 (Ruhm, 2007). Fourth, obesity generally develops over a lengthy period of time – since body weight is a stock resulting from flows of caloric intake and expenditures – and so may reflect an accumulation of the effects of SES differences on health inputs. Consistent with this, excess weight during childhood, particularly in late adolescence, is a strong predictor of adult obesity (Whitaker et al., 1997; Guo et al., 2002; McTigue et al., 2002).

We use longitudinal data from the National Longitudinal Survey of Youth (NLSY) to show how body weight changes with age for a cohort moving into and through early adulthood, investigate how SES differences in this age-obesity gradient, and to begin to examine channels for the SES disparities. Our analysis shows that weight increases with age and is inversely related to SES at given ages. The SES-obesity gradient widens over the lifecycle, a result consistent with research examining other health outcomes such as overall health status or specific medical conditions. A substantial portion of the SES “effect” operates through differences across race/ethnicity and through the translation of advantaged family backgrounds during childhood to higher levels of subsequent education. By contrast, differences in household composition, family incomes or the limited set of health behaviors for which we control do not appear to provide important mechanisms for the SES disparities. However, approximately half

¹ Chang and Christakis (2005) give similar reasons for using body weight/obesity outcomes to examine the relationship between income inequality and health.

of the SES-weight correlation persists after the inclusion of controls, illustrating the need for further study of the mechanisms for the gradient.

1. Socioeconomic Status and Obesity

Adult body weight and obesity are inversely related to variety of measures of social and economic advantage. For instance, previous research indicates that the prevalence of obesity declines with income and education, and is more common for minorities than whites. Thus, 31 percent of non-Hispanic whites aged 20 and older were obese in 2003-2004, using clinical measures of height and weight, compared to 37 percent of Hispanics and 45 percent of non-Hispanic blacks (Ogden et al., 2006). Using self-reported data (which results in lower prevalence estimates than with clinical observations), 26 percent of high school dropouts were obese in 2000 versus 22 percent of high school completers and 15 percent of college graduates (Mokdad et al., 2001). Similarly, 23 (14) percent of white women (men) with family incomes greater than 400 percent of the poverty line were obese in 1999-2002, compared to 40 (34) percent of their counterparts living in poverty, although this distinction is not always present for nonwhites (Chang and Lauderdale, 2005).

These patterns are consistent with substantial evidence that high SES adults are healthier than their less advantaged counterparts. Causal pathways, however, are difficult to identify. SES certainly may have causal effects on body weight. For example, Drewnowski and Specter (2004) attribute some of the high obesity rates of disadvantaged groups to the relatively low cost of energy-dense foods, since poor individuals can more easily cover caloric requirements by purchasing high-calorie products. On the other hand, economists provide evidence that obese individuals receive a wage penalty (Averett

and Korenman, 1996; Baum and Ford, 2004; Cawley, 2004), suggesting that obesity reduces SES. Finally, unobserved factors could determine both SES and body weight. For instance, high discount rates may simultaneously reduce educational investments and decrease the willingness to forgo current caloric intake for the future benefit of lower weight (Fuchs, 2004; Smith et al., 2005; Borghans and Golsteyn, 2006). Self-control problems may also be more common among low SES individuals (Cutler et al., 2003).²

Some researchers have focused on SES-health gradients among youths as a promising method of identifying whether SES casually affects health. Examining youths has two advantages. First, it seems unlikely that health could significantly affect SES, since the latter is largely determined by the education and economic circumstances of the parents.³ Second, although omitted factors transmitted across generations (such as genetics) could play a role, the influence of other potential confounders (like discount rates) would not be expected to affect SES until later in life.

Of particular relevance is research by Case et al. (2002) indicating that the SES-health gradient “rotates” (steepens) as individuals move from infancy through late adolescence. In subsequent work, Case et al. (2005) show that poor health during childhood is associated with lower educational attainment, lower social status and more health problems in adulthood, suggesting that health is an important mechanism through which economic status is transmitted. Smith (2004) confirms that health-SES disparities increase through at least age 50 but suggests that a narrowing occurs later in life.

² Sophisticated approaches have been used in an attempt to identify the direction of causality. For instance, Adams et al. (2003) employ procedures similar to (but more advanced than) Granger-causality methods to test for the absence of direct causal paths from SES to innovations in health and from health status to innovations in SES using data on U.S. senior citizens.

³ Some effect could remain when proxying socioeconomic status by household income if, for example, parents work less when their children have serious health problems (Powers, 2003; Noonan et al. 2005). This is less of an issue for the education-based SES measures on which we focus.

Although we do not fully understand why the gradient rotates, Janet Currie and her coauthors suggest that it is primarily because disadvantaged individuals are subjected to more deleterious health events, rather than because they are more adversely affected by given shocks (Currie and Hyson, 2002; Currie and Stabile, 2003). Case et al. (2002) suggest, but do not provide conclusive evidence of, a key role for differences in health behaviors.

Body weight and obesity are particularly useful outcomes for understanding age-related changes in SES-health disparities but have not directly been examined in this context. The most closely related investigation, of which we are aware, is by McTigue et al. (2002). They use data from the 1981 through 1998 years of the National Longitudinal Study of Youth (NLSY) (the same data set we analyze use but for a shorter period) to examine race/ethnicity differences in the evolution of body weight. That investigation contained a number of limitations: the role of SES was not explicitly examined, the sources of observed differences were not identified, an extremely limited set of covariates was controlled for, and the effects of aging and secular trends in body weight were confounded. Our analysis attempts to rectify each of these shortcomings.

2. Data and Analytical Methods

Data are from the 1979 cohort of the NLSY, which initially included 12,686 persons aged 14 to 21 in 1979, with oversamples of blacks, Hispanics, low-income whites and military personnel. Annual interviews were conducted through 1994, with biennially interviews since that time. The military sample was dropped in 1984 and the low-income white sample in 1990; therefore we exclude both from our analysis. We also omit females pregnant at the interview date or who have given birth in the last year, since

reported weight is unlikely to represent their non-pregnancy weight.⁴ Much of the analysis is performed separately for men and women, reflecting potential differences in lifecycle patterns of body weight and SES effects.

The NLSY collects data on individual and family background characteristics during each interview year, with additional retrospective information available from the baseline (1979) survey. Questions about body weight were included in 1981, 1982, 1985, 1986, 1988, 1989, 1990, 1992, 1993, 1994, 1996, 1998, 2000, 2002 and 2004; those on height were included in 1981, 1982, and 1985 (Center for Human Resource Research, 2004).⁵ Our analysis assumes that height does not change after 1985, since all NLSY respondents are at least 20 years of age at that time.

Using the self-reported data on height and weight, we calculate the respondent's Body Mass Index (BMI), defined as weight in kilograms divided by height in meters squared.⁶ BMI is less accurate than laboratory measures of body composition because it does not account for variations in muscle mass or in the distribution of body fat (e.g. intra-abdominal versus overall adiposity). Nevertheless, BMI is the favored method of assessing excess weight because it is simple, rapid, and inexpensive to calculate.⁷

Federal and international guidelines define adults with a BMI below 18.5 as “underweight” while those with BMIs in the ranges of 18.5 to <25, 25 to <30 and ≥ 30 are “normal weight,” “overweight” and “obese” (World Health Organization, 1997;

⁴ This exclusion eliminates 4,827 person-year observations including a maximum (minimum) of 504 (27) females in 1985 (2004).

⁵ The questions are “How much do you weigh” and “How tall are you”? Weight is reported in pounds and height in inches.

⁶ Equivalently, BMI is weight in pounds divided by height in inches squared times 703.

⁷ Some researchers prefer other anthropometric measures such as waist circumference (Sönmez et al., 2003), waist-hip ratio (Dalton et al., 2003), or waist-height ratio (Cox and Whichelow, 1996). Cawley and Burkhauser (2006) have recently recommended the use of Bioelectrical Impedance Analysis (BIA). However, none of these are available in the NLSY.

National Heart, Lung, and Blood Institute, 1998).⁸ Obesity is further divided into three categories: class 1 (BMI 30 to <35), class 2 (BMI 35 to <40) and class 3 (BMI \geq 40).

Our analysis focuses on BMI, obesity and (for some descriptive analysis) class 3 obesity.

There are at least two issues with our use of these BMI standards. First, for children (under the age of 21) official statistics use a more complicated criterion based on gender and age-specific growth charts compiled by the Center for Disease Control and Prevention's National Center for Health Statistics (Kuczmarski et al., 2000).⁹ For consistency across individuals and over time, we use the adult obesity standard (BMI \geq 30) for all respondents, including those under 21 years old at the interview date. However, in preliminary analysis, we confirmed that our results were robust to use of the CDC measure of overweight for respondents under the age of 21.

Second, self-reported data on height and weight is measured with error in ways that lead to an understatement of the prevalence of obesity. Specifically, there is a tendency for height to be over-reported and weight to be understated (Strauss, 1999; Goodman et al., 2000; Kuczmarski et al., 2001), leading to an underestimate of BMI.¹⁰ A number of (not entirely satisfactory) regression-based procedures have been proposed for correcting the self-report errors (Bolton-Smith et al. 2000; Spencer et al., 2002, Plankey et al., 1997). In the economics literature, researchers (Cawley, 2004; Chou et al., 2004; Ruhm, 2005) have used variations of the following method: 1) regress clinical measures of weight (height) on a quadratic of the corresponding self-reported variable, using data

⁸ The WHO terms those with BMI of 25.0 to <30 as "preobese."

⁹ Specifically, youths are classified as "overweight" if their BMI is at or above the gender and age-specific 95th percentile and "at risk of overweight" if their BMI is between the 85th and 95th percentile because children's BMI varies systematically with age. See www.cdc.gov/nccdphp/dnpa/bmi/bmi-for-age.htm for further information.

¹⁰ The magnitude of the underestimate appears to increase with BMI.

from the third National Health and Nutrition Examination Survey (NHANES 3); 2) use the resulting prediction equation to estimate actual weight (height) as a function of self-reported values in a target data set (such as the NLSY) containing only the latter; 3) calculate BMI using the corrected values for weight and height.¹¹ We explored the implications of using this procedure but found that the findings were not substantively altered. Therefore, most results below are based on the (uncorrected) self-report data, although we briefly discuss estimates obtained using corrected BMI.

The weights of NLSY respondents were tracked from 1981 through 2004. To maintain a consistent sample across all survey years, we eliminated persons failing to provide valid information on body weight for any of the 15 interview years in which it was collected. Otherwise, changes in sample averages might have reflected non-random attrition (e.g. higher attrition rates among low-income individuals who also tend to weigh more).¹² However, as mentioned, we made the exception of excluding women in years they were pregnant or had recently given birth (but include them in other periods).

Our primary proxy for SES is the highest grade completed by the respondent's mother, measured at the 1979 interview date. We focus on the maternal education due to the large number of corresponding missing values for fathers and because mothers may be more instrumental in establishing the eating habits and health behaviors of children. We frequently divide the sample into "low" "medium" and "high" SES groups, defined according to whether the mother has fewer than 12, exactly 12 or greater than 12 years of

¹¹ Reporting errors are typically allowed to differ across gender and race/ethnicity groups by estimating separate equations or including interaction terms in the first-stage regression.

¹² To determine the implications of this exclusion restriction, we examined (unweighted) average BMI for each survey year that collected weight information first using our balanced sample and then with an unbalanced sample that included observations from respondents with missing information on BMI in one or more years. Despite substantial differences in sample size, average BMI was similar across the two samples for each year, although always slightly higher in the unbalanced sample. See Appendix Table A.1 for details.

completed schooling. Approximately 29, 49 and 22 percent of the NLSY cohort fall into these categories.¹³ The NLSY does not collect data required to create a measure of SES that is based on permanent income during the respondent's childhood. However, previous research (Zhang and Wang, 2004; Classen and Hokayem, 2005) indicates substantial health-SES gradients when proxying SES with schooling, suggesting the usefulness of our education-based measure. As alternatives, we also experiment with SES indicators based on respondent scores on the Armed Forces Qualification Test (AFQT) – an indicator of cognitive skill measured in 1981 – and family structure during childhood, classified by the presence or absence of the father in the household when the respondent was 14 years old.¹⁴ We do not consider the AFQT and family structure-based measures of SES to be as informative as those based on maternal education, and so comment only briefly on the results obtained when using them.

Additional explanatory variables are included in some specifications. These include: highest grade completed, family income, race/ethnicity, marital status, number of biological children and health behaviors. Except where noted, this information pertains to the respondent in interview year in which body weight was measured. Family income refers to income from all sources during the previous calendar year and is expressed in constant (2004 year) dollars, adjusting for price changes using the all-items Consumer Price Index. Marital status is captured by a dummy variable indicating that the respondent was married at the survey date, rather than being separated, divorced, widowed or never married.

¹³ Since the NLSY oversamples minorities, 40, 42 and 17 percent of the unweighted sample were classified as low, medium and high SES using this criteria.

¹⁴ The AFQT variable is the residual from a regression of AFQT scores on age dummy variables; this residual controls for differences in respondent ages at the time of testing.

We use two strategies to avoid losing observations, in our regression analysis, due to missing values of non-weight variables. First, for several covariates (age, respondent education, family income and married), we replace missing values with values averaged from adjacent survey years when this information is available. For instance, if data on family income are missing in 1985 but present in 1984 and 1986, we replace the missing 1985 value with the average of income in 1984 and 1986. Second, we set values of regressors still missing (after using the first procedure) to zero and include a dummy variable denoting the presence of a missing value.

Our controls for health behaviors are limited, both with regard to the information available and the years in which it was collected. For instance, we have data on alcohol consumption, cigarette smoking and exercise but none on diet or caloric intake. Our strategy for alcohol and cigarette consumption was to use information from the most recent previous survey in which the relevant questions were asked or from the first survey the data were collected, when this occurred subsequent to the relevant interview date. For instance, if questions were included in 1984, 1992, 1994 and 1998, we would use information from 1984 for all survey waves before 1992, 1992 data for 1992 and 1993, the 1994 questions for the 1994 through 1996 and 1998 responses for the remaining interviews.¹⁵ Detailed questions on physical activity were limited to 2002 and 2004. Therefore, we averaged values for these two years and applied the resulting measures to all survey years analyzed.¹⁶ This procedure relies on the assumption that physical activity is highly correlated over time, in which case data from later survey years provides useful information on exercise occurring earlier in life.

¹⁵ To reduce the number of missing values, we used values for one of the nearest adjacent survey years if these contained valid responses but information was not provided in the specified year.

¹⁶ The physical activity variables were constructed using information from 2002 (2004) only if the corresponding data were missing in 2004 (2002). Information on physical activity was also available for 1998 and 2000 but it was much less detailed than subsequently and so was not used in this analysis.

Drinking questions were included in 1983, 1984, 1988, 1989, 1994 and 2002. From these, we constructed a dummy variable indicating binge drinking (consumption of 6 or more drinks in a single session) during the previous month and heavy drinking (monthly consumption of more than 60 alcoholic beverages).¹⁷ Information on smoking, available in 1984, 1992, 1994 and 1998, was used to construct three dummy variables. The first indicated whether the respondent had smoked 100 or more cigarettes during his or her lifetime. The second and third indicated current and heavy smoking, with the latter defined as consumption of 20 or more cigarettes per day.

Information on the frequency and duration of “light/moderate” and “vigorous” physical activity and on the frequency of strengthening exercises was available in 2002 and 2004.¹⁸ Using average values over these two years, four exercise variables were created. Individuals were defined as “physically inactive” if they engaged in less than one hour of exercise per week and “moderately active” if they exercised one to two hours weekly. They were classified as “physically active” if exercising at least two hours per week but less than two hours vigorously. “Vigorous exercisers” engaged in more than two hours per week of vigorous physical activities. The final variable indicates participation in strengthening exercises at least once per week.¹⁹

Our intent is that the included health behaviors capture the effects of a broad range of lifestyle factors, but we do not apply a causal interpretation to the coefficient estimates

¹⁷ A drink is defined as “equivalent to a can of beer, a glass of wine, or a shot of hard liquor.” Data on binge drinking were also provided in 1985 and number of beverages consumed in 1992; however, the questions were not comparable to those in the other years.

¹⁸ Light/moderate physical are defined as those that “cause only light sweating or slight to moderate increases in breathing or heart rate”. Vigorous activities “cause heavy sweating or large increases in breathing or heart rate.” Strengthening exercises are those “specifically designed to strengthen ... muscles such as lifting weights or doing calisthenics.”

¹⁹ Information on the duration of strengthening exercises is not provided. Since this variable is the average value of dummy variables from two years (2002 and 2004) it can be thought of as the fraction of months in which the individual engages in strengthening activities.

obtained for them. For example, we make no attempt to resolve the ongoing debate on whether reductions in smoking plays a role in explaining the growth in obesity (Chou et al., 2004; Gruber and Frakes, 2006). Instead, we incorporate information on tobacco use in hope that it proxies the effects of a constellation of health behaviors that may be related to obesity.

Table 1 presents descriptive statistics for BMI, obesity and many of the covariates used in our regression analysis. Results are displayed for the full sample and subsamples stratified by gender and SES. The sample means are generally similar for men and women, although males are heavier, have higher incomes, rates of obesity, and fewer children. Men have lifestyles that are healthier in some ways (like exercising more) but less so in others – they are much more often binge or heavy drinkers and are somewhat more likely to be heavy smokers. The differences by SES are more pronounced. Compared to less advantaged individuals, high SES respondents are lighter, much less often obese, more educated, have higher incomes, smoke less and are more physically active. Differences between medium and low SES individuals generally follow the same patterns, except that the middle group has relatively high rates of binge and heavy drinking. The fraction African-American falls monotonically with SES, but Hispanics are disproportionately represented among the lowest SES category. These differences motivate the use of multivariate regression analysis.

3. Age-Related Changes in Body Weight Trends

Figure 1 and Table 2 provide descriptive evidence of the growth in body weight occurring over time for the NLSY cohort. Unless otherwise noted, all results below incorporate sampling weights to provide nationally representative estimates. Standard errors,

shown in parentheses, are corrected for complex survey design using the Taylor-series linearization methods included in STATA (StataCorp, 2005).²⁰

The kernel density estimates demonstrate that much of the growth in self-reported BMI has occurred in the right-tail of the distribution (Figure 1). This is consistent with evidence using clinical data (Ruhm, 2007) and helps to explain why obesity and severe obesity have increased much faster than average body weight. Thus, as shown in Table 2, average self-reported BMI of NLSY respondents rose 23 percent (from 22.3 to 27.4 kg/m²) between 1981 and 2004, while obesity prevalence increased almost 8-fold (from 3.0 to 26.5 percent) and class 3 obesity by even more (from <0.1 to 2.5 percent). Average BMI and the prevalence of obesity (but not class 3 obesity) of males is higher than that of females, which initially seems surprising since clinical measures from NHANES show higher adult obesity rates for women than men (Flegal et al., 2002; Ogden et al., 2006). One explanation for this result, which has also been obtained by others using the NLSY (e.g. Cawley, 2004), is that self-reported BMI is likely to be understated by larger amounts for women than men in this survey. Furthermore, since clinical data indicate that women are less often overweight, even though more frequently obese, an equal underestimate of BMI for both genders could cause more clinically obese females to be classified as overweight based on self-report data.²¹

These reporting errors are not a major issue if they are similar across time periods and SES groups, but may be more problematic if this is not the case.

²⁰ This is necessary because the NLSY is a multi-stage, stratified sample with geographically clustered respondents. As a result, respondents within clusters tend to be relatively similar and standard errors calculated assuming random sampling will be understated. The corrected standard errors require information on the strata and primary sampling unit, which are provided in the restricted NLSY Geocode file.

²¹ Consistent with these possibilities, when we corrected for reporting errors using the procedures detailed above, the average BMI of females in our sample rose 2.0 percent, compared to 0.5 percent for males. Similarly, the estimated obesity prevalence grew 17.0 percent for women versus 9.0 percent for men.

The last three rows of Table 2 show how the results differ with SES, as proxied by maternal education. BMI, obesity and class-3 obesity are more common and increase faster over time for low than high SES sample members. Thus, while average BMI rose 5.4 kg/m² and obesity prevalence by 29.2 percentage points between 1981 and 2004 for the lowest SES group, corresponding increases were 5.1 kg/m² and 22.2 points for the middle category and 4.5 kg/m² and 18.7 points for the most advantaged respondents.²²

BMI and obesity trended upward in the United States throughout the NLSY sample period, implying that the results just described combine the effects of aging and these secular changes. The following procedure was employed to isolate the effects of aging. Data from the NHANES were first used to calculate changes in the average BMI of 24-38 year olds occurring between 1976-80 and 1999-2004. This was next converted to an annualized increase, using a linear trend, and adjustments to eliminate these effects were made to the BMI of each NLSY respondent. Finally, the adjusted BMI values were used to calculate mean BMI and compute obesity prevalence. The correction factors were implemented separately for males and females for estimates involving subsamples stratified by gender. Separate adjustments were not used for SES subsamples, since NHANES does not contain information on the mother's education (our main SES proxy). Previous research indicates that SES-BMI differentials are narrowing over time (Zhang and Wang, 2004; Chang and Lauderdale, 2005), suggesting that the results below provide a lower bound on the extent to which SES gradients steepen with age.²³

²²Disadvantaged respondents similarly exhibit larger weight gains when SES is measured by AFQT scores of the mother or presence of a father in the household at age 14, although class 3 obesity increases at similar rates regardless of the father's presence.

²³ This occurs because a full adjustment would result in a larger (smaller) increase in BMI for high (low) SES individuals at young ages.

An example helps to illustrate the procedure. NHANES data indicate that the average BMI of 24-38 year olds was 24.734 kg/m² in 1976-80 and 27.609 kg/m² in 1999-2004, implying an increase of 2.846 kg/m², or 0.119 kg/m² per year.²⁴ The trend variable *T* takes values ranging from 0 in 2004, the most recent NLSY interview, to 23 in 1981, the first year for which body weight data are available. Adjusted BMI is therefore calculated by adding 0.119 kg/m² x *T* to the respondent's self-reported BMI in the specified year. This has the effect of increasing BMI at younger ages, since these correspond to earlier survey dates and reflect the higher body weight that would have been expected using the 2004 BMI distribution. Trends in the NHANES data were calculated for 24-38 year olds because all NLSY sample members were eligible to be interviewed in this age range – the oldest respondents were 24 in 1981 and the youngest were 38 in 2004. The results, however, were not sensitive to this choice.²⁵

BMI and obesity prevalence increase rapidly with age. As shown in Figure 2, average BMI rises from 21.6 to 26.9 kg/m² between the ages of 18 and 40, while obesity prevalence increases from 1.0 to 23.2 percent (see Appendix Table A.2 for additional details). Over two-fifths of the BMI growth is due to secular trends, however, rather than the effects of aging, so that adjusted BMI rises from 24.3 to 27.3 kg/m². This correction has less effect on obesity – adjusted prevalence is 3.5 percent at age 18 and 24.9 percent for 40-year olds – because the procedure does not affect the obesity status of individuals with BMI substantially above or below the threshold level.

²⁴ This calculation is based on a 24-year average difference in the timing of interviews in NHANES 2 (1977.5) and the most recent NHANES survey (2001.5).

²⁵ For instance, the average annual change in BMI was 0.126 kg/m² for 16-48 year olds (representing full age range of the NLSY sample analyzed).

Figure 3 displays the age-related changes in BMI and obesity for gender and SES subgroups, all adjusted for secular trends in average BMI. Four points are noteworthy. First, BMI and obesity rise with age for all subsamples. Second, the age-related increase in BMI is somewhat faster for men than women but the growth in obesity prevalence is quite similar. Generally, the evolution of body weight over the ages studied does not vary sharply with gender. Third, not only do high SES individuals have lower BMI and obesity prevalence at all ages but, using the terminology of Case et al. (2002), the gradient rotates (steepens with age). For instance, 4.6 percent of lowest SES group are obese at age 18 and 31.3 percent at 40 years of age. Corresponding growth for their high SES counterparts is from 1.9 to 19.6 percent. The SES differences are also more pronounced for obesity than BMI, reflecting the importance of differences in the right tail of the distribution. Fourth, although the age profiles show weak evidence of concavity, a linear approximation is generally reasonable, particularly for the age range (24-38) covering all NLSY respondents.

4. Empirical Methods

We next use regression analysis to explore the association between age, SES and body weight. Our basic specification takes the form:

$$Y_{it} = \alpha_0 + \alpha_1 X_{it} + \alpha_2 AGE_{it} + \alpha_3 SES_i + \varepsilon_{it}, \quad (1)$$

where Y_{it} is the outcome (either BMI or obesity) for individual i at time t , X is a vector of control variables, AGE is the respondent's age at the survey date, SES is socioeconomic status, and ε is assumed to be a mean-zero error term. As discussed, maternal education is our primary proxy for SES, although other measures are sometimes used. The covariates included in X vary by model specification. All equations contain survey year dummy

variables. We do not initially include other controls (for race/ethnicity, respondent education, family income or health behaviors) because they may represent mechanisms through which SES is transmitted and so holding them constant would absorb a portion of the SES effect. However, we add them to subsequent models when attempting to understand how SES operates. Equation (1) is estimated for the full sample as well as for subsamples stratified by gender.

The assumption that the SES gradients are age-invariant can be relaxed by estimating:

$$BMI_{it} = \alpha_0 + \alpha_1 X_{it} + \alpha_2 AGE_{it} + \alpha_3 SES_i + \alpha_4 AGE * SES_{it} + \varepsilon_{it} \quad (2)$$

where $AGE * SES$ is the interaction between SES and age. For ease of interpretation, we generally express age and SES (when using a continuous proxy) as deviations from the sample averages. As a result $\hat{\alpha}_2$, $\hat{\alpha}_3$ and $\hat{\alpha}_4$ indicate marginal effects evaluated at means of the respective variables. Sample weights are incorporated throughout to provide nationally representative estimates and the standard errors account for complex survey design.

We report results of linear probability (LP) models when obesity is the dependent variable. Preliminary analysis revealed similar predicted effects when from logit or probit estimates, but the coefficients from the LP models are easier to interpret. This is especially true when including the age-SES interactions, where marginal effects depend on the values of the covariates and the associated coefficients are often misleading. For instance, Ai and Norton (2003) point out that the coefficients may have the opposite sign as the predicted effect of the interaction on the dependent variable.²⁶

5. Econometric Estimates of Age-Profiles and SES-Profiles

²⁶ Indeed, in preliminary work, we sometimes obtained positive coefficients on the AGE-SES interaction, even though the predicted values from the same equation indicated that the obesity rates of low SES respondents increased with age relative to those of their high SES counterparts.

Our initial econometric analysis examines age and SES gradients in BMI and obesity equations, with additional covariates limited to gender and the survey year. These results are summarized in Table 3. For each specification, column (b) includes an age-SES interaction whereas column (a) does not.

The first model controls for low and high SES, with the middle category (exactly 12 years of maternal education) constituting the reference group. These estimates correspond to the low, medium and high SES categories used in the descriptive analysis above. BMI is predicted to increase by 0.13 kg/m² and obesity prevalence by 0.60 percentage points per year of age (see column 1a). Low SES sample members are anticipated to have a BMI 0.74 kg/m² (1.39 kg/m²) above that of their medium (high) SES peers and a 4.3 (8.4) percentage point greater obesity prevalence. All these differences are statistically significant. The results displayed in specification (1b) indicate that the SES disparities widen with age. For instance, the BMI difference between low and high SES individuals is predicted to rise by 0.040 kg/m² per year of age, or 0.80 kg/m² over 20 years. The SES gap in obesity is expected to widen even more – by 0.41 percentage points per year or 8.2 points over 20 years. These magnitudes are reasonably consistent with those in the descriptive analysis where, for example, the BMI disparity between low and high SES sample members increased from 0.5 kg/m² for 20-year olds to 1.3 kg/m² at age 40, while the gap in obesity prevalence rose from 6.2 to 11.7 percentage points (see Table A.2).

The remainder of Table 3 specifies SES as a continuous variable measured by years of schooling completed by the respondent's mother. The basic model indicates that BMI (obesity prevalence) rises by a statistically significant 0.12 kg/m² (0.6 percentage points) per year of age and 0.20 kg/m² (1.2 percentage points) for each additional grade of maternal

education (see columns 2a and 2b). The age gradients are identical to those in model (1a) and the SES gradients accord closely with them. This can be seen by noting that maternal education is 3.2 (6.3) years greater for medium (high) than for low SES sample members. Model (2a) predicts that differences of this size result in 0.65 (1.27) kg/m² greater BMI for low than high (medium) SES individuals and a 3.8 (7.6) percentage point disparity in obesity prevalence. The corresponding SES gaps predicted by model (1a) were 0.74 (1.39) kg/m² and 4.3 (8.4) percentage points. Inclusion of an age-SES interaction provides further evidence that the SES gradients widen with age. The predicted effects are 0.007 kg/m² per year for BMI and 0.07 percentage points annually for obesity (see model 2b). To place these in perspective, the 10th (90th percentile) of maternal education is 9 (16) years of schooling and the BMI (obesity) gap for sample members whose mothers had these amounts of schooling is predicted to rise by 1.04 kg/m² (9.7 percentage points) between the ages of 20 and 40.

The continuous SES classification is convenient because it is easy to interpret and uses all available information on the mother's education. A concern, however, is that the results obtained may be sensitive to the treatment of outliers, particularly if the effects are nonlinear and because a substantial portion of the sample report that their mother received very little schooling.²⁷ To address this issue, columns (3a) and (3b) show the results of models where the SES variable has been "winsorized" (Angrist and Krueger, 1999) by setting maternal education less (greater) than the 5th (95th) percentile to 7 (16) years, which is the level of schooling at that percentile. The age profiles estimated from these specifications are essentially identical to those in models (2a) and (2b), while the SES gradients are 25 to 30

²⁷ For instance, fewer than six years of maternal education are reported for almost 6 percent of (unweighted) observations.

percent larger. This is not surprising since winsorizing compresses the variance of the SES variable. The estimates suggest that the previous results provide conservative predictions of the SES disparities.

An additional concern is it may be difficult to separate the effects of cohort aging and secular time trends, particularly at the top and bottom of the NLSY age range since observations at these ages will only be obtained early or late in the sample period. For example, data on 18-19 year olds come from 1981-82 and that on 46-47 year olds are from 2004. This problem can be reduced by limiting the sample to 24-36 year olds, for whom sample observations were available in each survey year. The results of doing so, shown in columns (4a) and (4b), are fairly similar to those using the full age range. The age coefficients and age-SES interactions are virtually identical to prior BMI equations and are somewhat larger for the prevalence of obesity. The SES coefficients are slightly greater as well, but the differences are modest. These results suggest that the findings reported below may understate the obesity gradients.

We conducted several other tests of robustness. First, to examine whether the findings were sensitive to the exclusion of 3,828 person-year observations (5.4 percent of the sample) lacking information on maternal education, we estimated models that included these observations, with mother's education coded to zero and a missing education dummy variable included as an additional covariate. None of the coefficients are substantially affected by this, with the largest change being a 5 percent reduction for the parameter estimate on age in the obesity equation. Second, to allow for nonlinear age effects, we added controls for age squared and its interaction with SES. The BMI models provided modest

evidence of concavity.²⁸ For obesity, the quadratic terms were small and insignificant, suggesting that the linear specification is reasonable. Third, we estimated models that removed the effects of secular changes in body weight from individual values, using trend estimates from the NHANES data and adjustment procedures described in section 3. The BMI estimates were identical to those previously reported and the obesity predictions were similar, although with somewhat steeper average age and SES disparities and less rotation of the SES gradient.²⁹ Finally, we ran specifications where BMI and obesity were corrected for errors in self-report data, using the methods discussed in section 2. Coefficients on SES and its interaction with age were essentially unaffected by this, while the parameter estimates for age increased by 10 to 17 percent.

We also examined results using the two alternative proxies for SES – the AFQT score of the respondent in 1981 and the presence of his/her father in the household at age 14. Both substitutes yielded results consistent with those using maternal education: BMI and obesity prevalence were lower for sample members who had high AFQT scores or a father in the household while growing up, and these disparities increased with age.

Age and SES gradients in BMI and obesity have been constrained to be the same for males and females up to this point. This will be a misspecification if there are gender differences in the evolution of body weight over the lifecycle or on the influences of family background in determining these outcomes. Table 4 examines this issue by providing separate estimates for males and females. Here and for the remainder of the analysis, we

²⁸ In these specifications, age rather than its deviation from sample mean values was controlled for. For BMI, the quadratic on age was statistically significant and indicated that BMI increases through 43 years of age. Similarly, the SES gradient was predicted to widen through age 59.

²⁹ The predicted SES gap in obesity was approximately twice as large for 20-year olds, as when using the unadjusted data, but essentially the same at age 40 using either method.

estimate variations of our “preferred” specification, corresponding to columns (2a) and (2b) of Table 3.

Table 4 demonstrates that the main results for males and females correspond to those for the combined sample: BMI and obesity increase with age and decline with SES, with larger SES disparities observed at more advanced ages. The average age gradient is similar for the two genders but the SES effects are considerably larger for women. For instance, the SES coefficient is over twice as big for females as males for BMI and 50 percent greater for obesity in the model without age interactions (columns 1a and 2a). Similarly, the interaction coefficients indicate that the SES disparities become much more pronounced with age for women (models 1b and 2b).

Predicted BMI and obesity prevalence for the full sample and gender-stratified subsamples are summarized in Table 5. Individuals are evaluated at 20 and 40 years of age and at 9 and 16 years of maternal education.³⁰ As noted, BMI and obesity prevalence is expected to increase with age for all groups and to decline with SES, while the SES-gradient widens with age. These disparities are particularly pronounced for obesity, which depends on outcomes in the right tail of the BMI distribution. For instance, the predicted obesity rate of persons whose mothers had 9 years of schooling almost triples between the ages of 20 and 40 (rising from 9.0 to 24.7 percent). Obesity prevalence of their advantaged counterparts is expected to be two-thirds as large (6.0 percent) at age 20 but less than half as great (12.1 percent) by 40 years of age. It is noteworthy that the obesity rate of 40-year olds high SES individuals is only slightly bigger than for the disadvantaged peers at age 20.

Table 5 also details the steeper age and SES gradients predicted for women than men. For instance, 20-year old females whose mothers had 9 years of education are 25 percent less

³⁰ These values of maternal education correspond to the 10th and 90th sample percentiles respectively.

likely to be obese than corresponding men (7.7 versus 10.2 percent) but have essentially identical predicted prevalence at age 40 (24.3 versus 24.7 percent). However, since obesity is projected to grow more slowly with age for advantaged females, 40-year old women whose mothers had 16 years of schooling are less than three-fifths as likely to be obese as their male peers (8.6 versus 15.1 percent).

6. Intergenerational Transmission Processes

The preceding analysis established that BMI and obesity prevalence rise during the transition from early to middle adulthood, that individuals growing up in disadvantaged families weigh more at given ages, and that the SES gradients widen with age. We next examine potential mechanisms or processes through which the effects of SES propagate across generations. The results refer to body weight but may be more generally applicable to other health inputs and outcomes.

We examine five specific transmission mechanisms: race/ethnicity, education, family income, household composition and individual health behaviors. Specifically, we test the hypotheses that: 1) some effects of SES either operate through or are linked to race-based disparities; 2) high SES children have better adult outcomes because they obtain more education than their disadvantaged peers; 3) holding education constant, persons growing up in advantaged households will subsequently have relatively high family incomes, with consequent health benefits; 4) SES is correlated with marriage and fertility rates, both of which may in turn be associated with BMI and obesity; 5) SES during the early years is linked to subsequent health behaviors related to body weight and health.

Before turning to the data, we emphasize several caveats that should be kept in mind when interpreting the results. First, the observed linkages are not necessarily causal. For

instance, we will not be able to determine whether race/ethnicity disparities result from genetic differences, measurement error in the other control variables (e.g. the quality of education) or omitted characteristics. Similarly, the sources of any SES differences that transmit through education will not be identified, except to the extent that the models directly control for them. For instance, we do not attempt to determine whether the schooling impacts operate through differences in discount rates or the speed of information acquisition on healthy lifestyles. Second, our controls are often quite limited, raising the likelihood of significant omitted variables biases. For example, data on physical activity is available only late in the sample period (2002 and 2004) and may be measured with significant error.³¹ Similarly, the NLSY lacks any information on energy intake, a presumably important determinant of body weight. Third, the mechanisms for any observed correlations may be difficult to ascertain, even if the latter are causal. Thus, even if the higher education levels obtained by advantaged children result in lower rates of subsequent obesity, we do not know whether this reflects differences in lifestyles, access to health information, schooling-induced changes in preferences or other unobserved factors.

The beneficial effects of advantaged family backgrounds during childhood on future body weight are primarily transmitted through education and, to a lesser extent, race/ethnicity. The first set of estimates, summarized in Table 6 (with additional details in Appendix Table A.3) do not include age-SES interactions and so constrain the SES gradients to be age-variant. The results in model (1) correspond to those presented previously (in Tables 4 and 5). Notice that the full sample SES coefficients fall by 26 percent for BMI and

³¹ Information on the frequency of exercise is provided through one variable listing the number of times the respondent engages in the activity and a second indicating the unit of time (daily, weekly, monthly or yearly). However, there appears to be significant coding errors for the latter variable. Similar but less severe problems exist for measuring exercise duration. We have made a substantial effort and used reasonable assumptions to correct for these problems but suspect that some measurement error remains.

35 percent for obesity with the addition of controls for the respondent's education (specification 2) and by 15 to 16 percent with the inclusion of race/ethnicity covariates (model 3). The two effects are essentially additive, as can be seen by noting that the SES coefficient is attenuated by 41 percent for BMI and 50 percent for obesity when education and race/ethnicity are simultaneously included (column 4).³²

Conversely, little of the SES effect appears to be propagated through family incomes, household composition, or the available health behaviors related to smoking, drinking and physical activity. Higher incomes predict lower body weight but the effects are small – attenuating the SES coefficient by around 4 to 5 percent when entered in the model alone. Including controls for household composition and health behaviors has even less effect – attenuating the SES parameter by less than 1 percent for BMI and below 5 percent for obesity. These effects are minimal because the household composition coefficients, while in the expected direction (being single and having more children is associated with higher body weight), are small in magnitude, while the behavior parameters provide little consistent evidence of lower weight being predicted by the lifestyles that are common among high SES individuals.³³ When including all supplementary controls in the model simultaneously (column 5), 44 (55) percent of the SES effect on BMI (obesity) is “explained,” with 93 (91) percent of this being due to race/ethnicity and income.³⁴

Broadly similar results are obtained when examining men and women separately (see the bottom four rows of Table 6). Education and race/ethnicity attenuate the SES parameters

³² Also, as shown in Table A.3, the education and race/ethnicity coefficients change little from models (2) or (3) to model (4).

³³ Specifically, current or past smoking is associated with lower body weight and binge drinking with higher BMI (but not obesity), with inconsistent patterns for the exercise variables.

³⁴ The results were not materially affected by including a quadratic in income or replacing the continuous education variable with categorical regressors (high school dropout, high school graduate, some college, or post-graduate education).

of men by 30 to 47 and 14 to 15 percent, when included separately, and by 45 to 62 percent if entered in combination. When family income, household composition and health behaviors are also controlled for, the magnitude of the SES coefficient declines by 48 to 68 percent. Education and race/ethnicity separately attenuate the SES parameter for females by 24 to 30 and 15 to 19 percent, and by 42 to 44 percent when included together. The SES coefficient falls 47 to 50 percent, in absolute value, in the model containing all of the supplementary covariates.

Next, we allowed the SES gradients to vary with age, by adding age-SES interactions to the models estimated in Table 6.³⁵ Since the resulting coefficient estimates are somewhat complicated to interpret, we used them to calculate predicted BMI and obesity prevalence for high and low SES sample members (defined as in Table 5) at 20 and 40 years of age. Differences between the two SES groups predicted in the basic model (and previously displayed in Table 5) were labeled as the “SES gap.” We then calculated the SES disparity predicted after controlling for supplementary covariates and calculated the share of the initial gap “explained” by the newly-included explanatory variables. An example illustrates the procedure. In the basic model, low SES 20-year olds were predicted to have a BMI of 24.134 kg/m² and their high SES counterparts a BMI of 23.309 kg/m², implying a gap of 0.825 kg/m². After adding education to the model, predicted BMI was 24.030 and 23.339 kg/m² for low and high SES 20-year olds, yielding a gap of 0.691 kg/m², which is 16.2 percent smaller than the initial disparity.

Results of this exercise, displayed in Table 7, confirm the dominant roles of education and, to a lesser extent, race/ethnicity in transmitting the effects of family background. The

³⁵ Additional interactions between age and respondent education, race/ethnicity were also included in specifications containing main effects for these variables.

decomposition procedure also shows that the supplementary variables account for virtually all of the SES differential in obesity at age 20 (but much less of the difference in BMI). However, these gaps are quite small, since 20-year olds are relatively infrequently obese. Much larger SES disparities are predicted, for both BMI and obesity, among 40-year olds and just under half of the predicted SES gradient at this age can be accounted for by the combination of controls for respondent education, race/ethnicity, family income, household composition and health behaviors. Schooling and race/ethnicity individually account for 59-63 and 33-38 percent of the explained portion of the gap. Education represents a particularly important propagation mechanism for males, where it independently accounts 39 to 43 percent of the total SES gap at age 40, compared to 23 to 24 percent for females.

7. Discussion

BMI and the prevalence obesity rise as individuals transition from early through middle adulthood. The increases are approximately linear over most ages examined, although with some evidence of concavity for the oldest sample members. Growth in body weight for the NLSY cohort represents a combination of the effects of aging and secular trends. Removing the latter reduces but does not eliminate the age-related increases in weight. The econometric estimates indicate that BMI rises by about 0.12 kg/m^2 per year of age, while obesity prevalence grows by around 0.6 percentage points per year. These estimates are virtually identical for men and women.

Excess body weight is inversely related to SES at all observed points of the lifecycle and these disparities increase with age. Our main proxy for SES is the number of years of schooling obtained by the respondent's mother. The regression estimates indicate that an additional year of maternal education reduces BMI (obesity) by an average of 0.20 kg/m^2

(1.2 percentage points) and that this effect rises by 0.07 kg/m² (0.07 points) per year of age, with considerably larger SES disparities predicted for women than men.

We provide a preliminary examination of the mechanisms by which the beneficial effects of childhood advantage are transmitted to future weight outcomes. Our analysis highlights the importance of educational attainment and race/ethnicity as propagation mechanisms. When entered into the models separately, the inclusion of years of schooling attenuates the average SES effect by 26 to 35 percent and race/ethnicity does so by 15 to 16 percent. The role of these factors is not uniform. For instance, in combination, they explain a large majority of the (relatively small) SES gap in the obesity of 20-year olds but less of the (larger) disparity observed at age 40. Conversely, little of the SES effect appears to be propagated through differences in family income, marital status, number of children, or the health behaviors included in our models.

Evidence that SES disparities in BMI and obesity grow with age is consistent with the findings of related research focusing on other health outcomes. As with that literature, the pathways for these effects are only partially understood. The inclusion of education and race/ethnicity covariates explains (in a statistical sense) close to half of the disparity observed at age 40 and an even larger share of the gap for males. Yet between one-third and three-fifths of the differential predicted for 40-year olds remains unaccounted for, after including the full set of controls. We know even less about how education and race/ethnicity operate. Such uncertainty is by no means unique to this study. For instance, Cutler and Lleras-Muney (2006, p.1-2), in their careful review of the evidence, state that “work on the mechanisms underlying the link between health and education has not been conclusive. Not all theories have been tested and ... studies often will conflict with each other.” Similarly, racial

disparities in health outcomes such as infant mortality are large, persistent and difficult to explain (Stockwell et al., 2005).

We find little evidence that the SES gradients in body weight are related to income or the health behaviors controlled for (drinking, smoking and exercise) – either directly or mediated through education or race/ethnicity. A possible reason is that these determinants are poorly measured (e.g. information on physical activity is only included in the later NLSY survey years). Perhaps even more importantly, we lack data on other key inputs, such as those related to eating patterns and diet, which may have a larger impact on the outcomes.

Our findings should also be interpreted in light of several other caveats. For example, the self-reports of height and weight contained in the NLSY are likely to be measured with error and it is not clear whether these reporting inaccuracies differ with SES. Future investigations should also experiment more fully with alternative measures of socioeconomic status and, as additional data become available, investigate whether the patterns observed here persist into middle and late adulthood. It would also be interesting to link the results obtained for BMI and obesity more closely to other health outcomes, including those directly affected by excess weight.

References

- Adams, Peter, Michael D. Hurd, Daniel McFadden, Angela Merrill, and Tiago Ribeiro. 2003. "Healthy, Wealthy, and Wise? Test for Direct Causal Paths Between Health and Socioeconomic Status" *Journal of Econometrics* 112(1), January, 3-56.
- Ai, Chunrong, and Edward C. Norton. 2003. "Interaction Terms in Logit and Probit Models" *Economic Letters* 80(1), 123-129.
- Allison, David B., Kevin R. Fontaine, JoAnne E. Manson, June Stevens, and Theodore B. VanItallie. 1999. "Annual Deaths Attributable to Obesity in the United States" *JAMA* 282(16), October 27, 1530-1538.
- Andreyeva, Tatiana, Roland Sturm, and Jeanne S. Ringel. 2004. "Moderate and Severe Obesity Have Large Differences in Health Care Costs" *Obesity Research* 12(12), December, 1936-1943.
- Angrist, Joshua D., and Alan B. Krueger. 1999. "Empirical Strategies in Labor Economics" in Orley Ashenfelter and David Card (eds.) *Handbook of Labor Economics, Volume 3A*. Amsterdam: Elsevier, 1277-1366.
- Averett, Susan, and Sanders Korenman. 1996. "The Economic Reality of the Beauty Myth" *Journal of Human Resources* 31(2), Spring, 304-330.
- Baum, Charles L., and William F. Ford. 2004. "The Wage Effects of Obesity: A Longitudinal Study" *Health Economics* 13(9), September, 885-899.
- Bolton-Smith, Caroline, Mark Woodward, Hugh Turnstall-Pedoe, and Caroline Morrison. 2000. "Accuracy of the Estimated Prevalence of Obesity from Self-Reported Height and Weight in an Adult Scottish Population" *Journal of Epidemiology and Community Health* 54(2), February, 143-148.
- Borghans Lex, and Bart H. H. Golsteyn. 2006. "Time Discounting and the Body Mass Index: Evidence from the Netherlands" *Economics and Human Biology* 4(1), January, 39-61.
- Case, Anne, Darren Lubotsky, and Christina Paxson. 2002. "Economic Status and Health in Childhood: The Origin of the Gradient." *American Economic Review* 92(5), December, 1308-1334.
- Case, Anne, Angela Fertig, and Christina Paxson. 2005. "The Lasting Impact of Childhood Health and Circumstance" *Journal of Health Economics* 24(2), March, 365-389.
- Cawley, John. 2004. "The Impact of Obesity on Wages" *Journal of Human Resources* 39(2), Spring, 451-474.
- Cawley, John, and Richard V. Burkhauser. 2006. "Beyond BMI: The Value of More Accurate Measures of Fatness and Obesity in Social Science Research" National Bureau of Economic Research Working Paper No. 12291, July.
- Center for Human Resource Research. 2004. *The National Longitudinal Surveys NLSY User's Guide, 1979-2004*. U. S. Department of Labor, Bureau of Labor Statistics. Columbus, OH: Center for Human Resource Research, Ohio State University.

- Chang, Virginia W., and Nicholas A. Christakis. 2005. "Income Inequality and Weight Status in US Metropolitan Areas" *Social Science and Medicine* 61(1), July, 83-96.
- Chang, Virginia W., and Diane S. Lauderdale. 2005. "Income Disparities in Body Mass Index and Obesity in the United States, 1971-2002" *Archives of Internal Medicine* 165(18), October 10, 2122-2128.
- Chou, Shin-Yi, Michael Grossman, and Henry Saffer. 2004. "An Economic Analysis of Adult Obesity: Results from the Behavioral Risk Factor Surveillance System" *Journal of Health Economics* 23(3), May, 565-587.
- Classen, Timothy, and Charles Hokayem. 2005. "Childhood Influences on Youth Obesity" *Economics and Human Biology* 3(2), July, 165-187.
- Cox, Brian D., and Margaret J. Whichelow. 1996. "Ratio of Waist Circumference to Height is Better Predictor of Death than Body Mass Index" *British Medical Journal* 313(7070), December 17, 1487-1488.
- Currie, Janet, and Rosemary Hyson. 1999. "Is the Impact of Health Shocks Cushioned by Socioeconomic Status? The Case of Low Birthweight" *American Economic Review* 89(2), May, 245-250.
- Currie, Janet, and Mark Stabile. 2003. "Socioeconomic Status and Child Health: Why Is the Relationship Stronger for Older Children?" *American Economic Review* 93(5), December, 1813-1823.
- Cutler, David M., Edward L. Glaeser, and Jesse M. Shapiro. 2003. "Why Have Americans Become More Obese?" *Journal of Economic Perspectives* 17(3), Summer, 93-118.
- Cutler, David M., and Adriana Lleras-Muney. 2006. "Education and Health: Evaluating Theories and Evidence" National Bureau of Economic Research Working Paper No. 12352, June.
- Dalton, M., A. J. Cameron, P. Z. Zimmet, J. E. Shaw, D. Jolley, D. W. Dunstan et al. 2003. "Waist Circumference, Waist-Hip Ratio and Body Mass Index and Their Correlation with Cardiovascular Disease Risk Factors in Australian Adults" *Journal of Internal Medicine* 254(6), December, 555-563.
- Drewnowski, Adam, and S. E. Specter. 2004. "Poverty and Obesity: The Role of Energy Density and Costs" *American Journal of Clinical Nutrition* 79(1), January, 6-16.
- Finkelstein, Eric A., Ian C. Fiebelkorn, and Guijing Wang. 2003. "National Medical Spending Attributable to Overweight and Obesity: How Much, and Who's paying" *Health Affairs* May 14; web exclusive, w3.219-w3.226.
- Flegal, Katherine M., Rong Wei, and Cynthia Ogden. 2002. "Weight-for-Stature Compared with Body Mass Index-for-Age Growth Charts for the United States from the Centers for Disease Control and Prevention" *American Journal of Clinical Nutrition* 75(4), April, 761-766.
- Flegal Katherine M., Barry I. Graubard, David F. Williamson, and Mitchell H. Gail. 2005. "Excess Deaths Associated with Underweight, Overweight, and Obesity" *JAMA* 293(15), April 20, 1861-1867.

- Fontaine, Kevin R., David T. Redden, Chenxi Wang, Andrew O. Westfall, and David B. Allison. 2003. "Years of Life Lost Due to Obesity" *JAMA* 289(2), January 8, 187-193.
- Fuchs, Victor R. 2004. "Reflections on the Socio-economic Correlates of Health" *Journal of Health Economics* 23(4), July, 653-661.
- Goodman, Elizabeth, Beth R. Hinden, and Seema Khandelwal. 2000. "Accuracy of Teen and Parental Reports of Obesity and Body Mass Index" *Pediatrics* 106(1), July, 52-58.
- Gruber, Jonathan, and Michael Frakes. 2006. "Does Falling Smoking Lead to Rising Obesity?" *Journal of Health Economics* 25(2), March, 183-197.
- Guo, Shumei Sun, Wei Wu, William Cameron Chumlea, and Alex F. Roche. 2002. "Predicting Overweight and Obesity in Adulthood from Body Mass Index Values in Childhood and Adolescence" *American Journal of Clinical Nutrition* 76(3), September, 653-658.
- Kuczmarski, Robert J., Cynthia L. Ogden, Laurence M. Grummer-Strawn, et al. 2000. *CDC Growth Charts: United States. Advance Data from Vital and Health Statistics, no. 314*. Hyattsville, MD: National Center for Health Statistics.
- Kuczmarski, Marie Fanelli, Robert J. Kuczmarski, and Matthew Najjar. 2001. "Effects of Age on Validity of Self-Reported Height, Weight, and Body Mass Index: Findings from the Third Health and Nutrition Examination Survey, 1988-1994" *Journal of the American Dietetic Association* 101(1), January, 28-34.
- Marmot, M., G. D. Smith, S. Stansfeld, C. Patel, F. North, J. Head, I. White, E. Brunner, and A. Feeny. 1991. "Health Inequalities Among British Civil Servants: The Whitehall II Study" *Lancet* 337(8754), June 8, 1387-93.
- McTigue, Kathleen M., Joanne M. Garrett, and Barry M. Popkin. 2002. "The Natural History of the Development of Obesity in a Cohort of Young U.S. Adults Between 1981 and 1998" *Annals of Internal Medicine* 136(12), June 18, 857-864.
- McTigue Kathleen, Joseph C. Larson, Alice Valoski, Greg Burke, Jane Kotchen, Cora E. Lewis, Marcia L. Stefanick, Linda Van Horn, and Lewis Kuller. 2005. "Mortality Outcomes and Cardiac and Vascular Outcomes in Extremely Obese Women" *JAMA* 296(1), July 5, 79-86.
- Mokdad, Ali H., Barbara A. Bowman, Earl S. Ford, Frank Vinicor, James S. Marks, and Jeffrey P. Koplan. 2001. "The Continuing Epidemic of Obesity and Diabetes in the United States" *JAMA* 286(10), September 21, 1195-1200.
- Must, Aviva, Jennifer Spadano, Eugenie H. Coakley, Alison E. Field, Graham Colditz, and William H. Dietz. 1999. "The Disease Burden Associated With Overweight and Obesity" *JAMA* 282(16), October 27, 1523-1529.
- National Heart, Lung, and Blood Institute. 1988. *Clinical guidelines on the identification, evaluation, and treatment of overweight and obesity in adults: the evidence report*. Washington D.C.: US Government Printing Office.
- Noonan, Kelly, Nancy E. Reichman, and Hope Corman. 2005. "New Fathers' Labor Supply: Does Child Health Matter?" *Social Science Quarterly* 86(S1), December, 1399-1417.

- Ogden, Cynthia L., Margaret D. Carroll, Lester R. Curtin, Margaret A. McDowell, Carolyn J. Tabak, and Katherine M. Flegal. 2006. "Prevalence of Overweight and Obesity in the United States, 1999-2004" *JAMA* 295(13), April 5, 1549-1555.
- Plankey, Michael W., June Stevens, Katherine M. Flegal, and Philip F. Rust. 1997. "Prediction Equations Do Not Eliminate Systematic Error in Self-Reported Body Mass Index" *Obesity Research* 5(4), July, 308-14.
- Powers, Elizabeth T. 2003. "Children's Health and Maternal Work Activity: Estimates Under Alternative Disability Definitions" *Journal of Human Resources* 38(3), Summer, 522-556.
- Ruhm, Christopher J. 2005. "Healthy Living in Hard Times" *Journal of Health Economics* 24(2), March 341-63.
- Ruhm, Christopher J. 2007. "Prevalence and Trends of Extreme Obesity Among US Children, Adolescents and Adults, 1960-2004" mimeo, University of North Carolina at Greensboro, January.
- Smith, James P. 2004. "Unraveling the SES-Health Connection" in Waite, Linda J. (ed.). *Aging, Health, and the Public Policy: Demographic and Economic Perspectives*, Supplement to *Population and Development Studies* 30, 108-132.
- Smith, Patricia K., Barry Bogin, and David Bishai. 2005. "Are Time Preference and Body Mass Index Associated? Evidence from the National Longitudinal Survey of Youth" *Economics and Human Biology* 3(2), May, 259-270.
- Sönmez, K., M. Akcakoyun, A. Akcay, D. Demir, N. E. Duran, M. Gencbay et al. 2003. "Which Method Should be Used to Determine the Obesity, in Patients with Coronary Artery Disease? (Body Mass Index, Waist Circumference, or Waist-Hip Ratio)" *International Journal of Obesity* 27(3), March, 341-346.
- Spencer, Elizabeth A., Paul N. Appleby, Gwyneth K. Davey, and Timothy J. Key. 2002. "Validity of Self-Reported Height and Weight in 4808 EPIC-Oxford Participants" *Public Health Nutrition* 54(4), August 2002, 561-5.
- StataCorp. 2005. *Stata User's Guide*. College Station, TX: STATA Press.
- Stockwell, Edward D., Franklin W. Goza, and Kelly S. Belistreri. 2005. "Infant Mortality and Socioeconomic Status: New Bottle, Same Old Wine" *Population Research and Policy Review* 24(4), August, 387-399.
- Strauss, R. S. 1999. "Comparison of Measured and Self-Reported Weight and Height in a Cross-Sectional Sample of Young Adolescents" *International Journal of Obesity* 23(8), August, 904-908.
- Quesenberry, Charles P., Bette Caan, and Alice Jacobson. 1998. "Obesity, Health Services Use, and Health Care Costs Among Members of a Health Maintenance Organization" *Archives of Internal Medicine* 158(5), March 9, 466-472.
- Whitaker, Robert C., Jeffrey A. Wright, Margaret S. Pepe, Kristy D. Seidel, and William H. Dietz. 1997. "Predicting Obesity in Young Adulthood from Childhood and Parental Obesity" *New England Journal of Medicine* 337(13), September 25, 869-873.

World Health Organization. 1997. *Obesity: Preventing and managing the global epidemic. report of a WHO consultation on obesity*. Geneva, Switzerland: World Health Organization.

Zhang, Qi, and Youfa Wang. 2004. "Trends in the Association between Obesity and Socioeconomic Status in U.S. Adults: 1971 to 2000" *Obesity Research* 12(10), October, 1622-1632.

Table 1: Descriptive Statistics by Gender and Socioeconomic Status

Variable	Full Sample	Gender		SES		
		Male	Female	Low	Medium	High
BMI	25.2 (0.1)	26.0 (0.1)	24.3 (0.1)	25.8 (0.1)	25.1 (0.1)	24.4 (0.2)
Obese	14.5% (0.5%)	15.4% (0.8%)	13.4% (0.6%)	18.4% (0.9%)	14.2% (0.7%)	10.0% (0.9%)
Age (Years)	31.2 (0.0)	31.0 (0.1)	31.5 (0.1)	31.3 (0.1)	31.2 (0.1)	31.2 (0.1)
Male	53.1% (0.8%)	--	--	48.8% (1.4%)	55.2% (1.3%)	53.6% (1.7%)
Black	13.1% (1.6%)	12.8% (1.6%)	13.4% (1.6%)	22.2% (2.7%)	9.1% (1.3%)	8.0% (1.4%)
Hispanic	5.3% (0.8%)	5.3% (0.8%)	5.2% (0.8%)	12.0% (1.8%)	2.3% (0.4%)	2.3% (0.5%)
Married	52.7% (0.8%)	52.2% (0.9%)	53.3% (1.0%)	50.8% (1.2%)	54.4% (0.9%)	52.3% (1.2%)
Number of Children	1.14 (0.02)	1.03 (0.02)	1.26 (0.03)	1.38 (0.03)	1.08 (0.03)	0.90 (0.04)
Education of Respondent (Years)	13.2 (0.1)	13.2 (0.1)	13.3 (0.1)	12.1 (0.1)	13.3 (0.1)	14.8 (0.1)
Family Income (\$1,000s)	64.8 (1.5)	66.2 (1.9)	63.1 (1.6)	48.4 (1.4)	65.6 (2.0)	87.6 (3.4)
Education of Mother (Years)	11.8 (0.1)	11.9 (0.1)	11.7 (0.1)	8.8 (0.1)	12.0 (0.0)	15.1 (0.1)
Ever Smoked (≥ 100 cigarettes)	45.8% (0.9%)	45.0% (1.2%)	46.6% (1.2%)	51.8% (1.6%)	45.1% (1.3%)	37.0% (1.6%)
Current Smoker	32.2% (0.7%)	31.8% (1.1%)	32.5% (1.0%)	38.7% (1.4%)	31.4% (1.0%)	23.4% (1.3%)
Heavy Smoker (≥ 20 /day)	17.6% (0.7%)	19.0% (1.0%)	16.0% (0.8%)	22.1% (1.3%)	17.3% (0.9%)	11.6% (1.3%)
Binge Drinker in Last Month	34.1% (0.9%)	44.9% (1.0%)	21.9% (0.9%)	32.3% (1.2%)	35.7% (1.3%)	32.5% (1.4%)
Heavy Drinker (≥ 60 last month)	9.2% (0.4%)	13.6% (0.6%)	4.1% (0.3%)	7.8% (0.5%)	9.9% (0.6%)	9.0% (0.7%)
Physically Inactive	12.3% (0.7%)	10.1% (0.8%)	14.8% (0.9%)	16.6% (1.3%)	10.4% (0.9%)	9.8% (1.1%)
Moderately Active	12.8% (0.6%)	10.9% (0.7%)	15.0% (0.9%)	12.4% (1.0%)	13.0% (0.9%)	13.9% (1.5%)
Physically Active	29.9% (0.7%)	26.6% (1.1%)	33.6% (1.1%)	30.5% (1.3%)	30.7% (1.1%)	27.3% (1.7%)
Vigorous Physical Activity	45.1% (0.9%)	52.5% (1.3%)	36.7% (1.2%)	40.6% (1.6%)	46.0% (1.3%)	49.2% (2.1%)
Strengthening Exercises	38.1% (0.7%)	40.0% (1.1%)	35.9% (1.1%)	31.2% (1.3%)	39.3% (1.2%)	45.3% (1.5%)
Sample Size	70,908	37,470	33,438	27,095	28,260	11,725

Note: Table displays descriptive statistics from the National Longitudinal Survey of Youth for those years where information on body weight was obtained (1981, 1982, 1985, 1986, 1988, 1989, 1990, 1992, 1993, 1994, 1996, 1998, 2000, 2002 and 2004). Standard errors, corrected for population weights and complex survey design, are displayed in parentheses. Body Mass Index (BMI) is obtained from self-reported information on weight and height. Obesity is defined as $BMI \geq 30$. Low, middle and high SES refer to respondents whose mothers have completed <12, 12 or >12 years of education. Family income is measured in 2004-year dollars. Data on alcohol consumption is from 1982-1984, 1988, 1989, 1994 and 2002; that on smoking is from 1984, 1992, 1994 and 1998. These health behaviors refer to either the first or the most recent previous interview for which the information was obtained. Binge drinking refers to consuming six or more drinks in a single session and heavy drinking to consumption of more than 60 drinks per month. Data on leisure-time physical activity is from 2002 and 2004 and refers to the month prior to the survey. Respondents are defined as physically inactive if they exercise less than one hour per week and moderately active if they did so 1 to 2 hours weekly. Vigorous exercisers engage in more than 2 hours of vigorous physical activity per week and those who are physically active participate in more than two hours per week of all types of exercise but less than 2 hours of vigorous activities. Strengthening exercise indicates participation in these activities at least once per week.

Table 2: Body Mass Index and Obesity By Survey Year and Sample Characteristics

Group	Body Mass Index (BMI)		Obese (BMI \geq 30)		Class 3 Obesity ((BMI \geq 40)	
	1981	2004	1981	2004	1981	2004
Full Sample	22.3 (0.1)	27.4 (0.1)	3.0% (0.3%)	26.5% (0.8%)	0.05% (0.03%)	2.53% (0.25%)
Males	23.2 (0.1)	28.2 (0.1)	3.5% (0.4%)	28.8% (1.1%)	0.04% (0.04%)	2.00% (0.04%)
Females	21.4 (0.1)	26.6 (0.1)	2.4% (0.4%)	24.1% (1.0%)	0.06% (0.04%)	3.05% (0.39%)
Low SES	22.7 (0.1)	28.1 (0.2)	3.4% (0.5%)	32.6% (1.3%)	0.14% (0.09%)	2.95% (0.43%)
Medium SES	22.3 (0.1)	27.4 (0.1)	3.3% (0.4%)	25.5% (1.1%)	0.00% (0.00%)	2.59% (0.40%)
High SES	22.0 (0.1)	26.5 (0.2)	1.5% (0.5%)	20.2% (1.7%)	0.00% (0.00%)	1.80% (0.47%)

Note: See note on Table 1. Information is from the 1981 and 2004 years of the National Longitudinal Survey of Youth (NLSY). Samples sizes in 1981 are 4,628, 2,498, 2,130, 1,716, 1,861 and 806 for the full sample, males, females, low, medium and high SES respondents. Corresponding sample sizes are 5,022, 2,498, 2,524, 1,929, 1,997 and 824 in 2004. The sample sizes differ across time periods because females who are pregnant or have given birth in the last year are excluded.

Table 3: Econometric Estimates of Age and SES Gradients in BMI and Obesity

Regressor	(1a)	(1b)	Regressor	(2a)	(2b)	(3a)	(3b)	(4a)	(4b)
Body Mass Index									
Age	0.1227 (0.0285)	0.1176 (0.0288)	Age	0.1225 (0.0286)	0.1221 (0.0285)	0.1233 (0.0286)	0.1231 (0.0285)	0.1274 (0.0313)	0.1273 (0.0312)
Low SES	0.7370 (0.1685)	0.7356 (0.1684)	SES	-0.2017 (0.0242)	-0.2009 (0.0241)	-0.2549 (0.0291)	-0.2544 (0.0291)	-0.2145 (0.0251)	-0.2143 (0.0251)
Age x Low SES		0.0268 (0.0094)	Age x SES		-0.0074 (0.0015)		-0.0090 (0.0018)		-0.0073 (0.0021)
High SES	-0.6514 (0.1981)	-0.6526 (0.1985)							
Age x High SES		-0.0131 (0.0120)							
Obesity Prevalence									
Age	0.0060 (0.0019)	0.0055 (0.0019)	Age	0.0060 (0.0019)	0.0059 (0.0019)	0.0060 (0.0019)	0.0060 (0.0019)	0.0081 (0.0022)	0.0081 (0.0021)
Low SES	0.0432 (0.0114)	0.0430 (0.0114)	SES	-0.0120 (0.0015)	-0.0120 (0.0014)	-0.0153 (0.0017)	-0.0152 (0.0017)	-0.0129 (0.0016)	-0.0129 (0.0016)
Age x Low SES		0.0027 (0.008)	Age x SES		-6.9E-4 (1.2E-4)		-8.8E-4 (1.4E-4)		-7.9E-4 (2.0E-4)
High SES	-0.0407 (0.0120)	-0.0408 (0.0121)							
Age x High SES		-0.0014 (0.009)							
Comment	Trichotomous SES			Continuous SES		Winsorized SES		24-38 Year Olds	

Note: See note on Table 1. Linear probability models are used to estimate the relationship between age, socioeconomic status and their interaction on obesity. Sample weights are incorporated in the estimates and standard errors, reported in parentheses, are adjusted to account for complex survey design. Model (1) uses a trichotomous variable where low, medium and high SES refer to <12, 12, and >12 years of schooling completed by the respondent's mother. SES is proxied by years of education completed by the respondent's mother in models (2) and (4). This SES variable is winsorized in model (3) by setting years of education equal to 7 (16) for persons reporting that their mothers had less (more) schooling than this amount; these correspond to the 5th and 95th percentiles of the distribution. Age and SES are measured as deviations from the sample means. Model (3) limits the sample to 24-38 year olds. The sample is limited to observations with information available on maternal education. Sample sizes are 67,047 for the full sample and 41,254 for 24-38 year olds. The regressions also include controls for gender and the interview year.

Table 4: Econometric Estimates of Age and SES Gradients by Gender

Regressor	Males		Females	
	(1a)	(1b)	(2a)	(2b)
Body Mass Index				
Age	0.1222 (0.0396)	0.1221 (0.0396)	0.1179 (0.0415)	0.1173 (0.0414)
SES	-0.1215 (0.0279)	-0.1212 (0.0277)	-0.2877 (0.0390)	-0.2865 (0.0399)
Age x SES		-0.0046 (0.0017)		-0.0093 (0.0021)
Obesity Prevalence				
Age	0.0059 (0.0028)	0.0059 (0.0028)	0.0060 (0.0025)	0.0059 (0.0025)
SES	-0.0094 (0.0018)	-0.0093 (0.0018)	-0.0149 (0.0022)	-0.0148 (0.0022)
Age x SES		-4.9E-4 (1.5E-4)		-8.8E-4 (1.6E-4)

Note: See notes on Tables 1 and 3. Models correspond to those in columns (2a) and (2b) of Table 3. Sample sizes are 35,228 for males and 31,819 for females.

Table 5: Predicted BMI and Obesity Prevalence by Age and SES

Maternal Years of Schooling	BMI		Obesity Prevalence	
	20 Years Old	40 Years Old	20 Years Old	40 Years Old
	Full Sample			
9	24.1	27.0	9.0%	24.7%
16	23.3	25.1	6.0%	12.1%
	Males			
9	24.9	27.6	10.2%	24.7%
16	24.4	26.4	7.4%	15.1%
	Females			
9	23.4	26.2	7.7%	24.3%
16	22.1	23.7	4.4	8.6

Note: See notes on Tables 1 and 4. Predictions are obtained from models corresponding to (2a) and (2b) of Table 3.

Table 6: Mechanisms and Correlates of SES and Age Gradients in BMI and Obesity Prevalence

Regressor	Body Mass Index					Obesity Prevalence				
	(1)	(2)	(3)	(4)	(5)	(1)	(2)	(3)	(4)	(5)
Full Sample										
Age	0.1225 (0.0286)	0.1339 (0.0285)	0.1209 (0.0288)	0.1319 (0.0286)	0.1354 (0.0289)	0.0060 (0.0019)	0.0069 (0.0019)	0.0059 (0.0019)	0.0068 (0.0019)	0.0071 (0.0020)
SES	-0.2017 (0.0242)	-0.1499 (0.0256)	-0.1686 (0.0256)	-0.1192 (0.0273)	-0.1130 (0.0263)	-0.0120 (0.0015)	-0.0077 (0.0016)	-0.0103 (0.0016)	-0.0060 (0.0017)	-0.0054 (0.0017)
Males										
Age	0.1222 (0.0396)	0.1303 (0.0392)	0.1226 (0.0396)	0.1301 (0.0393)	0.1292 (0.0400)	0.0059 (0.0028)	0.0068 (0.0029)	0.0059 (0.0029)	0.0068 (0.0029)	0.0066 (0.0030)
SES	-0.1215 (0.0279)	-0.0845 (0.0319)	-0.1048 (0.0301)	-0.0668 (0.0328)	-0.0627 (0.0323)	-0.0094 (0.0018)	-0.0050 (0.0021)	-0.0080 (0.0020)	-0.0036 (0.0022)	-0.0030 (0.0022)
Females										
Age	0.1179 (0.0415)	0.1133 (0.0418)	0.1158 (0.0415)	0.1305 (0.0418)	0.1509 (0.0428)	0.0060 (0.0025)	0.0070 (0.0025)	0.0058 (0.0025)	0.0068 (0.0025)	0.0083 (0.0026)
SES	-0.2877 (0.0390)	-0.2174 (0.0423)	-0.2344 (0.0417)	-0.1678 (0.0457)	-0.1528 (0.0440)	-0.0149 (0.0022)	-0.0105 (0.0024)	-0.0126 (0.0024)	-0.0084 (0.0026)	-0.0075 (0.0025)
Education	No	Yes	No	Yes	Yes	No	Yes	No	Yes	Yes
Race	No	No	Yes	Yes	Yes	No	No	Yes	Yes	Yes
Other	No	No	No	No	Yes	No	No	No	No	Yes

Note: See notes on Tables 1 through 4. Regressions correspond to model (2a) of Table 3, with the inclusion of additional covariates detailed at the bottom of the table. Education indicates the highest grade completed by the respondent at the survey date. “Race” refers to dummy variables for (non-Hispanic) blacks and Hispanics. “Other” regressors include controls for family income, household

composition (marital status and number of children), and health behaviors related to smoking, drinking and physical activity. See Table 1 and the text for additional details on these variables.

Table 7: Decomposition of SES Gap in BMI and Obesity and 20 and 40 Years of Age

Explanatory Variables	Full Sample		Males		Females	
	BMI	Obesity	BMI	Obesity	BMI	Obesity
20-year olds						
SES Gap	0.825	0.0296	0.497	0.0275	1.258	0.0329
% of Gap Explained by:						
Education	16.2%	68.6%	-8.8%	76.1%	26.9%	61.4%
Race	12.9%	12.6%	9.6%	12.4%	16.5%	16.1%
Education and Race	28.0%	80.0%	2.7%	89.4%	41.7%	75.4%
All Controls	33.5%	98.5%	-7.0%	91.5%	58.0%	112.4%
40-year olds						
SES Gap	1.862	0.1261	1.136	0.0962	2.563	0.1567
% of Gap Explained by:						
Education	27.9%	29.3%	43.2%	38.6%	22.9%	23.8%
Race	17.9%	15.4%	15.7%	15.7%	19.3%	15.4%
Education and Race	44.6%	43.6%	59.4%	54.5%	41.0%	38.0%
All Controls	46.9%	46.2%	67.8%	61.4%	42.5%	39.5%

Note: The SES gap is the predicted difference for respondents whose mothers had 9 and 16 years of schooling respectively. These estimates are obtained from regressions corresponding to model (2b) in Table 3. The percentages of the gap explained are obtained from models that add covariates for race and the respondent's level of schooling at the survey date, as specified, as well as interactions between (mean deviations of) these variables and age. The "all controls" models also include covariates for family income, household composition and health behaviors.

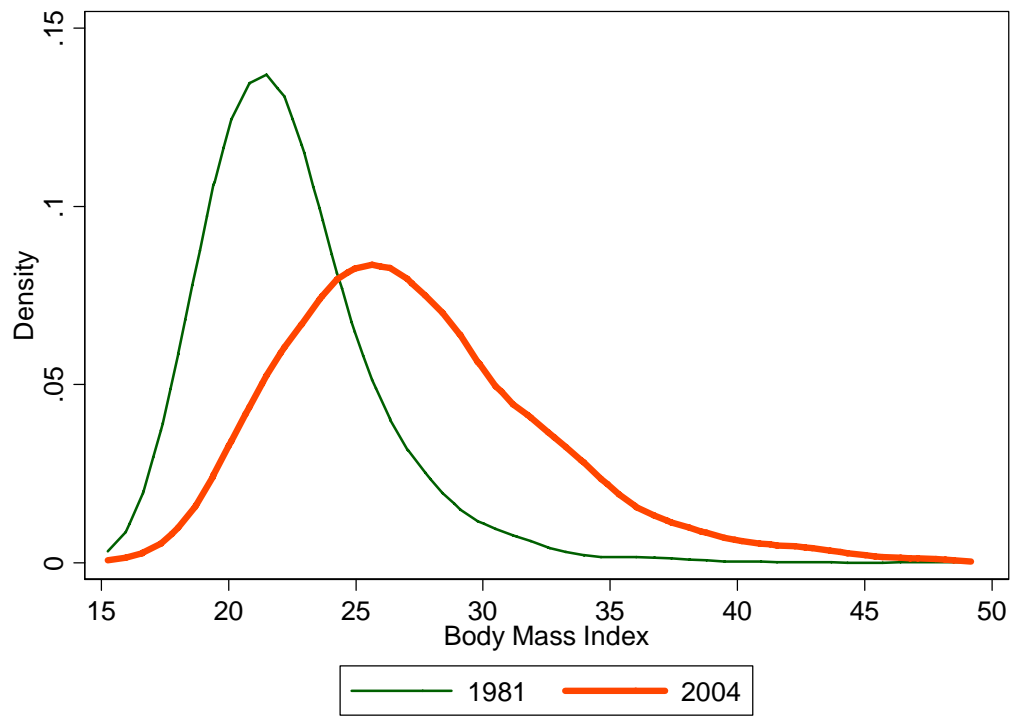


Figure 1: Body Mass Index in 1981 and 2004

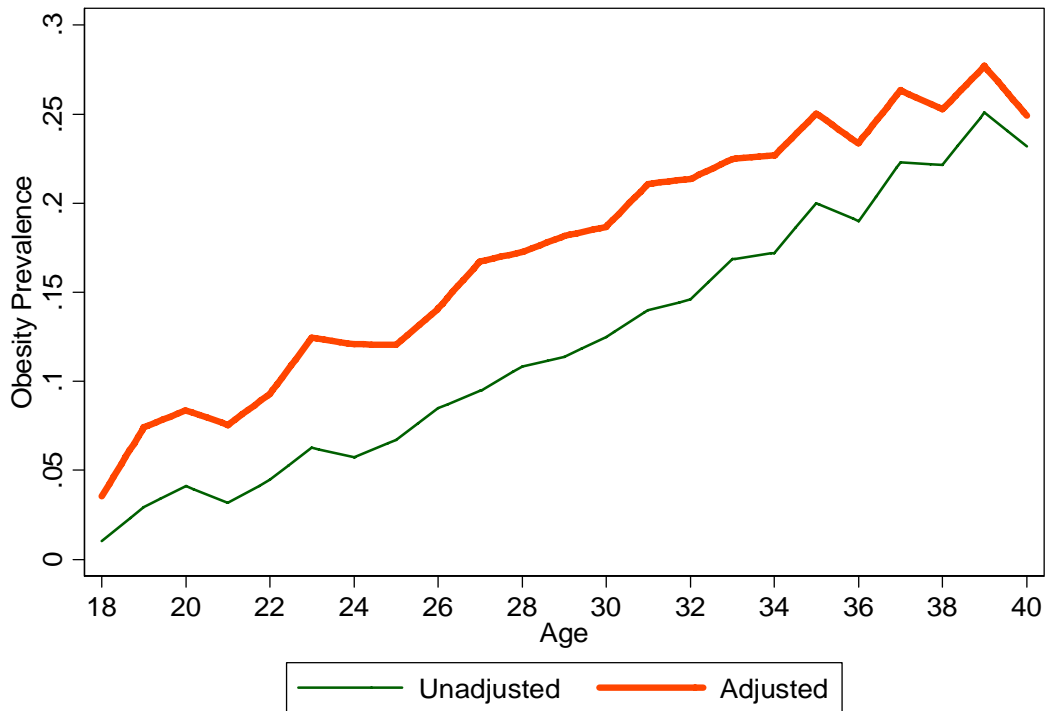
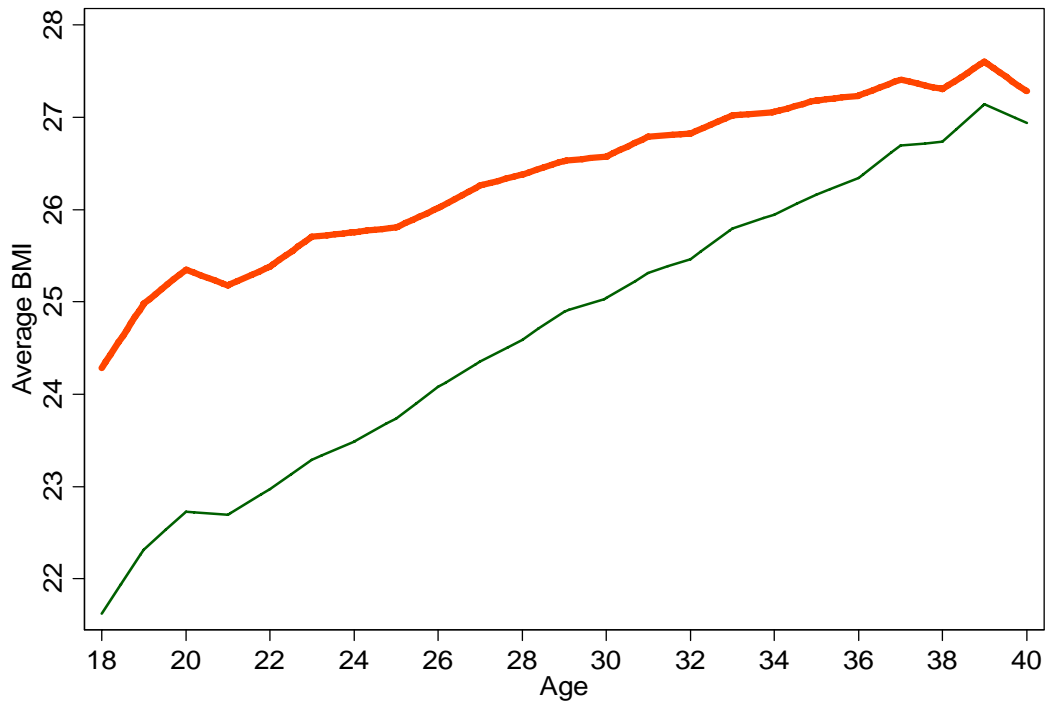


Figure 2: Average BMI and Obesity Prevalence by Age, With and Without Adjustment for Secular Trends

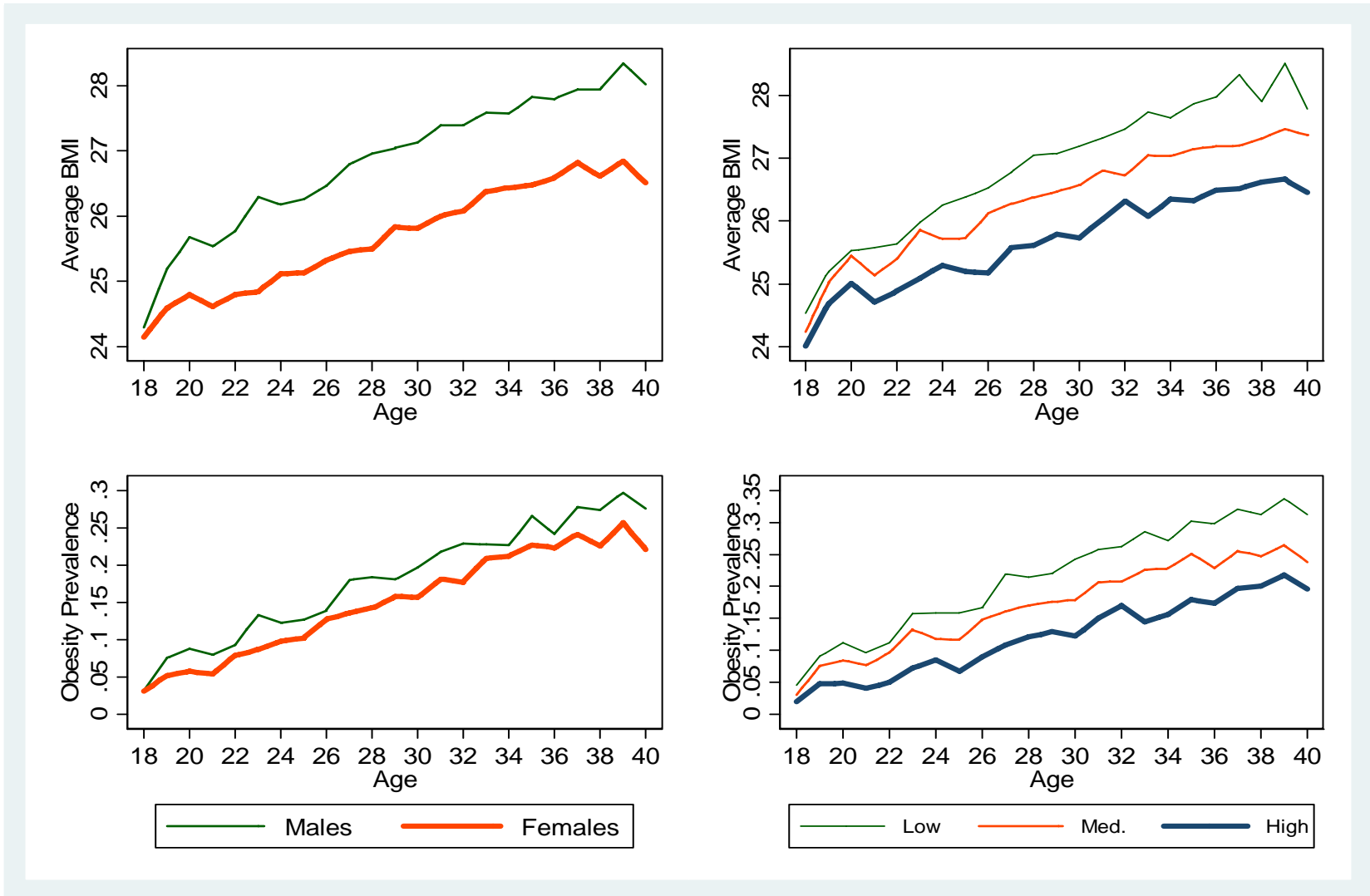


Figure 3: Age-Specific Average BMI and Obesity Prevalence By Gender and SES, Adjusted for Secular Trends

Appendix Tables

Table A.1: Descriptive Statistics on BMI for Balanced and Unbalanced Samples

Year	Balanced Panel			Unbalanced Panel		
	Sample Size	Mean	Standard Error	Sample Size	Mean	Standard Error
1981	4,628	22.344	0.065	8,578	22.508	0.056
1982	4,582	22.604	0.069	8,452	22.769	0.057
1985	4,545	23.401	0.079	8,180	23.558	0.067
1986	4,580	23.797	0.086	7,899	23.951	0.067
1988	4,564	24.340	0.088	7,714	24.537	0.072
1989	4,590	24.493	0.086	7,819	24.691	0.073
1990	4,609	24.865	0.084	7,713	25.068	0.073
1992	4,667	25.328	0.094	7,748	25.585	0.082
1993	4,708	25.532	0.092	7,783	25.738	0.080
1994	4,767	25.754	0.091	7,752	25.969	0.081
1996	4,823	26.240	0.095	7,622	26.395	0.084
1998	4,878	26.657	0.098	7,428	26.761	0.088
2000	4,949	27.009	0.100	7,226	27.219	0.089
2002	4,996	27.289	0.099	7,059	27.515	0.098
2004	5,022	27.387	0.100	7,062	27.679	0.094

Note: Balanced sample excludes sample members with missing data on body weight during at least one survey year. The unbalanced sample includes these individuals. The size of the balanced sample varies over time because women are excluded in years they are pregnant or have recently given birth.

Table A.2: Age-Specific Body Mass Index and Obesity Prevalence With and Without Adjustment for Secular Trends in Body Weight

Age	Full Sample: Not Adjusted	Adjusted for Secular Trends in Body Weight					
		Full Sample	Gender		SES		
			Males	Females	Low	Medium	High
Body Mass Index							
20	22.7 (0.1)	25.3 (0.1)	25.7 (0.1)	24.8 (0.2)	25.5 (0.2)	25.4 (0.1)	25.0 (0.3)
25	23.7 (0.1)	25.8 (0.1)	26.3 (0.1)	25.1 (0.1)	26.4 (0.2)	25.7 (0.2)	25.2 (0.2)
30	25.0 (0.1)	26.6 (0.1)	27.1 (0.1)	25.8 (0.1)	27.2 (0.2)	26.6 (0.1)	25.7 (0.2)
35	26.2 (0.1)	27.2 (0.1)	27.8 (0.1)	26.5 (0.2)	27.9 (0.2)	27.1 (0.2)	26.3 (0.3)
40	26.9 (0.1)	27.3 (0.1)	28.0 (0.1)	26.5 (0.2)	27.8 (0.2)	27.4 (0.2)	26.5 (0.3)
Obesity Prevalence							
20	4.1% (0.6%)	8.4% (0.8%)	8.8% (1.1%)	5.8% (1.2%)	11.1% (1.7%)	8.4% (1.2%)	4.9% (1.6%)
25	6.7% (0.7%)	12.0% (0.8%)	12.7% (1.2%)	10.2% (1.0%)	15.8% (1.4%)	11.6% (1.3%)	6.7% (1.4%)
30	12.5% (0.7%)	18.7% (0.9%)	19.7% (1.3%)	15.7% (1.1%)	24.2% (1.7%)	17.9% (1.1%)	12.2% (1.6%)
35	20.0% (0.9%)	25.0% (1.0%)	26.6% (1.5%)	22.7% (1.3%)	30.2% (1.8%)	25.1% (1.4%)	17.9% (2.2%)
40	23.2% (1.0%)	24.9% (0.9%)	27.5% (1.5%)	22.1% (1.3%)	31.3% (1.8%)	23.8% (1.5%)	19.6% (2.2%)

Note: See notes on Tables 1 and 2. Adjusted BMI and obesity is calculated by using data from the National Health and Nutrition Examination Surveys to remove the effects of secular trends. Details of the procedure are discussed in the text. Low, medium and high socioeconomic status refer to respondent's whose mothers completed <12, 12 and >12 years of education.

Table A.3: Additional Econometric Estimates of Mechanisms and Correlates of SES and Age Gradients

Regressor	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Body Mass Index							
Age	0.1225 (0.0286)	0.1339 (0.0285)	0.1209 (0.0288)	0.1319 (0.0286)	0.1253 (0.0285)	0.1167 (0.0295)	0.1354 (0.0289)
SES	-0.2017 (0.0242)	-0.1499 (0.0256)	-0.1686 (0.0256)	-0.1192 (0.0273)	-0.1936 (0.0241)	-0.2005 (0.0245)	-0.1130 (0.0263)
Education		-0.1464 (0.0343)		-0.1390 (0.0340)			-0.1783 (0.0356)
Hispanic			0.6422 (0.1853)	0.6818 (0.1837)			0.6149 (0.1839)
Black			1.1463 (0.1488)	1.0995 (0.1480)			1.1762 (0.1523)
Income					-0.0017 (.0004)		-0.0012 (.0004)
Obesity Prevalence							
Age	0.0060 (0.0019)	0.0069 (0.0019)	0.0059 (0.0019)	0.0068 (0.0019)	0.0061 (0.0019)	0.0057 (0.0020)	0.0071 (0.0020)
SES	-0.0120 (0.0015)	-0.0077 (0.0016)	-0.0103 (0.0016)	-0.0060 (0.0017)	-0.0114 (0.0015)	-0.0115 (0.0014)	-0.0054 (0.0017)
Education		-0.123 (0.0022)		-0.119 (0.0022)			-0.139 (0.0024)
Hispanic			0.0317 (0.0127)	0.0351 (0.0125)			0.0316 (0.0124)
Black			0.0657 (0.0102)	0.0617 (0.0102)			0.0617 (0.0106)
Income					-1.3E-4 (2.1E-5)		-8.1E-5 (1.9E-5)
Additional Controls	No	No	No	No	No	Yes	Yes

Note: See notes on Table 6. Additional controls here refer to health behaviors and household composition. These estimates are for a combined sample of men and women.