Age, socioeconomic status and obesity growth

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Abstract:
We use panel data from the National Longitudinal Survey of Youth (NLSY) to examine how body weight changes with age for a cohort moving through early adulthood, to investigate how the age-obesity gradient differs with socioeconomic status (SES) and to study channels for these SES disparities. Our results show first that weight increases with age and is inversely related to SES during childhood. Second, the obesity gradient widens over the lifecycle, consistent with research on other health outcomes. Third, a substantial portion of the "effect" of early life conditions operates through race/ethnicity and the translation of advantaged family backgrounds during childhood into higher levels of subsequent education. By contrast, little of the SES gap appears to propagate through household composition, family income or health behaviors. Fourth, adult SES has independent effects after controlling for childhood status.
Keywords: Obesity, Body weight, Age, Socioeconomic status

Article:
Although 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 under-stood. Medical researchers and epidemiologists tend to emphasize the causal effects of SES, whereas economists frequently focus on how health influences SES or on potential confounding factors (like discount rates or genetics). In an effort to identify the impact of SES, some analysts have recently focused on health disparities early in life and on the evolution of these gradients as age increases. A primary advantage 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. These investigations suggest that SES-health gradients become more pronounced with age, through at least early adulthood. Socioeconomic status during childhood may also be of interest to the extent that it affects adult outcomes independently of any relationship it might have with SES later in life.[2]

This analysis uses data from the National Longitudinal Survey of Youth (NLSY) to investigate how body weight and obesity change with age for a cohort moving through middle adulthood, SES differences in this age-obesity gradient, and channels for the SES disparities. 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, stresses the health care system and decreases productivity (Quesenberry et al., 1998; Finkelstein et al., 2003; Andreyeva et al., 2004). Second, changes in weight are easily observable, whereas health indicators such as overall status or specific morbidities are likely to be measured with greater error or require interaction with the medical system for diagnosis.[3] Third, obesity represents a rapidly increasing health risk. Using conventional definitions, 32% of 20–74 years old were obese in 1999–2004, compared to just 14% in 1976–1980 (Ruhm, 2007). Fourth, obesity generally develops over a lengthy period – 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. Consistent with this, excess weight during childhood and particularly late adolescence is a strong predictor of adult obesity (Whitaker et al., 1997; Guo et al., 2002; McTigue et al., 2002).

Our results show first that weight increases with age and is inversely related to SES. Second, the obesity gradient widens over the lifecycle, consistent with research on other health outcomes. Third, a substantial portion of the "effect" of early life conditions operates through race/ethnicity and the translation of advantaged family backgrounds during childhood into higher levels of subsequent education. By contrast, little of the SES gap appears to propagate through household composition, family income or the health behaviors accounted for. Fourth, adult SES has independent effects, after controlling for childhood status.

Previous research has used cross-sectional data to document an inverse association between current SES and weight that strengthens through at least middle-age. However, since such data provide only contemporaneous information, it is not possible to study how socioeconomic status earlier in life is related to adult obesity. Conversely, the longitudinal data and rich information on family background characteristics in the NLSY permit us to investigate whether adult body weight is independently affected by current and previous SES and the extent to which economic conditions early in life operate through the "stickiness" of SES over time.

1. Socioeconomic status and obesity

Adult body weight and obesity are negatively related to social and economic advantage for most groups. For instance, using self reported data, 26% of high school dropouts were obese in 2000 versus 22% of high school graduates and 15% of college graduates (Mokdad et al., 2001). Similarly, 23 (14)% of white women (men) with family incomes greater than 400% of the poverty line were obese in 1999–2002 compared to 40 (34)% of their poor counterparts, although this distinction is not always present for nonwhites and may have weakened over time (Chang and Lauderdale, 2005). Using clinical measures of height and weight (which result in higher prevalence estimates), 31% of non-Hispanic whites aged 20 and older were obese in 2003–2004, versus 37% of Hispanics and 45% of non-Hispanic blacks (Ogden et al., 2006).

These patterns are consistent with high SES adults being healthier than their less advantaged peers. 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; similarly, the "weathering" hypothesis (Geronimus et al., 2006) emphasizes the cumulative impact of social or economic adversity and political marginalization. On the other hand, economists show that obese women receive a wage penalty (Averett and Korenman, 1996; Baum and Ford, 2004; Cawley, 2004), suggesting that excess weight reduces income-based measures of SES. Finally, unobserved factors could determine both SES and body weight. For instance, high discount rates may simultaneously reduce educational investments and the willingness to forgo current caloric intake for the future benefit of lower weight (Fuchs, 2004; Smith et al., 2005; Borghans and Golsteyn, 2006). In addition, self-control problems could be more common among low SES individuals (Cutler et al., 2003).

Some investigators have focused on SES-health gradients among youths as a promising method of identifying the causal effects of life circumstances. Examining youths has two advantages. First, it seems unlikely that health could significantly affect child SES, since the latter is largely determined by the education and economic situation of the parents.[4] Second, omitted factors transmitted across generations (such as genetics) could play a role, but 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) between infancy and late adolescence. In subsequent work, Case et al. (2005) show that poor health during childhood is associated with reduced educational attainment, lower social status and more health problems in adulthood, suggesting that health is an important mechanism through which economic status is transmitted.[5] We do not fully understand why the SES gradient rotates. Janet Currie and co-authors indicate that it is primarily because disadvantaged individuals are subjected to a greater number of deleterious health events rather than being more adversely affected by given shocks (Currie and Hyson, 1999; Currie and Stabile, 2003). Case et al. (2002) suggest that health behaviors may play an important role. Body weight and obesity are useful outcomes for understanding age-related changes in SES-health disparities, but they have not been directly examined in this context.

A vast literature investigates how adult socioeconomic status is related to obesity (Sobal and Stunkard, 1989; McLaren, 2007) but most analyses use cross-sectional data and so are unable to distinguish between age and period effects or identify the potentially distinct effects of contemporaneous and childhood SES; they also cannot account for differential selection due to the higher mortality rates of obese individuals.[6] There is evidence of a weaker relationship between SES and body weight for children than adults (Parsons et al.,1999), suggesting that age-related weight gains may be larger for disadvantaged individuals. However, the sources and timing of these differences remain poorly understood. McTigue et al. (2002) did use NLSY data for 1981 through 1998 – the same source we use but for a shorter period – to investigate race/ethnicity differences in the evolution of body weight. However, their study did not explicitly examine the role of SES, contained an extremely restricted set of controls and had other limitations that our research attempts to rectify.[7]

2. The production of body weight

The body weight (W) of individual i increases between periods t and $t-1$ if energy intake (N) exceeds energy expenditure. Dividing the latter into basal metabolism (B) and calories consumed during physical activity (P), this can be expressed as[8]

$$\Delta W_{it} = W_{it} - W_{it-1} = N_{it} - B_{it} - P_{it} . \qquad (1)$$

Basal metabolism depends on body weight and individual specific components (e.g. genetics) according to

$$B_{it} = a_i + bW_{it} , \qquad (2)$$

where b is positive because increased weight raises the amount of energy required to sustain bodily functions. For simplicity, we assume that a is time-invariant.

Substituting Eqs. (2) into (1) and using subscripts to indicate partial derivatives:

$$\Delta W_{it} = N_{it} - a_i - bW_{it} - P_{it} = f(N_{it}, a_i, P_{it}), \qquad (3)$$

where $f_N > 0$ and $f_a, f_P < 0$.

Equation (3) shows that, *ceteris paribus*, individuals are more likely to gain weight if they have a high energy intake and low basal metabolism or physical activity.[9]

An individual's weight is in steady-state if $Wit = Wit-1$. This occurs when:

$$W^*_i = \frac{N_{it} - a_i - P_{it}}{} = g(N_{it} , a_i , P_{it}) \qquad (4)$$

where $g_N > 0$ and $g_a, g_P < 0$.Steady-state weight is therefore determined by the same factors (N, a, and P) operating in the same direction as weight gain.

Equations (3) and (4) highlight possible pathways for an inverse relationship between weight and SES. Specifically, disadvantaged individuals will weigh more than their counterparts if they consume a greater number of calories, are less active, or have lower baseline metabolisms. Assuming their weight has not reached a steady-state, these same conditions are likely to lead to a faster rate of weight gain, implying rotation of the SES gradient with age.

This framework is useful for considering how adult weight is related to both early life conditions and current socioeconomic status. For instance, low childhood SES may raise weight by influencing patterns of energy intake and expenditure later in life, independent of adult status (e.g. if eating habits are determined at a young age). Analyses of cross-sectional data containing information on current but not previous status would then miss

important sources of SES-related disparities. On the other hand, early life conditions might transmit to adult weight entirely because advantaged children become high SES adults.10 Childhood and contemporary socioeconomic status might also have effects that are partially independent or reinforcing.[11]

These possibilities can be empirically tested using the NLSY (but not cross-sectional data) by estimating equations that simultaneously control for socioeconomic status during adolescence and adulthood. A significant coefficient on childhood but not contemporaneous status then suggests that only the former are determinative. The reverse pattern implies either that early conditions are not important or that they operate entirely by affecting adult status.[12] Significant coefficients for both SES variables suggest effects that are at least partially independent.

A final possibility is that the observed SES gradients are spurious, occurring, for example, because disadvantaged individuals happen to have genetic characteristics leading to low basal metabolisms. Although we are not able to fully investigate this scenario, such biological causes probably imply a similar evolution of SES–age gradients across race/ethnicity groups since there is no reason to think that the sources of confounding differ by race/ethnicity. Conversely, large differences cast doubt upon such explanations.

3. Data

Data are from the 1979 cohort of the NLSY, which initially included 12,686 14–21 years old, with oversamples of blacks, His-panics, low-income whites and military personnel. Annual surveys were conducted through 1994, with biennial interviews thereafter. 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.[13] Most of our analysis is performed separately for men and women, and we sometimes stratify the sample by race/ethnicity.

The NLSY collects data on individual and family characteristics at each interview, 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 incorporated in 1981,1982 and 1985 (Centerfor Human Resource Research, 2004).[14] Our analysis assumes that height does not change after 1985, since all NLSY respondents were at least 20 years old at that time. Body Mass Index (BMI) – defined as weight in kilograms divided by height in meters squared – 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. It, nevertheless, is a favored method of assessing excess weight because it is simple, rapid, and inexpensive to calculate.[15]

Our analysis focuses on BMI, obesity and, for some descriptive analysis, class 3 obesity. Adults are obese if their BMI is ≥ 30 and class 3 obese if it is ≥ 40 (World Health Organization, 1997; National Heart, Lung, and Blood Institute, 1998). A more complicated criterion is used for persons under 21 years of age, based on gender and age-specific growth charts compiled by the Center for Disease Control and Prevention (Kuczmarski et al., 2000).[16] We use the adult obesity standard for all respondents, including those under 21 years of age, to provide consistency across individuals and over time. Preliminary analysis confirmed that our results were robust to the use of the CDC classification of overweight for respondents under 21.

Self-reported data on height and weight contain errors: height tends to be over-reported and weight to be understated (Strauss, 1999; Goodman et al., 2000; Kuczmarski et al., 2001). A number of regression-based correction procedures have been proposed (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 regressed clinical measures of weight (height) on a quadratic of the corresponding self-reported variable using National Health and Nutrition Examination Survey (NHANES) data and then used the resulting regression estimates to predict actual weight (height) as a function of the self-reported values in a target data set (such as the NLSY) containing only the latter.[17] We found that our results were not substantively altered by using this procedure. Therefore, most findings below are based on the self-reported data.

To maintain a consistent sample across survey years, our main estimates eliminate persons failing to provide valid body weight information for any of the 15 interviews in which it was collected.18 We examined the implication of these deletions by comparing average BMI for our main analysis sample with an unbalanced sample that included respondents with missing BMI information in one or more year. 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.19 We also replicated some of our analysis using a less restricted sample that included individuals providing valid body weight information in 2004 (the last interview year), even if corresponding data were missing in some earlier survey waves. Results from these estimates were qualitatively similar to those using the balanced sample.

Our primary proxy for childhood SES is the highest grade completed by the respondent's mother, measured at the 1979 interview. We focus on maternal education because previous research suggests it is more directly related to child health than the schooling of fathers (Currie et al., 2007), for whom education data are also more often missing, and because mothers may be more instrumental in establishing the eating habits and health behaviors of children. The descriptive analysis divides the sample into "low" "medium" and "high" SES groups, defined by <12, 12, or >12 years of maternal schooling; 29, 49 and 22% of the NLSY cohort fall into these categories.

Previous research (Zhang and Wang, 2004; Classen and Hokayem, 2005) finds substantial health gradients when proxying SES with schooling, suggesting that our education-based measure is informative. As alternatives, we experimented with indicators based on: (i) the presence or absence of the father in the household when the respondent was 14; (ii) highest grade completed by the respondent's mother *or* father; (iii) highest Duncan Socioeconomic Index occupation score of the respondent's mother or father for the longest job held in 1978; and (iv) respondent scores on the Armed Forces Qualification Test (AFQT), an indicator of cognitive skill measured in 1981.[20] The NLSY does not collect data on permanent income during the respondent's childhood, which otherwise would be a logical alternative SES measure.

Some specifications include controls for adult socioeconomic status, proxied by the respondent's highest grade completed, total family income, or both. When examining SES transmission mechanisms, we also control for race/ethnicity, marital status, number of biological children and health behaviors. Family income refers to all sources during the previous calendar year and is expressed in year-2004 dollars. Marital status takes the value of one if the respondent was married at the survey date and zero if he or she was separated, divorced, widowed or never married.

We use two strategies to avoid losing observations due to missing values of variables other than BMI. First, we replace missing data with averages for adjacent survey years. For instance, if family income is missing in 1985, we use average income in 1984 and 1986. Second, we set values still missing (after using the first procedure) to zero and include a dummy variable denoting that the information is missing.

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, exercise, and job-related physical demands but none on diet or caloric intake. Our strategy for alcohol and cigarette consumption is to use information from the most recent previous survey in which the relevant questions were asked or from the first survey in which it was collected, when this occurred subsequent to the relevant interview date. Using drinking questions included in 1983, 1984, 1988,1989,1994 and 2002, we constructed dummy variables indicating binge consumption (≥ 6 drinks in a single session) and heavy drinking (consumption of >60 alcoholic beverages during the previous month). Information on smoking, available in 1984, 1992, 1994 and 1998, was used to create three dummy variables. The first indicates whether the respondent has smoked 100 or more cigarettes during his or her lifetime. The second and third denote current and heavy smoking, with the latter defined as consumption of 20 or more cigarettes per day.

Data on the frequency and duration of "light/moderate" and "vigorous" physical activity and the frequency of strengthening exercises was provided in 2002 and 2004. Four variables were created using averages over these 2 years. [21] Individuals are "physically inactive" if they engaged in less than 1 h of exercise per week, "moderately active" if they exercised 1–2 h weekly, "physically active" if exercising at least 2 h per week but less than 2 h vigorously, and "vigorous exercisers" if they engaged in more than 2 h per week of vigorous physical activity. An additional activity variable indicates participation in strengthening exercises at least once per week. Following Lakdawalla and Philipson (2007), we also constructed a measure of the number of fitness activities (climbing, reaching, stooping, kneeling, crouching and crawling) required by the individual's occupation and an ordinal strength ranking ranging from one for sedentary occupations to five for those with very heavy strength demands.[22] These behaviors are included to capture the effects of a broad range of lifestyle factors, but we do not apply a causal interpretation to their estimated coefficients.[23]

Table 1 presents descriptive statistics for BMI, obesity and many variables used in our analysis. Sample means are generally similar for men and women, although males are heavier, have higher incomes, greater rates of obesity and fewer children. Men also have more physically demanding jobs and lifestyles that are healthier in some ways (like exercising more) but less so in others (more binge or heavy drinking and heavy smoking). The SES differences are more pronounced. Compared to less advantaged individuals, respondents with highly educated mothers are lighter, 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.

Table 1
Descriptive statistics by gender and socioeconomic status.

Variable	Full sample	Gender		Maternal education		
		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 ($1000s)	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^{-1})	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%)
Job-related fitness demands	1.564 (0.010)	1.689 (0.011)	1.423 (0.014)	1.546 (0.020)	1.585 (0.012)	1.558 (0.016)
Job-related strength demands	0.577 (0.006)	0.648 (0.009)	0.498 (0.008)	0.572 (0.012)	0.578 (0.008)	0.591 (0.014)
Sample size	70,908	37,470	33,438	27,095	28,260	11,725

Note: 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 ≥ 30. Low, middle and high maternal education 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 to 2004 and refers to the month prior to the survey. Respondents are defined as physically inactive if they exercise less than 1 h per week and moderately active if they did so 1–2 h weekly. Vigorous exercisers engage in more than 2 h of vigorous physical activity per week and those who are physically active participate in more than 2 h per week of all types of exercise but less than 2 h of vigorous activities. Strengthening exercise indicates participation in these activities at least once per week. Data on job-related fitness and strength demands are collected from 1981 through 2000; the former refers to the number of fitness activities required by the occupation and the latter is the score, ranging from 1 to 5, on an ordinal index.

Table 2
Body Mass Index and obesity by survey year and sample characteristics.

Group	Body Mass Index (BMI)		Obese (BMI ≥ 30)		Class 3 obesity (BMI ≥ 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 Maternal education	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 maternal education	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 maternal education	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 in Table 1. Information is from the 1981 and 2004 years of the National Longitudinal Survey of Youth (NLSY). Samples sizes in 1981 are 4628, 2498, 2130, 1716, 1861 and 806 for the full sample, males, females and respondents whose mothers have low, medium and high levels of education. Corresponding sample sizes are 5022, 2498, 2524, 1929, 1997 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.

4. Age-related changes in body weight

Fig. 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.24 Kernel density estimates demonstrate that much of the growth in self-reported BMI occurred in the right-tail of the distribution (Fig. 1). This is consistent with evidence obtained using clinical data (Ruhm, 2007) and resulted in faster increases in obesity or severe obesity than in average body weight. Thus, mean self-reported BMI of NLSY respondents rose 23% (from 22.3 to 27.4 kg/m2) between 1981 and 2004, obesity prevalence increased almost 8-fold (from 3.0 to 26.5%), and class 3 obesity increased by even more (from <0.1 to 2.5%).[25]

Average BMI and obesity (but not class 3 obesity) prevalence is higher for men than women, which initially seems surprising since clinical measures from NHANES show the opposite pattern (Flegal et al., 2002; Ogden et al., 2006). We suspect that three factors explain this result, which has also been obtained in previous analyses of the NLSY (e.g. Cawley, 2004). First, self-reported BMI understates true values by larger amounts for women. Second, since clinical data indicate that females are less often overweight but more frequently obese, an equal BMI underestimate for both sexes could cause more obese women to be classified as overweight based on self-reported data.[26] These are not major issues if the reporting errors are similar across time periods and SES groups, but may be more problematic if this is not the case. Third, heavier women are disproportionately likely to have missing weight information in some survey years (and so to be excluded from our balanced sample), whereas this selection is weaker for men.[27]

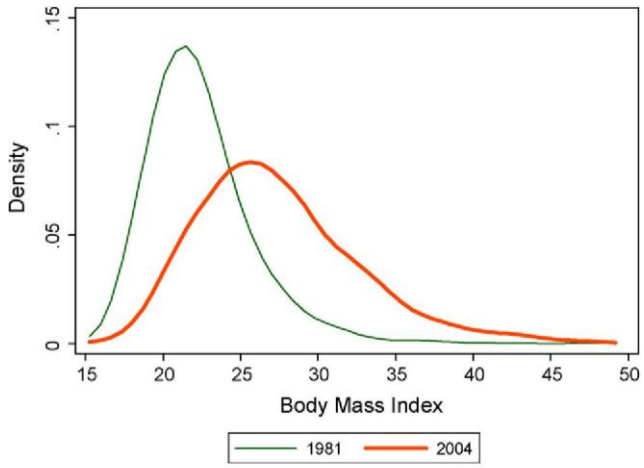

Fig. 1. Body Mass Index in 1981 and 2004.

The last three rows of Table 2 show how the results differ with childhood SES, proxied by maternal education. BMI, obesity and class-3 obesity are more common and increase faster over time for the disadvantaged: average BMI rose 5.4kg/m^2 and obesity prevalence by 29.2 percentage points between 1981 and 2004 for the lowest

SES group, compared with 5.1 kg/m^2 and 22.2 points for the middle category and 4.5 kg/m^2 and 18.7 points for the most advantaged.[28]

Since body weight trended upward throughout the NLSY sample period, the preceding results combine the impacts of aging and secular changes. The following procedure was employed to isolate the effects of aging. NHANES data were first used to calculate growth in the average BMI of 24–38 years old occurring between 1976–1980 and 1999–2004. This was converted to an annualized increase using a linear trend and adjustments to eliminate these effects were made to the BMI of each NLSY respondent.[29] Finally, adjusted BMI values were used to calculate mean BMI and obesity prevalence. These corrections were implemented separately for males and females when examining gender-specific findings.[30]

BMI and obesity prevalence grew rapidly with age. As shown in Fig. 2, average BMI rose from 21.6 to 26.9 kg/m^2 between the ages of 18 and 40, while obesity prevalence increased from 1.0 to 23.2%. Over two-fifths of BMI growth was due to secular trends rather than aging, so that adjusted BMI rose from 24.3 to 27.3 kg/m^2 (see the on-line appendix for details). This correction had less effect on obesity – adjusted prevalence was 3.5% at age 18 and 24.9% for 40 years old – because the procedure did not change the status of individuals with BMI substantially above or below the threshold.

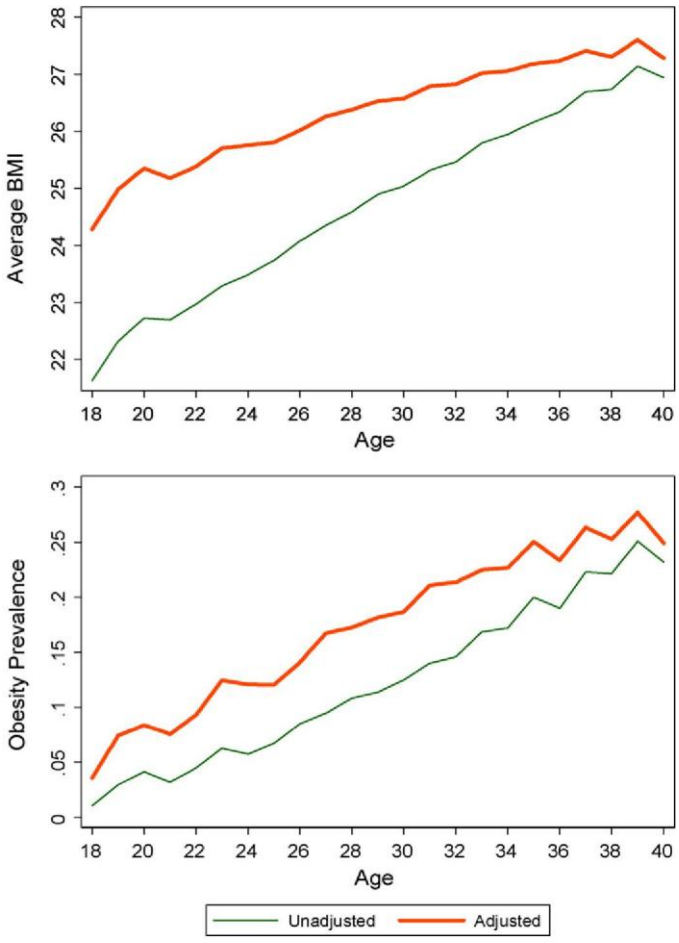

Fig. 2. Average BMI and obesity prevalence by age, with and without adjustment for secular trends.

Fig. 3 displays age-related changes in body weight for gender and maternal education subgroups, all adjusted for secular trends in average BMI. BMI and obesity rise with age for all subsamples. The age-related increase in BMI is somewhat faster for men than women while growth in obesity prevalence is similar. Generally, the evolution of body weight does not vary sharply with gender over the ages studied. Not only do high SES individuals have lower BMI and obesity prevalence but the gradient steepens with age.[31] SES differences are

more pronounced for obesity than BMI, reflecting the importance of differences in the right tail of the distribution. Finally, although the age profiles show weak evidence of concavity, a linear approximation is reasonable for NLSY-aged respondents.

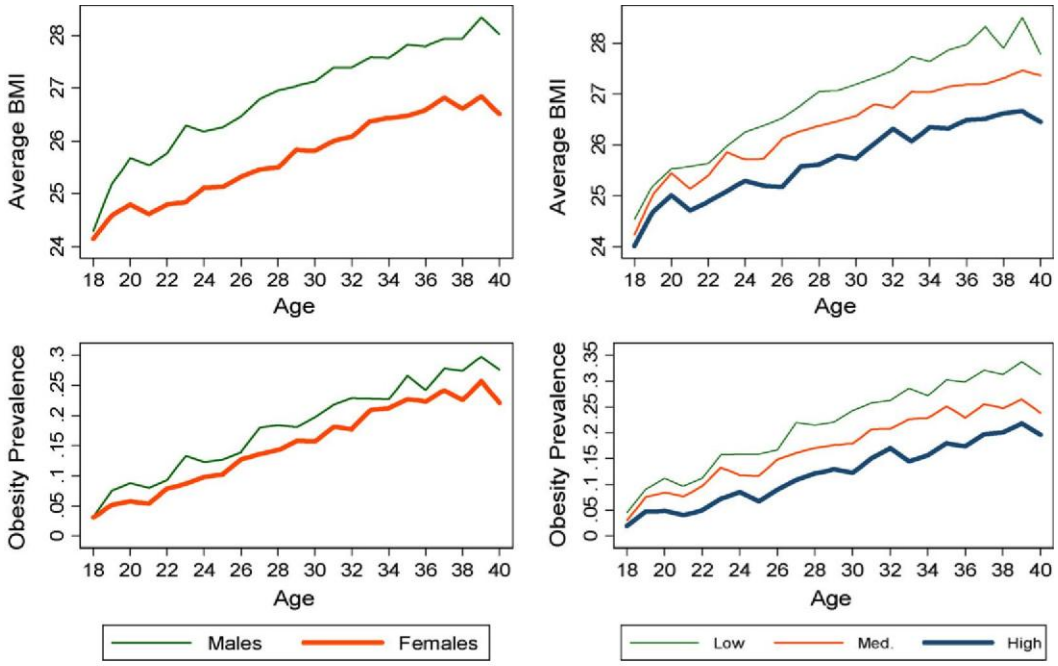

Fig. 3. Age-specific average BMI and obesity prevalence by gender and maternal education, adjusted for secular trends.

5. Empirical methods

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

$$Y_{it} = \alpha_0 + \alpha_1 \mathbf{X}_{it} + \alpha_2 AGE_{it} + \alpha_3 SES_i + \varepsilon_{it}, \qquad (5)$$

where Y_{it} indicates BMI or obesity for individual i at time t, $\boldsymbol{X}$ is a vector of control variables, *AGE* is the respondent's age at the survey date, *SES* is socioeconomic status during childhood, and ε is the error term. The supplementary covariates included vary by model specification. All equations contain survey year dummy variables and, in the full sample estimates, control for gender. We do not initially include regressors for race/ethnicity, respondent education, family income, household composition, or health behaviors because they represent mechanisms through which early life conditions may be transmitted. However, we add them to subsequent models when attempting to understand how childhood SES operates. We also sometimes stratify the sample by sex and race/ethnicity.

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

$$BMI_{it} = \alpha_0 + \alpha_1 \mathbf{X}_{it} + \alpha_2 AGE_{it} + \alpha_3 SES_i + \alpha_4 AGE_{it} \times SES_i + \varepsilon_{it} \quad (6)$$

where *AGE* × *SES* is the interaction between *SES* and age. We also examine whether socioeconomic status early and later in life have independent effects from:

$$BMI_{it} = \alpha_0 + \alpha_1 \mathbf{X}_{it} + \alpha_2 AGE_{it} + \alpha_3 SES_i + \alpha_4 ADULT - SES + \varepsilon_{it}, \quad (7)$$

where *ADULT-SES* refers to socioeconomic status at the survey date.

For ease of interpretation, we generally express age and SES as deviations from the sample averages, so that $\hat{\alpha}_2$, $\hat{\alpha}_3$ and $\hat{\alpha}_4$ indicate marginal effects evaluated at the means of respective variables. Sample weights are incorporated to provide nationally representative estimates and the standard errors account for complex survey design. We use linear probability (LP) models when obesity is the dependent variable. Preliminary analysis revealed similar predicted effects with logit or probit specifications, but the LP coefficients are easier to interpret, especially when including the *AGE–SES* interactions (Ai and Norton, 2003).

6. Age and SES profiles

Initially, we examine age and SES gradients for BMI and obesity with additional covariates limited to gender and the survey year. These results are summarized in Table 3. For each specification, column (b) includes an interaction between age and maternal education, whereas column (a) does not. The first model controls for low and high childhood SES, with the middle category (12 years of maternal education) constituting the reference group. These correspond to the categories used in the descriptive analysis above. We do not examine contemporaneous socioeconomic status until Section 8 and, for ease of notation, refer to childhood status simply as "SES" in this and the next section.

BMI is predicted to increase by 0.12 kg/m^2 and obesity prevalence by 0.60 percentage points per year of age (see column 1 a). Low SES individuals are anticipated to have a BMI 0.74 (1.39) kg/m^2 above that of their medium (high) SES peers and a 4.3 (8.4) percentage point greater obesity prevalence. These differences are all statistically significant.

The SES disparities widen with age. The BMI gap between low and high SES individuals is predicted to rise by 0.040 kg/m^2 per year of age (0.80 kg/m^2 over 20 years) and the obesity disparity by 0.41 percentage points per year (8.2 points over 20 years). These magnitudes are reasonably consistent with those in the descriptive analysis, where the BMI disparity between low and high SES sample members increased from 0.5 kg/m^2 for 20 years old to 1.3 kg/m^2 at age 40, and the obesity gap rose from 6.2 to 11.7 percentage points. (The on-line appendix provides further details.)

The remainder of Table 3 specifies SES as a continuous variable measured by the mother's years of completed schooling. The basic model indicates that BMI (obesity) rises a statistically significant 0.12 kg/m^2 (0.6 percentage points) per year of age and falls 0.20 kg/m2 (1.2 points) for each additional grade completed by the mother (columns 2a and 2b). The age effects are identical to those in model (1 a) and the SES gradients accord closely with them.[32] Inclusion of an age–SES interaction confirms that the gradient rotates: the predicted disparity rises 0.007 kg/m^2 for BMI and 0.07 percent-age points for obesity per year of age (see column 2b). To place these in perspective, the BMI gap between respondents at the 10th and 90th percentiles of maternal education (9 and 16 years of schooling) is anticipated to grow 1.04 kg/m^2 between the ages of 20 and 40; the corresponding disparity in obesity prevalence increases 9.7 percentage points.

The continuous SES classification uses all available information on the mother's education. However, the findings may be sensitive to outliers, particularly if the effects are nonlinear and because a substantial portion of the sample report that their mother received very little schooling.[33] To address this concern, columns (3a) and (3b) "winsorize" SES (Angrist and Kruegar, 1999) by setting the minimum (maximum) level of maternal education to 7 (16) years, corresponding to the 5th (95th) percentile. The resulting age profiles are essentially identical to those in models (2a) and (2b), while the SES gradients are 25–30% larger. This is not surprising, since winsorizing reduces the variance of SES, but it suggests that the previous results provide conservative predictions of the SES disparities.[34]

It may be difficult to separate the effects of cohort aging and secular time trends at the top and bottom of the NLSY age range, since data at these ages are only obtained early or late in the sample period.35 This problem may be reduced by limiting the sample to 24–36 years old, for whom observations are available in each survey year. These results, shown in columns (4a) and (4b), are fairly similar to those using the full age range: age

coefficients and age–SES interactions are virtually identical for BMI and are somewhat larger for obesity, while the SES coefficients rise slightly.

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 maternal education	0.7370 (0.1685)	0.7356 (0.1684)	Maternal education	−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 × low education		0.0268 (0.0094)	Age × education		−0.0074 (0.0015)		−0.0090 (0.0018)		−0.0073 (0.0021)
High maternal education	−0.6514 (0.1981)	−0.6526 (0.1985)							
Age × high education		−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 maternal education	0.0432 (0.0114)	0.0430 (0.0114)	Maternal education	−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 × low education		0.0027 (0.008)	Age × education		−6.9E−4 (1.2E−4)		−8.8E−4 (1.4E−4)		−7.9E−4 (2.0E−4)
High maternal education	−0.0407 (0.0120)	−0.0408 (0.0121)							
Age × high education		−0.0014 (0.009)							
Comment	Trichotomous SES			Continuous SES		Winsorized SES		24–38 years old	

Note: See note in 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 maternal education refer to <12, 12, and >12 years of schooling completed by the respondent's mother. Maternal education 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 maternal education are measured as deviations from the sample means. Model (4) limits the sample to 24–38 years old. 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 years old. The regressions also include controls for gender and the interview year.

We conducted other tests of robustness. First, we included the 3828 person-year observations (5.4% of the sample) lacking information on maternal education, with mother's education coded to zero and a missing education dummy variable added. None of the coefficient estimates were substantially affected, with the largest change being a 5% reduction for age in the obesity equation. Next, we allowed nonlinear age effects by controlling for age squared and its interaction with SES. The BMI models provided modest evidence of concavity, while the quadratic terms were small and insignificant for obesity. A third set of models removed secular changes in body weight from individual values using the adjustment procedures described in Section 4. The BMI estimates were identical to those previously reported and the obesity predictions were similar, although with somewhat stronger age and SES disparities and less rotation of the gradient. Fourth, we corrected BMI and obesity for self-reported errors using the methods discussed in Section 3. Coefficients on SES and its interaction with age were essentially unaffected, while the age parameters increased 10–17%. Fifth, we expanded the sample to include all persons with valid weight information in 2004, even if values were missing for some preceding years. This led to slightly (3–8%) larger age effects, no change in the average SES disparity, and marginally attenuated (3–7%) coefficient estimates on the age–SES interaction. Sixth, we used the following alternative SES proxies: presence of the father in the household at age 14, highest grade completed by the mother or father, highest Duncan occupation score of the mother or father, and respondent AFQT score in 1981. These yielded results consistent with those obtained using maternal education.

Table 4
Econometric estimates of age and SE 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)
Maternal education	−0.1215 (0.0279)	−0.1212 (0.0277)	−0.2877 (0.0390)	−0.2865 (0.0399)
Age × maternal education		−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)
Maternal education	−0.0094 (0.0018)	−0.0093 (0.0018)	−0.0149 (0.0022)	−0.0148 (0.0022)
Age × maternal education		−4.9E−4 (1.5E−4)		−8.8E−4 (1.6E−4)

Note: See notes in 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. Sample weights are incorporated in the estimates and standard errors, reported in parentheses, are adjusted to account for complex survey design.

Finally, we estimated models where the dependent variable was the change in BMI or obesity status occurring between 1981 and 2004. Such "long-difference" specifications automatically control for time-invariant factors that influence body weight and are confounded with SES. We expected the age coefficients to be negative, since age changes by the same amount for all sample members and weight rises relatively rapidly in early adulthood, implying larger growth for younger respondents. We also anticipated relatively large weight increases for the disadvantaged, assuming that the SES gradient rotates with age. These expectations were confirmed. Each additional year of age (in 1981) reduced predicted BMI growth by 0.242 kg/m^2; an additional year of maternal education did so by 0.154 kg/m^2. This latter prediction is 24% smaller than the corresponding estimate in column (2a) of Table 3, suggesting that time-invariant confounding factors account for up to one-quarter of the cross-sectional SES gradient. [36]

7. Subgroups

Table 4 relaxes the assumption that males and females have the same age and SES gradients by providing separate gender-specific estimates. Throughout the remaining analysis, our "preferred" specifications correspond to variants of columns (2a) and (2b) of Table 3.

The average age effect is similar for males and females but SES disparities are considerably larger for women—the coefficient on maternal education is over twice as big for females for BMI and 50% greater for obesity. The interaction coefficients also indicate greater rotation of the SES gradient for women .[37]

Table 5 displays predicted BMI and obesity prevalence for individuals at 20 and 40 years of age and 9 and 16 years of maternal education. As noted, BMI and obesity prevalence increase with age for all groups and decline

with SES, while the SES-gradient widens with age. These disparities are particularly pronounced for obesity. For instance, while the predicted obesity rate of those whose mothers had 9 years of schooling almost triples between the ages of 20 and 40 (rising from 9.0 to 24.7%), expected prevalence for their counterparts whose mothers had 16 years of education is two-thirds as large at age 20 (6.0%) but less than half as great by 40 years of age (12.1%).

Table 5
Predicted BMI and obesity prevalence by age and SES.

Maternal education	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 in Tables 1, 3 and 4. Predictions are obtained from models corresponding to specification (2b) of Table 3.

Table 5 also shows that the SES gradient rotates more for women than men. A 20 years old female whose mother had 9 years of education is 25% less likely to be obese than a corresponding male (7.7% versus 10.2%) but has essentially identical predicted prevalence at age 40 (24.3% versus 24.7%). Conversely, a 40 years old woman whose mother had 16 years of schooling is less than three-fifths as likely to be obese as her male peer (8.6% versus 15.1%).

Table 6 summarizes results from subsamples stratified by race/ethnicity and reveals additional differences.[38] First, SES gaps are generally larger for whites than minorities, with little evidence of an SES gradient for black males. This last result is consistent with previous research (e.g. Chang and Lauderdale, 2005), and disparate results will be obtained for African-American men throughout the remaining analysis. Second, age-related weight increases are larger for black than white males and nonexistent for Hispanic men, but with smaller differences for women. Third, the SES gradient rotates more for whites than nonwhites, although not always by statistically significant amounts. A full understanding of these sex and race/ethnicity disparities is beyond the scope of this analysis. However, it seems unlikely that the age-related rotation of the SES gradients is purely biological in nature since, in this case, we would expect the gradients to be similar across race/ethnicity groups.[39]

Table 6
Econometric estimates of age and SES gradients by gender and race/ethnicity.

Regressor	White males (1a)	Black males (1b)	Hispanic males (1c)	White females (2a)	Black females (2b)	Hispanic females (2c)
Body Mass Index						
Age	0.1140 (0.0467)	0.2433 (0.0724)	−0.0049 (0.0893)	0.1240 (0.0500)	0.0913 (0.0792)	0.0711 (0.1033)
Maternal education	−0.1331 (0.0408)	0.0498 (0.0536)	−0.1080 (0.0477)	−0.2708 (0.0519)	−0.1742 (0.0902)	−0.0796 (0.0608)
Age × education	−0.0043 (0.0026)	−0.0016 (0.0037)	−0.0030 (0.0029)	−0.0093 (0.0029)	0.0014 (0.0056)	0.0041 (0.0026)
Obesity prevalence						
Age	0.0056 (0.0032)	0.0121 (0.0053)	−0.0050 (0.0068)	0.0064 (0.0030)	0.0054 (0.0049)	−0.0007 (0.0069)
Maternal education	−0.0094 (0.0026)	−0.0016 (0.0044)	−0.0067 (0.0039)	−0.0137 (0.0029)	−0.0119 (0.0056)	−0.0061 (0.0036)
Age × education	−5.2E−4 (2.2E−4)	−1.3E−4 (4.3E−4)	−1.5E−4 (2.8E−4)	−8.6E−4 (2.2E−4)	−4.8E−4 (4.7E−4)	−3.7E−4 (2.8E−4)

Note: See notes in Tables 1 and 3. Models correspond to those in column (2b) of Table 3. Sample sizes are 19,986, 9651 and 5591 for white, black and Hispanic males and 17,678, 8990 and 5151 for corresponding females. Sample weights are incorporated in the estimates and standard errors, reported in parentheses, are adjusted to account for complex survey design.

8. SES transmission processes

This section investigates potential mechanisms for our findings that individuals growing up disadvantaged weigh more at given ages and that the gradient rotates. We begin by emphasizing several caveats. 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 (although we touched on this above), measurement

error in other covariates or omitted characteristics. Similarly, differences in schooling could be correlated with discount rates that are an underlying causal determinant of adult weight. Second, our regressors are often limited. In particular, data on leisure-time physical activity are available only late in the sample period and may be measured with significant error, and the NLSY lacks information on energy intake, an important determinant of body weight.[40] Third, the mechanisms for any observed correlations may be difficult to ascertain. For example, even if the higher education levels obtained by advantaged children cause lower rates of subsequent obesity, we will not know whether this reflects differences in lifestyles, access to health information, or schooling-induced changes in preferences.

8.1. Child and adult socioeconomic status

Our evidence that socioeconomic status at young ages is related to adult obesity is consistent with a large body of research showing lasting health effects of early life conditions. There are three reasons why this might occur. Adult weight could be affected only by contemporaneous status but with SES itself being highly correlated over time. In this case, childhood SES matters because advantaged youths become advantaged adults. Alternatively, early life conditions might affect adult weight, whereas contemporaneous status does not. Finally, child and adult SES might have independent effects. These possibilities are examined in Table 7, which provides results for models that control for previous and current SES as measured by maternal education and the respondent's highest grade completed.[41] We also briefly discuss (but do not display) findings using family income to proxy current SES.

The results suggest that early life conditions are important, partly because they are correlated with subsequent status, but that child and contemporaneous SES also have distinct effects. When maternal and respondent education are *separately* controlled for, an extra year of maternal education reduces estimated BMI by a statistically significant 0.202 kg/m^2 and obesity by 1.20 percentage points (model 1), while the decreases associated with an extra year of the respondent's education are 0.218 kg/m^2 and 1.59 percentage points (column 2). Including both simultaneously (specification 3) attenuates the coefficient on child (adult) SES by 26–37 (23–33)% but remains substantial: an extra year of maternal (respondent) education is predicted to lower adult BMI by 0.150 (0.146)kg/m^2 and obesity by 0.76 (1.23) percentage points.[42]

The findings are similar when stratifying by sex but with larger race/ethnicity differences. Maternal and respondent education are negatively related to weight outcomes for males and females, entered either separately or together, with stronger SES gradients for women than men. There is somewhat greater attenuation of the coefficient on maternal than respondent schooling for males when controlling for both simultaneously, whereas the reverse is true for females. Contemporaneous and childhood SES are negatively correlated with BMI and obesity for most race/ethnicity subgroups, but maternal education is only weakly correlated with the body weight of black males and respondent education is *positively* related to it (the on-line appendix provides further information).

In models proxying contemporaneous SES by family income (in the previous calendar year), income was significantly negatively related to the weight outcomes when respondent education was not controlled for but much less so when it was: the full sample income coefficient was −0.0023 (−1.67E−4) for BMI (obesity) without respondent education compared to −0.0014 (−1.01E−4) with its addition. By contrast, the parameter estimate for respondent education was only slightly attenuated by controlling for income—changing from —0.2176 to —0.2066 (-0.0159 to —0.0153) for BMI (obesity).

8.2. Additional propagation mechanisms

This section examines the following additional mechanisms through which the effects of early life conditions might transmit to adult body weight: (1) some effects of childhood SES are linked to race-based disparities; (2) persons growing up in advantaged households have relatively high adult incomes; (3) SES is correlated with marriage and fertility rates; and (4) SES early in life is linked to subsequent health behaviors.

The beneficial effects of advantaged childhood circumstances primarily propagate through education and, to a lesser extent, race/ethnicity. The estimates in Table 8 exclude age-maternal education interactions and so constrain the SES gradient to be age-invariant. Results in columns (1) and (2), previously presented in Table 7, show that controlling for the respondent's education attenuates the full sample coefficient on maternal schooling by 26% for BMI and 37% for obesity. Also accounting for race/ethnicity (specification 3) shrinks the maternal schooling parameter by a total of 41 (50)% for BMI (obesity), with the education and race/ethnicity effects being essentially additive.

Table 7
Econometric estimates of childhood and current SES gradients.

Regressor	Body Mass Index			Obesity		
	(1)	(2)	(3)	(1)	(2)	(3)
Full sample						
Maternal education	−0.2017 (0.0242)		−0.1499 (0.0256)	−0.0120 (0.0014)		−0.0076 (0.0016)
Respondent education		−0.2176 (0.0317)	−0.1464 (0.0343)		−0.0159 (0.0019)	−0.0123 (0.0021)
Males						
Maternal education	−0.1215 (0.0278)		−0.0845 (0.0317)	−0.0093 (0.0018)		−0.0049 (0.0021)
Respondent education		−0.1392 (0.0359)	−0.1000 (0.0407)		−0.0141 (0.0026)	−0.0117 (0.0030)
Females						
Maternal education	−0.2877 (0.0390)		−0.2173 (0.0423)	−0.0149 (0.0021)		−0.0104 (0.0023)
Respondent education		−0.3157 (0.0488)	−0.2094 (0.0541)		−0.0183 (0.0026)	−0.0132 (0.0029)

Note: See notes in Tables 1–4. Regressions correspond to model (2a) of Table 3, except for the additional controls for the highest grade completed by the respondent at the survey date. Sample weights are incorporated in the estimates and standard errors, reported in parentheses, are adjusted to account for complex survey design.

Conversely, little of the impact of childhood SES appears to be transmitted through family income, household composition, or the available health behaviors. Higher income predicts lower body weight but the effects are small and controlling for them only slightly attenuates the maternal education coefficient (column 4). Nor is the latter much affected by holding constant household composition or health behaviors.[43] When simultaneously including all supplementary controls (model 5), 45 (55)% of the maternal education effect on BMI (obesity) is "explained," with 92 (90)% of this being due to education and race/ethnicity. There is also no evidence that the additional covariates account for much of the predicted effect of contemporaneous SES. Controlling for race/ethnicity attenuates the respondent education parameter by 5 (3)% for BMI (obesity); also accounting for family income decreases its magnitude by a total of 10 (8)%; and holding constant household status and health behaviors substantially *increases* this coefficient.[44]

Broadly similar results are obtained when examining men and women separately (see the bottom four rows of Table 8). Respondent education and race/ethnicity separately attenuate the maternal education parameter by 30–47 and 14–15%, respectively, for men and 45–62% in combination. When also controlling for family income, household composition and health behaviors, the predicted effect of childhood SES declines 49–69%. Education and race/ethnicity individually attenuate the maternal education coefficient for females by 24–30 and 15–19% and by 42–44% when included together. This coefficient falls 47–50%, in absolute value, when controlling for all supplementary covariates. Finally, the predicted effect of contemporaneous SES increases with the additional controls, although by less for women than men.[45]

Lastly, we use estimates from models that include age–SES inter-actions to calculate predicted BMI and obesity prevalence at 20 and 40 years of age for high and low childhood SES (defined as 9 and 16 years of maternal education).[46] The difference between the two groups, predicted from the basic model and previously summarized in Table 5, is the "SES gap." We then calculate expected SES disparities, after controlling for supplementary covariates, and compute the share of the initial gap "explained" by them.[47]

The results, displayed in Table 9, confirm the dominant role of education and, to a lesser extent, race/ethnicity in transmit-ting the effects of family background. The supplementary variables "explain" almost the entire relatively small SES differential in obesity for 20 years old but much less of the BMI gap. Much larger disparities are predicted at age 40, with just under half of the gap accounted for by the additional controls.

Table 8
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										
Maternal education	−0.2017 (0.0242)	−0.1499 (0.0256)	−0.1192 (0.0273)	−0.1169 (0.0271)	−0.1118 (0.0264)	−0.0120 (0.0015)	−0.0076 (0.0016)	−0.0060 (0.0017)	−0.0058 (0.0017)	−0.0053 (0.0016)
Respondent education		−0.1464 (0.0343)	−0.1390 (0.0340)	−0.1315 (0.0340)	−0.1769 (0.0358)		−0.0123 (0.0021)	−0.0119 (0.0022)	−0.0113 (0.0021)	−0.0137 (0.0023)
Males										
Maternal education	−0.1215 (0.0279)	−0.0845 (0.0317)	−0.0668 (0.0328)	−0.0665 (0.0328)	−0.0616 (0.0324)	−0.0094 (0.0018)	−0.0049 (0.0021)	−0.0036 (0.0022)	−0.0034 (0.0021)	−0.0029 (0.0021)
Respondent education		−0.1000 (0.0407)	−0.1021 (0.0411)	−0.1012 (0.0407)	−0.1737 (0.0450)		−0.0117 (0.0030)	−0.0118 (0.0031)	−0.0114 (0.0030)	−0.0152 (0.0035)
Females										
Maternal education	−0.2877 (0.0390)	−0.2173 (0.0423)	−0.1678 (0.0457)	−0.1634 (0.0454)	−0.1529 (0.0439)	−0.0149 (0.0022))	−0.0104 (0.0023)	−0.0084 (0.0026)	−0.0081 (0.0025)	−0.0075 (0.0025)
Respondent education		−0.2094 (0.0541)	−0.1965 (0.0533)	−0.1818 (0.0539)	−0.2208 (0.0546)		−0.0132 (0.0029)	−0.0125 (0.0029)	−0.0117 (0.0030)	−0.0139 (0.0031)
Race	No	No	Yes	Yes	Yes	No	Yes	Yes	Yes	Yes
Income	No	No	No	Yes	Yes	No	No	Yes	Yes	Yes
Other	No	No	No	No	Yes	No	No	No	Yes	Yes

Note: See notes in Tables 1–4 and 7. Regressions correspond to model (2a) of Table 3, with the inclusion of additional covariates detailed within and at the bottom of the table. Adult SES is proxied by respondent education at the survey date. Income refers to total family income in the calendar year before the survey date. Respondent 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 household composition (marital status and number of children), and health behaviors related to smoking, drinking, exercise, and job-related fitness and strength demands. See Table 1 and the text for additional details on these variables.
Sample weights are incorporated in the estimates and standard errors, reported in parentheses, are adjusted to account for complex survey design.

Table 9
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 years old						
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	34.1%	99.6%	−5.8%	92.7%	57.6%	111.9%
40 years old						
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	47.5%	46.5%	68.4%	62.5%	42.6%	39.1%

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.

Schooling and race/ethnicity are individually responsible for 59–63 and 33–38% of the explained disparity among 40 years old, with education representing a particularly important propagation mechanism for males. A possible concern is that respondent education may be a more important mechanism for transmitting the effects of childhood advantage when the latter is proxied by maternal schooling than when using other SES measures. For this reason, we replicated Tables 8 and 9 using Duncan occupation scores as an alternative indicator of childhood status. The results were quite similar to those presented and confirmed the dominant role of contemporaneous education and, to a smaller degree, race/ethnicity as propagation mechanisms.[48]

9. Discussion

Body weight rises during the transition from early to middle adulthood. BMI (obesity prevalence) is predicted to grow about 0.12 kg/m^2 (0.6 percentage points) per year. The increase is approximately linear over most ages examined, although with some evidence of concavity for the oldest sample members. Virtually identical estimates are obtained for men and women with some race/ethnicity differences (e.g., faster growth for black males and slower increases for Hispanics).

Excess body weight is inversely related to childhood SES and the disparity increases with age. Our main proxy for early socioeconomic status is schooling by the respondent's mother. The regression results suggest that an additional year of maternal education reduces BMI (obesity) by an average of 0.20kg/m^2 (1.2 percentage points) and that this effect increases by 0.007 kg/m^2 (0.07 points) per year of age. Larger SES disparities are predicted for women than men, with smaller differences for minorities than whites and little evidence of an SES gap for black males.

Preliminary examination of the mechanisms through which the beneficial effects of childhood advantage translate into future outcomes highlights the importance of educational attainment and race/ethnicity. When entered into the models separately, respondent schooling attenuates the childhood SES effect by 26–37% and race/ethnicity by 15–16%. In combination, they explain a large majority of the (small) SES gap in the obesity of 20 years old but less of the (larger) disparity at age 40. Conversely, family income, marital status, number of children, and health behaviors play only a minor role.

Evidence that SES differences in body weight grow with age is consistent with research results for other health outcomes. As with that literature, pathways for these effects are only partially understood. Between one-third and three-fifths of the differential predicted for 40 years old remains unaccounted for, after including our full set

of controls, and we know even less about how education and race/ethnicity operate. Such uncertainty is by no means unique to this study. Cutler and Lleras-Muney (2006, pp. 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." Racial disparities in health outcomes such as infant mortality are similarly large, persistent and difficult to explain (Stockwell et al., 2005).

We are also unable to identify mechanisms for the correlation between *current* SES and weight outcomes – the included covariates increase the estimated gap for most groups – and we view this as a fruitful future research topic. That said, substantial race/ethnicity differences in the lifecycle evolution of SES gradients make it unlikely that the latter result purely from biological causes.

Our findings may be applicable to countries other than the United States. For instance, Hardy et al. (2000), using British data, provide evidence that BMI rises with age, with faster increases for those growing up in low SES households and substantial attenuation of the SES gradient obtained when controlling for adult education but not levels of smoking or exercise.[49]

The preceding results should be interpreted in light of several caveats. First, self-reports of height and weight are measured with error and these reporting inaccuracies could differ with SES, probably such that our estimates understate the age-related rotation of the gradient. Second, although we obtain little evidence that income or the health behaviors are important SES transmission mechanisms, these are sometimes poorly measured (e.g. exercise information is only included in the later NLSY survey years) and we lack data on other key inputs, particularly those related to eating patterns and diet.

Third, although much research examines how socioeconomic status generates or is related to health disparities, the meaning of SES is imprecise and subject to a variety of conceptualizations. Economists often focus on income-based measures, with education sometimes used to proxy for permanent income, while other social sciences more frequently emphasize the role of social class. Our analysis is specific in using maternal and respondent education levels as our primary SES proxies, but we do not claim that these are complete or necessarily the best definitions. For instance, maternal education could influence body weight in ways (e.g. through parenting style or health knowledge) that some researchers may consider unrelated to socioeconomic status. One strategy to address this was to repeat our analysis using alternative measures of childhood and adult SES. The results were generally not sensitive to these choices, but future investigations should experiment with additional proxies and, as the necessary data become available, examine whether the observed patterns persist later in life. It would also be interesting to link the results obtained for obesity to other health outcomes, particularly those directly affected by excess weight.

We conclude by reiterating what our findings add to the existing literature. Previous related studies using cross-sectional data (such as NHANES) provide evidence of age-related increases in body weight and an inverse relationship for most groups between contemporaneous socioeconomic status and the latter. Our analysis confirms these results but goes further. Specifically, we distinguish between the effects of contemporaneous SES and status in childhood, and we supply some information on the processes by which early life conditions transmit to adult outcomes. Our evidence suggests that low SES children are more likely than their counterparts to be obese in adulthood, partly because disadvantaged youths become disadvantaged adults, but early life conditions and adult status also have independent effects. These patterns differ substantially by race/ethnicity in ways that we do not yet fully understand.

Notes:

[3] Chang and Christakis (2005) give similar reasons for using body weight and obesity to examine the relationship between income inequality and health.

[4] Some effect could remain when proxying SES by household income if, for example, parents reduce work when their children have serious health problems (Powers, 2003; Noonan et al., 2005).

[5] Smith (2004) confirms that health-SES disparities increase through at least age 50 but suggests that a narrowing occurs later in life.

[6] The limited research examining adult SES and age-related weight changes typically analyzes convenience samples and/or follows individuals for only a short period of time (Ball and Crawford, 2005). In addition, it is generally difficult to establish whether the statistical associations between adult SES and body weight are causal.

7 For instance, the effects of aging and secular trends in body weight were confounded.

[8] This discussion draws heavily on the framework developed by Cutler et al. (2003).

[9] Weight gain also depends on *b*, which may differ systematically by sex.

[10] Even if energy intake and expenditure are fully determined by current status, SES at younger ages could have a temporary "residual" effect if steady-state weight has not yet been reached.

[11] For instance, advantaged youths may be more exposed to healthy eating habits, and these effects may be reinforced through higher levels of subsequent education.

[12] Whether early conditions matter at all could then be examined through models controlling for child but not adult SES.

[13] This drops 4827 person-year observations including a maximum (minimum) of 504 (27) in 1985 (2004).

[14] The questions are "How much do you weigh" (in pounds) and "How tall are you" (in inches)?.

[15] Some researchers prefer measures such as waist circumference, waist-hip or waist-height ratio, or body fat estimated using bioelectrical impedance analysis. None of these are available in the NLSY.

[16] 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 BMI is between the 85th and 95th percentile.

[17] 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.

[18] As mentioned, we also excluded women in years they were pregnant or had recently given birth.

[19] Details are available in the on-line appendix.

[20] Duncan scores have been widely used (Featherman et al., 1975; Stevens and Featherman, 1981), with higher scores indicating greater prestige. Respondents were excluded from this portion of the analysis if neither parent worked or was alive in 1978. The AFQT variable used is the residual from a regression of AFQT scores on age dummy variables.

[21] This procedure relies on the assumption that exercise is highly correlated over time, in which case data from later survey years provides useful information on physical activity earlier in life. The few studies examining longitudinal patterns of physical activity (Kimm et al., 2002,2003; Gordon-Larsen et al., 2004; Nelson et al., 2006) provide evidence that activity levels are correlated over time.

[22] These are based on 1970 and 2000 U.S. Census occupational classification codes (U.S. Department of Labor, Employment and Training Administration, 1991) used in the NLSY to classify each respondent's current or most recent job. The measures are set to zero for respondents not employed at the survey date.

[23] For example, we make no attempt to resolve the debate over whether smoking reductions play a major role in explaining the growth in obesity.

[24] This is necessary because the NLSY is a multi-stage stratified sample with geo-graphically clustered respondents.

[25] BMI is marginally higher (22.4 kg/m^2 in 1981 and 27.6 kg/m2 in 2004) for the sample of all individuals providing weight data in 2004, with larger differences in obesity and class 3 obesity (prevalence rates were 28.0 and 3.50% in 2004).

[26] 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% compared to 0.5% for males, and estimated obesity prevalence grew 17.0% for women versus 9.0% for men.

[27] For example, the obesity prevalence of women in 2004 was 24.1 %in the balanced sample versus 27.3% when including all persons providing weight information in that year, whereas the corresponding prevalences for men were 28.8 and 28.7%.

[28] The disadvantaged also exhibit larger weight gains when SES is proxied by presence of a father in the household at age 14, highest parental grade completed, Duncan occupation scores and AFQT scores.

[29] For example, average BMI increased 2.875 or 0.119 kg/m2 per year between 1976–1980 and 1999–2004. The trend variable *T* takes values ranging from 0 in 2004, the most recent NLSY interview, to 23 in 1981, the first year weight is reported, and adjusted BMI is calculated by adding 0.119 kg/m^2 x *T* to the respondent's self-reported BMI. This calculation is based on a 24-year average difference in the timing of interviews in NHANES II (1977.5) and the most recent NHANES survey (2001.5). NHANES trends were calculated for 24–38 years old because all NLSY respondents were eligible to be interviewed in this age range. However, the results are not sensitive either to the ages chosen or to calculating trend growth in BMI for more narrowly defined age groups.

[30] Separate adjustments were not used for SES subsamples, since NHANES does not contain information on maternal education (our main childhood SES measure). This is likely to lead to an *understatement* of the extent to which SES gradients steepen with age. Previous research indicates that SES-BMI differentials are narrowing over time (Zhang and Wang, 2004; Chang and Lauderdale, 2005), implying that full adjustment would result in larger (smaller) increases in BMI for high (low) SES individuals at young ages. Analysis of NHANES data with SES proxied by the respondent's education (a measure of adult SES shown below to be related to early life conditions) provides additional evidence that the SES gaps have diminished over time.

[31] For example, 4.6% of the lowest SES group are obese at age 18 and 31.3% at 40 years of age, while corresponding growth for their high SES counterparts is from 1.9 to 19.6%.

[32] Maternal education is 3.2 (6.3) years greater for medium (high) than 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 medium (high) SES individuals and a 3.8 (7.6) percentage point disparity in obesity. Corresponding SES gaps predicted by model (1 a) are 0.74 (1.39) kg/m2 and 4.3 (8.4) points.

[33] Less than 6 years of maternal education is reported for almost 6% of (unweighted) observations.

[34] Similar results are obtained in estimates that excluded persons whose mothers reported fewer than 6 years of education (without winsorizing).

[35] For example, data on 18–19 years old come exclusively from 1981/1982 and those on 46–47 years old are only from 2004.

[36] The age and SES coefficients are both statistically significant. We do not present results from long-difference models in subsequent analysis because they only capture that portion of the SES effect occurring between 1981 and 2004 and miss the effects at younger ages (before 1981).

[37] Gender disparities in the age and SES coefficients are increased and the magnitude of the age–SES interaction is slightly reduced by expanding the sample to include all persons providing data on body weight in 2004.

[38] The age and SES main effects are essentially identical in models that exclude the interaction between them, as expected since these variables are expressed as deviations from means.

[39] Conversely, biological differences could be important when comparing males with females.

[40] Information on the frequency of exercise was provided through a variable listing the number of times the respondent engaged in the activity and a second variable indicating the unit of time (daily, weekly, monthly or yearly). There appear to be significant coding errors for the latter variable. We made substantial efforts and used reasonable assumptions to correct the data but suspect that some measurement error remains.

[41] The coefficients on age are similar to those obtained previously and so are not shown on the table.

[42] When comparing the coefficients on childhood and contemporaneous SES, it is important to note that the mean value of respondent schooling is higher than that of the mother's education (13.2 years versus 11.8 years).

[43] The attenuation is minimal because the household composition coefficients are in the expected direction (being single and having more children is associated with higher body weight) but small in magnitude and with little evidence of greater weight for the lifestyles common among low SES individuals. Specifically, current or past smoking is associated with lower body weight and binge drinking with higher BMI but not obesity. Exercise and job-related physical requirement variables yield mixed results.

[44] This largely occurs because smoking is negatively correlated with weight and respondent education.

[45] There are also race/ethnicity differences. For whites, one-third to two-thirds of the child SES differential is transmitted through respondent education. Education is also important for Hispanics, accounting for one-fifth to one-half of the total effect but, in contrast to whites, the other included mechanisms together have approximately equal explanatory power. Including respondent education attenuates the maternal schooling coefficient by a relatively small amount (15–23%) for black females and moves this parameter from positive to zero or zero to negative for black males.

[46] Interactions of age with respondent education and race/ethnicity are included in specifications containing main effects for these variables.

[47] For example, in the basic model, low (high) SES 20 years old were predicted to have a BMI of 24.134 (23.309) kg/m^2, for a gap of 0.825 kg/m^2. Predicted BMI is 24.030 (23.339)kg/m^2 after controlling for respondent education yielding an SES difference of0.691 kg/m^2.This is 16.2% smaller than the initial disparity.

[48] For instance, in the models corresponding to Table 8, the full sample Duncan score coefficient declined 32 (38)% for BMI (obesity) when accounting for respondent education and 22 (17)% with the inclusion of race/ethnicity covariates. Holding both constant attenuated the Duncan parameter by 53 (55)%, and including all supplementary controls "explained" 56 (61)% of the total effect.

[49] Hardy et al.'s study contains a number of potential shortcomings: all participants were born during a single week during 1946, so it is not possible to separate age and period effects; the SES measures are relatively crude with no sensitivity testing with alternative proxies; and the outcomes are measured at just four ages (20, 26,36 and 43).

Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at doi:10.1016/jjhealeco.2009.01.004.

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