

CUMULATIVE DISADVANTAGE AND HEALTH: LONG-TERM CONSEQUENCES OF OBESITY?

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Drawing from cumulative disadvantage theory, the health consequences of obesity are considered in light of the accumulation of risk factors over the life course. Two forms of compensation are also examined to determine if the risk due to obesity is persistent or modifiable. Analyses make use of data from a national survey to examine the consequences of obesity on disability among respondents 45 years of age or older, tracked across 20 years (N = 4,106). Results from tobit models indicate that obesity, especially when experienced early in life, is consistently related to lower-body disability. The results also show that obesity has long-term health consequences during adulthood, altering the life course in an enduring way. Compensation was not manifest from risk-factor elimination (weight loss), but rather through regular exercise. Although there is evidence for long-term consequences of risk factors on health, the findings suggest that more attention should be given to compensatory mechanisms in the development of cumulative disadvantage theory.

SOCIOLOGICAL studies of life chances and well-being have contributed much to our understanding of the life course and the structure of inequality. As empirical generalizations accumulate on how inequality develops over the life course, recent research points to two innovations that have greatly shaped the course of inquiry. First, examining physical or biological markers—whether measured or reported—reveal much about life course opportunities and constraints (Barker 1997; Booth, Carver, and Granger 2000; Conley and Bennett 2000; Seeman et al. 2001). Many markers of physical characteristics, such as birth weight, hormones, and skin tones, may be associated with social ar-

rangements and, hence, quality of life. Indeed, there are many ways that these or other physical characteristics may shape opportunities in life, including perceived attractiveness, stigma, and stratification. Second, long-term examinations of life chances, health, and well-being demonstrate the importance of taking the “long view” in studying life course inequality—integrating information over decades of the life course (Blackwell, Hayward, and Crimmins 2001; Ferraro and Farmer 1999; Sampson and Laub 1996; Warren, Hauser, and Sheridan 2002). Some risks are immediate in their effects, but others take considerable time to become manifest.

One risk that has garnered considerable attention during the past decade is excess body weight. Obesity has long been recog-

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nized as a target of stigma in many societies (DeJong 1980; Stunkard, LaFleur, and Wadden 1998) and has influenced labor market location (Averett and Korenman 1999) and wages (Register and Williams 1990). Despite the social consequences of obesity, the prevalence of persons with a body mass index (BMI) greater than 30 in the United States has risen substantially in recent decades.¹ Estimates vary across studies of the U.S. population, but between 18 and 25 percent of adults are now considered very overweight or obese, up from about 12 percent in the early 1960s (Kuczumski et al. 1994; McTigue, Garrett, and Popkin 2002; Taubes 1998). This increase, especially in the past decade, has prompted some to describe the growing prevalence of obesity in the United States as an epidemic (Flegal et al. 1998; Mokdad et al. 1999). At the same time, dozens of epidemiological studies and several federal reports have drawn attention to the health consequences of obesity (e.g., NHLBI 1998). The bulk of the empirical research has documented a substantial effect of obesity on morbidity and mortality (Bender et al. 1998; Durazo-Arvizu et al. 1998), but more recent research has begun to examine the consequences of obesity on other dimensions of health-related quality of life.

The purpose of this paper is to investigate the long-term risks of obesity on health inequality in adulthood. Drawing from cumulative disadvantage theory, the analysis examines the consequences of obesity throughout adulthood on multiple health-status measures. Data from a long-term, national panel study of adults is used to examine whether early and/or recurrent obesity accelerate health decline in adulthood.

CUMULATIVE DISADVANTAGE AND LIFE COURSE INEQUALITY

Cumulative disadvantage theory provides the framework for this analysis and helps to

formulate the specific research questions. Although related to age stratification (Riley 1987), life course (Elder 1985, 1994), and life-span developmental perspectives (Baltes, Reese, and Lipsitt 1980), cumulative disadvantage theory emerged with a focus on intracohort differentiation (Dannefer 1987). A wide array of research on status attainment provided the background for the development of cumulative disadvantage theory. Merton (1968) noted how the reward system in science favors those who have established reputations, especially those scientists deemed "eminent." He described the phenomenon of cumulative advantage in scientific careers as the "Matthew effect" (based on the gospel of Matthew).² In addition, research on organizational dynamics and status attainment provided support for a tournament-mobility model, emphasizing the importance of *early* success in a career (Rosenbaum 1979).

Cumulative disadvantage theory emphasizes how early advantage or disadvantage is critical to how cohorts become differentiated over time. Not only do the early risk factors shape trajectories in the short-term outcomes but in the long-term outcomes as well. The effects of risk factors accumulate over the life course, thereby increasing heterogeneity in later life (Dannefer 1987, 1988a, 1988b; Dannefer and Sell 1988; O'Rand 1996; Ross and Wu 1996; Settersten 1999). Personal change is seen as closely related to traits or characteristics displayed earlier in the life course. According to cumulative disadvantage theory, change is anticipated, but the nature of the change is conceptually linked with earlier experiences, abilities, and resources (Elder 1995).

Life course studies point to a wide array of biological and social forces that differentiate a population over time, but cumulative disadvantage theory emphasizes that different trajectories arise from early inequalities.

¹ Obesity is defined here as having a body mass index (BMI) greater than or equal to 30, in accordance with the National Heart, Lung, and Blood Institute (NHLBI) (1998) guidelines. The BMI equals weight in kilograms divided by squared height in meters (kg/m^2). To illustrate, a person 68 inches tall would need to weigh at least 197 pounds to be classified as obese.

² Merton (1968) used the following Biblical text to describe how many scientists, afforded special opportunities early in their careers, were able to transform those opportunities into scientific prominence: "For everyone who has will be given more, and he will have abundance. Whoever does not have, even what he has will be taken from him" (Matthew 25:29).

Some persons are advantaged in their early years, and this advantage may compound over time. Others are disadvantaged because of genetic or environmental factors, and these disadvantages also accumulate (Priestley 2000; Smith 1968). In a sense, disadvantages may "scar" the person's life chances (Preston, Hill, and Drevenstedt 1998). This phenomenon is seen as one of accentuation, thereby increasing diversity in a population as the population ages (Elder 1969). Some people are launched on promising trajectories: The early advantages and successes lead to further advantage. Others are disadvantaged early and, thus, face a cascade of additional risks to health, wealth, and well-being. They might compensate for the early disadvantage by working harder or enduring more, but the challenges they face are clear. Evidence supporting the effect of cumulative disadvantage on measures of IQ, income, and status attainment is considerable (Crystal and Shea 1990; Kerckhoff 1993; O'Rand 1996; Rosenbaum 1975, 1984), and tests of the theory for understanding human health are growing (Ross and Wu 1996).

Epidemiological studies in the past decade have given fresh attention to the role of biological and social factors early in the life course for impacting health in adulthood and older ages. According to Wadsworth (1997), there is a growing interest in taking a "lifetime view of the natural history of some common serious illnesses which usually begin in middle or later life" (p. 860). Health and health resources in childhood and early adulthood are increasingly seen as shaping health in later life (Wadsworth 1991). One limitation of much of the previous research on health inequalities is that many risk factors are measured at the same point in time as is the health outcome. Therefore, it is not surprising, according to Wadsworth, that many potentially consequential factors are not identified. Instead, research must refocus on those potential advantages and disadvantages to health that occur much earlier in life.

Cumulative disadvantage theory also stresses the possibility that feedback mechanisms and cyclical change can occur over the life course. The growing use of longitudinal data is pointing precisely to such mechanisms of spiral decline (Bruce et al. 1994; Ferraro, Farmer, and Wybraniec 1997;

Verbrugge and Jette 1994). For persons beginning with a serious health disadvantage, Verbrugge and Jette (1994:1) describe this process as involving "pernicious loops of dysfunction." For health consequences such as disability, however, the timing of these risk factors in the life course can affect the onset and progression of health decline (Irwin 2001). Identifying the onset of initial risk, the spacing between subsequent feedback risks, and the timing of cyclical change throughout adulthood would help elucidate how cumulative disadvantage shapes health trajectories over the life course (Ferrucci et al. 1996; Grundy and Holt 2000).

Beyond a description of increasing diversity in the population, the literature has moved largely to considering whether the effects of biological, personal, or structural characteristics on health outcomes actually increase, decrease, or remain stable over the life course. Emphasis is placed upon life course trajectories leading to advantage or disadvantage and whether the effects of risk factors change over the life course. Concerning health, Ross and Wu (1996) examine whether the effect of education (and social class, more generally) changes over the life course. Using both cross-sectional and longitudinal analyses, they found that health inequality due to education increased over the life course, leading to greater diversity and inequality in health (also see Ross and Wu 1995). Not all other studies concur. House and colleagues found that the effects of socioeconomic status on health actually decrease in later life: In their view, aging levels the effects of socioeconomic status on health (House, Kessler, et al. 1990; House, Lepkowski, et al. 1994). Other studies report decreasing heterogeneity with age (Clausen 1986) or stability of differences across the life course (Clark and Maddox 1992). At present, the literature is inconsistent regarding how socioeconomic disadvantage accumulates in shaping health inequality over the life course, and this may be partly due to the reliance on cross-sectional data to address age changes in health.

Another issue that merits attention in further developing cumulative disadvantage theory is the degree to which disadvantages are reversible. It should not be assumed that there is a linear relationship between risk-

factor exposure and health consequences. Some life course studies point to fairly permanent effects on health or status attainment resulting from early inequalities (Barker 1997; Wadsworth 1991, 1997), but other studies suggest that effects can be reversed (Elman and O'Rand forthcoming; U.S. Department of Health and Human Services 1990). It is important to identify whether there are compensatory mechanisms that can eliminate, or at least reduce, the effects of earlier disadvantages. Alternatively, do the effects of certain risks propel a fixed trajectory of health decline?

We identify two main types of compensation: *risk-factor elimination* and *countervailing mechanism*. If exposure, especially early exposure, to a health risk is associated with disadvantage in later health or well-being, can elimination of the risk undo the early exposure? Risk-factor exposure at early ages has been likened to a "scarring" effect (Preston et al. 1998). First, we would like to know if risk-factor elimination is sufficient to reduce the consequences of earlier risk exposure. In this sense, it would be useful to know the "reach" of the risk factors as disadvantage accumulates. In other words, are scarring effects temporary or permanent? Second, there may be one or more countervailing mechanisms that can interrupt the effects of risk-factor exposure. Countervailing mechanisms could take the form of events that trigger a change in the effect of the risk factor, or more gradual processes that are sufficient to interfere with or suspend the effects of early-life advantages or disadvantages (Dannefer and Sell 1988). The key distinction is that the risk factor might still be present, but the countervailing mechanism may alter the presumed effect of the early exposure. In the analysis that follows, we consider the role of compensation, via risk-factor elimination and a countervailing mechanism, on changes in health over decades of the life course.

OBESITY AND HEALTH: CUMULATIVE DISADVANTAGE?

The proposed study seeks to extend our understanding of cumulative disadvantage and health with a focus on the role that obesity plays over the life course. We believe that

applying cumulative disadvantage theory to the study of obesity and health is beneficial for a number of reasons that span theoretical, methodological, and policy issues. First, the relationship between body weight and life course health inequality is an important and unstudied topic. Heterogeneity in body weight exists early in the life course, but the health consequences of obesity may take years to develop. Moreover, middle age (ages 35 to 55) is the life course period with the highest prevalence of obesity, which is most often antecedent to the development of many serious illnesses (McTigue et al. 2002). Two factors combine to reduce the prevalence of obesity in later life: premature mortality risk for obese persons and weight loss in later life due to serious illness and/or its treatment (Launer et al. 1994). Thus, studying whether early obesity leads to increases in health inequality represents an appropriate arena for examining the utility of cumulative disadvantage theory. Moreover, applying cumulative disadvantage theory to a topic that has not been investigated from that perspective should provide fresh empirical generalizations. Ultimately, the theory's application to obesity may help to refine or limit the theory.

Second, most of the previous research on cumulative disadvantage theory suffers from an important conceptual and methodological concern: selective survival. As noted above, the emerging body of research testing cumulative disadvantage theory on health yields inconsistent results; evidence exists for increasing divergence (Ross and Wu 1996), increasing convergence (Ferraro and Booth 1999; House et al. 1994), and even stability (Clark and Maddox 1992). Missing, however, from most of these studies is a rigorous consideration of how the findings might be influenced by selective survival (cohort shrinkage). Indeed, much of the evidence for cumulative disadvantage comes from cross-sectional studies in which age differences are used to reflect age changes (for an exception, see Crystal and Waehrer 1996). Whereas obesity is associated with higher mortality risk (Andres, Muller, and Sorkin 1993; Tayback, Kumanyika, and Chee 1990), failure to account for selective survival would likely bias findings toward decreasing heterogeneity in later life. Ross and Wu (1996) state the problem succinctly: "Survival effects can produce

the appearance of convergence in community studies" (p. 107). For the most part, on these grounds they explained the inconsistency between their findings and those of other researchers. We agree that Ross and Wu have identified a critical issue for studies of cumulative disadvantage theory as well as health trajectories, but we propose to take the next logical step in the consideration of selection effects by explicitly accounting for them in statistical models (Winship and Mare 1992). We are aware of only one national study of cumulative disadvantage and health that does this, albeit with fairly limited health measures and over only a three-year period (Ferraro and Booth 1999). We anticipate that applying models that account for selection bias will provide additional evidence for cumulative disadvantage theory, especially because the data used here span 20 years of observation.

Third, the public health rationale for our research is important. Obesity is a serious health risk and a preventable one in the United States as well as in other nations (Andres et al. 1993; James 1995).³ If the effects of BMI on health increase with age, as suggested by cumulative disadvantage theory, then one may anticipate greater health care complications and costs in coming decades with the aging of the baby boom cohort. Thus, the study of compensatory mechanisms is timely and important. We seek to understand not only the long-term effects of obesity on health, but also to identify compensatory processes that may attenuate or eliminate the health risk of obesity.

Fourth, the health outcome considered here, disability, is critical to health and social life but has received relatively little attention as a consequence of obesity. Some studies show that obesity is related to impaired function (Ferraro and Booth 1999; Galanos et al. 1994), while others find no

such link (Kaplan et al. 1993; Lawrence and Jette 1996). Beyond the limited empirical work on obesity and disability, there are important biological mechanisms that may lead to deterioration in physical functioning and, concomitantly, perceptions of health. Although testing the biological mechanisms is beyond the scope of this study, there is ample evidence to suggest that obesity may accelerate disability and health decline more generally.⁴

Finally, several studies have shown that obesity is more common among persons of low socioeconomic status (Croft et al. 1992; Gordon-Larsen, Adair, and Popkin 2003; Sobal 1991). There is increasing evidence that obesity may be a disadvantage in the status-attainment process, and even that it may suppress wages (Averett and Korenman 1999; Register and Williams 1990). At the same time, if obesity is related to functional disability, it could be that the wage or status attainment penalty is due to the compromised physical function associated with obesity (Stone et al. 1992; Thomas 2001). By focusing on the obesity-health connection, the present study examines a possible pathway for how obesity shapes role performance and status attainment.

The overarching research question we address is whether cumulative disadvantage operates in the link between obesity and health. Four specific research questions guide the analysis:

(1) *Is obesity associated with physical disability?* Although there is evidence of obesity's effect on mortality, less attention has been given to other domains of health-related quality of life. We consider the effect of obesity on disability to address an understudied but socially significant health outcome. We anticipate that obesity accelerates disability, even after accounting for selective mortality.

(2) *If obesity is related to disability, is the effect of obesity more substantial during certain periods of the life course?* Do the health

³ The majority of large, well-designed, epidemiological studies show that obesity is associated with higher morbidity and mortality in adulthood (NHLBI 1998; Sjöström 1992). Evidence comes from a variety of sources based on samples from the United States (e.g., National Center for Health Statistics 1991; Tayback et al. 1990) as well as in other nations (e.g., on the United Kingdom, see Fitzgerald and Jarrett 1992; on Finland, see Rissanen et al. 1989).

⁴ Biological mechanisms linking obesity and physical disability include skeletal stress and osteoarthritis (Hart and Spector 1993), glycation of proteins and DNA in soft connective tissue (Pratley et al. 1995), and atherogenesis (Stevens, Gautman, and Keil 1993).

consequences of obesity increase or decrease with age? Based on cumulative disadvantage theory, we hypothesize that obesity's effect is greater in later life. At the same time, some studies suggest that the effects of excess body weight on health may attenuate in later life (Andres 1995).

(3) *Does the timing of obesity influence health inequality?* Does early entry into the obese category, as measured by a BMI > 30, shape a steeper health decline than does later entry into obesity? In addition, does long-term or chronic obesity further accelerate health decline? It is known that body weights change during adulthood, but does prolonged obesity exacerbate health problems? On the basis of cumulative disadvantage theory, we predict that prolonged risk exposure results in heightened disability.

(4) *Can compensatory mechanisms reduce or eliminate the effects of obesity on health decline?* Specifically, does losing weight ameliorate the effect of obesity on health? Or are there other behavioral changes that can undo the effects of risk exposure? Answers to these questions have important public policy implications for ameliorating the health risk of obesity.

Panel data from a national sample, studied for more than 20 years, are used to address these research questions. We are unaware of any studies that have addressed these or similar questions with comparable data.

METHOD

SAMPLE

Data for these analyses come from the National Health and Nutrition Examination Survey I (NHANES I) and its Epidemiologic Followup Study (NHEFS). Data were first collected from 1971 to 1974 and included both interview and medical examination components for the subsample that was administered the "detailed component." This subsample, designed as nationally representative, was a multistage, stratified probability sample of noninstitutionalized persons ages 25 to 74 ($N = 6,913$). Subjects were re-interviewed in 1982, then again in 1987 and 1992. These follow-up surveys are hereafter referred to as Waves 2 through 4 and provide the opportunity to observe health status

change in a nationally representative sample for about 20 years. The percentage of cases at baseline receiving the detailed component and traced through follow-ups was excellent (88 percent of survivors at Wave 2 and 89 percent of survivors at Waves 3 and 4).

Other features of the NHEFS design make it especially appropriate for testing cumulative disadvantage theory regarding the effects of obesity on health. In addition to body weight gathered at the four waves, eligible subjects were asked in the follow-up surveys to recall their weights at ages 25, 40, and 65. Thus, with four weights observed in the surveys and three weights recalled, it is possible to obtain seven body-weight measures at discrete time points over the adult life course for persons who were 45 years of age or older at baseline. To keep the total number of possible body-weight measurements consistent across persons, these analyses use only the age-restricted subsample ($N = 4,107$). Casting the data in this way is useful for assessing the consequence of being obese over multiple points in the life course. It permits one to distinguish the effects of chronic obesity from those of being obese at one time only, especially as it pertains to compensatory mechanisms. In supplementary analyses, we predicted disability with just the measured weights at each wave for the full sample, but the logic of the analyses presented here is to make use of all weight-history information in order to uncover long-term effects of excess body weight on health. This means that the seven points of weight-history information, including both the observed and recalled weights, span about 40 years for the youngest sample members and nearly 70 years for the oldest sample members.

Race was categorized as white, black, or other in the baseline survey. Because there was insufficient statistical power to test differences on the outcomes for the 43 people classified as "other," they were deleted from further analyses. Comparisons of race in subsequent models are between black subjects and white subjects. The final total sample includes 4,106 cases.⁵

⁵ All analyses apply sample weights and adjust for clustering in the multistage sample using Stata 7.0.

BODY WEIGHT ASSESSMENT

For the NHANES I, weight and height were measured by research staff. This was done during a medical examination at the mobile clinic for the baseline survey using a self-balancing scale. For Wave 2, the first follow-up, staff measured weight in the home of respondents with a scale placed on a hard surface. For Waves 3 and 4, weight was based on respondent report. While self-reported weight measures generally are closely related to physical measurements, they may slightly underestimate the tails of the weight distribution (Bowman and DeLucia 1992; Kuskowska-Wolk, Bergstrom, and Bostrom 1992). Some of the analyses presented below do not require use of the self-reported weight measures. For those that do, however, it should be recognized that the relationships considered might be underestimated.

As noted above, beginning at Wave 2, respondents were asked to recall their body weight at ages 25, 40, and 65. If the respondent was older than 45 but not yet 65, he or she obviously was not asked to recall his or her weight at the older age. At subsequent waves, respondents were asked only to recall their body weight at the age category not previously asked.⁶ The seven weights were used with baseline height to calculate seven measures of the body mass index (BMI). The BMI, is often referred to by sociologists as the Quetelet index (see note 1 for a definition). Although there may be situations where this measure is used in its continuous form, previous research clearly shows a curvilinear relationship between age and BMI—average BMI increases in middle age but decreases in later life (Flegal et al. 1998). Given our interest in evaluating obesity as a risk factor over the life course, a categorization of the BMI is a parsimonious way to account for potential nonlinearities. Based on the NHLBI guidelines, persons

were classified as obese if their BMI was 30 or greater (NHLBI 1998).⁷ A binary variable with 1 signifying obese at each age and each survey was used to identify these persons (all others were coded 0). Seven total binary weight variables were thus created. For selected analyses, binary variables were also created to differentiate those who changed from one state to another (e.g., obese to non-obese).

Because both recalled weights and measured weights are used, we performed a series of analyses to examine potential bias between the two forms of measurement and minimize total survey error (Andersen, Kasper, and Frankel 1979). First, we examined correlations between the measured and recalled BMI variables that were less than 20 years apart for all respondents in the analysis (45 years or older). These correlations ranged from .80 to .89, indicating considerable stability in the BMI, even though change is commonplace over a 20-year window. Second, we compared the slopes of two estimates of BMI generated from each mode of data collection. Although the intercept was slightly lower for recalled weight, the slope for recalled weight fell within a 95-percent confidence interval of the measured weights. In summary, recalled weights are closely related to measured weights but may slightly underestimate obesity in this sample.

To apply cumulative disadvantage theory in the present research, the binary variables for obesity were also arrayed into three different formulations based on the timing of the obesity. For selected analyses, it was useful to differentiate early, late, and chronic obesity. Early obesity included those who were obese at age 25 or obese at baseline.⁸

⁷ Considerable previous research also shows that the BMI is related to mortality and other health incomes in a nonlinear fashion (e.g., Durazo-Arvizu et al. 1998). We conducted preliminary analyses with raw and polynomial forms of the BMI, but the analyses based on the binary indicator led to the most parsimonious and straightforward interpretation. Alternative cut-points for defining obesity were also considered—ranging from a BMI of 27 to 31—but the results of preliminary models were consistent across these classifications.

⁸ Because of the high correlation between obesity at age 40 and obesity at baseline in this

⁶ For instance, if a respondent was age 62 at Wave 2, she or he was asked to recall her or his weight at age 45. At Wave 3, the same respondent, who would now be 67, was asked only to recall her or his weight at age 65. Respondents who were the exact age of the recall (45 or 65) were not asked to recall their weight until the following wave.

Late obesity included those who were obese at Waves 2, 3, or 4. Lifetime or chronic obesity was a count variable of the total number of times that a person was obese from age 25 to Wave 4. Chronic obesity ranged from a count of 0, meaning the respondent was never obese, to 7, meaning the respondent was obese at all observation points. For models with an outcome at Wave 2, the count ranged from 0 to 5.⁹

Underweight persons were identified if they had a BMI of less than 17 (NHLBI 1998). Just as with the measurement of obesity, binary variables were created to identify those respondents at each of the seven time points who were underweight. Preliminary models showed that specific times of being underweight were not consequential to disability. A binary variable identifying those who were ever underweight at any of the seven time points was used instead. Table 1 provides a summary of coding and descriptive statistics for the variables used in the analysis.¹⁰

sample, the former was excluded from the early obesity formulation. Either measure could be used, but our preference was to use obesity at baseline because it was measured by the medical examination staff (obesity at age 40 relied on respondent recall). Obesity at age 40, however, was used in calculating the chronic obesity measure.

⁹ In lieu of the count variable for lifetime obesity, preliminary analyses tested for the set of seven (or five) binary variables simultaneously to determine if selected periods of obesity were more consequential to health. No clear pattern emerged from these analyses, probably because of multicollinearity among multiple measures of obesity and the endogeneity of later obesity on earlier states. In the models presented below, we differentiate early obesity from late obesity and use the measure of lifetime obesity to parsimoniously capture the effect due to the chronicity of obesity.

¹⁰ To examine the consequences of childhood weight on adult health, two additional body weight variables were included in preliminary models. Respondents were asked to classify their childhood body weight based on the question: "When you were about 12 to 13 years old, compared to other (boys/girls) of the same age, were you considered to be . . . skinny, somewhat slender, average, chubby or very heavy?" It would be preferable to have measured weight and height for these respondents, but such measures are not

MEASUREMENT OF HEALTH STATUS

There are two health-status outcomes considered in this research: lower-body disability and upper-body disability. Respondents were asked how much difficulty they had performing daily tasks at all follow-up interviews (no measure of disability is available at the baseline survey). The response categories were no difficulty (= 1), some difficulty (= 2), a lot of difficulty (= 3), or unable to perform (= 4). Lower-body disability is a sum of the difficulty performing 10 tasks that focus on ambulation such as walking one-quarter mile. Scores on lower-body disability range from 10, which indicates no difficulty with any task, to 40, which represents an inability to perform any of the tasks. Reliability coefficients (α) for lower-body disability were .93 or greater at Waves 2 and 4.

Upper-body disability is a sum of the amount of difficulty a respondent has performing nine different actions that require upper-body function, such as opening new jars (the index ranges from 9 to 36).¹¹ Reliability coefficients (α) for upper-body disability were .91 or greater at both waves. As anticipated in a community sample of adults, the distributions of these variables are skewed, with more than 70 percent of the sample reporting no disability at each wave. Therefore, tobit models are used to estimate models (Long 1997).

available. Two binary variables, scored 0 and 1, were created from this question to identify those who reported that they were (1) chubby compared with their peers, and (2) skinny compared with their peers. Neither variable, however, had significant effects in any of the models tested; thus, they were deleted from the final analysis.

¹¹ Indicators of lower-body disability are: difficulty in standing from an armless chair, getting in or out of bed, walking one-quarter mile, walking from one room to another, walking up two steps, getting on or off the toilet, getting in or out of a car, picking clothes off the floor, performing light chores, and performing heavy chores.

Indicators of upper-body disability are: difficulty cutting one's own meat, lifting a full cup to one's mouth, washing and drying the whole body, combing hair, lifting a five-pound object, opening push-button doors, opening new jars, using a pen or pencil, and lifting or carrying a bag of groceries.

Table 1. Means, Standard Deviations, and Coding of Obesity and Other Variables: Adults Aged 45 and Older, National Health and Nutrition Examination Survey, 1971 to 1992

Measures	Coding and Range	Mean	(S.D.) ^a
Upper-body disability, Wave 2	Ranges from 9 (low) to 36 (high)	10.11	(3.18)
Upper-body disability, Wave 4	Ranges from 9 (low) to 36 (high)	10.41	(3.95)
Lower-body disability, Wave 2	Ranges from 10 (low) to 40 (high)	11.92	(4.56)
Lower-body disability, Wave 4	Ranges from 10 (low) to 40 (high)	12.23	(5.78)
<i>Early Obesity</i>			
Obese at age 25	1 = yes; 0 = no	.03	—
Obese, Wave 1	1 = yes; 0 = no	.19	—
<i>Late Obesity</i>			
Obese, Wave 2	1 = yes; 0 = no	.14	—
Obese, Wave 3	1 = yes; 0 = no	.11	—
Obese, Wave 4	1 = yes; 0 = no	.10	—
<i>Lifetime Obesity</i>			
Chronic obesity, Wave 2 (times obese by Wave 2)	Count from 0 to 5	.53	(1.05)
Chronic obesity, Wave 4 (times obese by Wave 4)	Count from 0 to 7	.74	(1.46)
Ever-underweight by Wave 2	1 = yes; 0 = no	.15	—
Ever-underweight by Wave 4	1 = yes; 0 = no	.17	—
<i>Change in Obesity</i>			
Became obese by Wave 2	1 = yes; 0 = no	.05	—
Became nonobese by Wave 2	1 = yes; 0 = no	.09	—
Obese, Wave 1 and Wave 2	1 = yes; 0 = no	.09	—
Serious illness, Wave 1	Count from 0 to 4	.49	(.71)
Chronic illness, Wave 1	Count from 0 to 3	.40	(.55)
Change in serious illness from Wave 1 to Wave 2	Count from 0 to 4	.53	(.69)
Change in chronic illness from Wave 1 to Wave 2	Count from 0 to 3	.28	(.52)
<i>Change in Regular Exercise</i>			
Ceased regular exercise by Wave 2	1 = yes; 0 = no	.32	—
Began regular exercise by Wave 2	1 = yes; 0 = no	.03	—
Continued regular exercise, Wave 1 to Wave 2	1 = yes; 0 = no	.59	—
Age at Wave 1	Ranges from 45 to 77	57.41	(8.16)
Female	1 = yes; 0 = no	.53	—
Black	1 = yes; 0 = no	.10	—
Lives alone	1 = yes; 0 = no	.14	—
Education	0 = no formal education; 7 = post-college	3.47	(1.54)
Yearly family income, Wave 1 ^b	1 = < \$1,000; 12 = > \$25,000	7.46	(2.75)
Yearly family income, Wave 2 ^b	1 = < \$3,000; 14 = > \$100,000	7.43	(2.78)
Regular physician	1 = yes; 0 = no	.87	—
Medicaid	1 = yes; 0 = no	.03	—
Private insurance	1 = yes; 0 = no	.85	—
Past smoker	1 = yes; 0 = no	.29	—
Current smoker	1 = yes; 0 = no	.40	—

Note: Number of cases equals 4,107 at Wave 1. Women who gave birth one year before or after any survey wave were omitted from the analysis because of weight implications of pregnancy. Estimates are weighted to U.S. proportions.

^a Numbers in parentheses are standard deviations; standard deviations not shown for binary variables.

^b Income categories are mutually exclusive at each time of measurement but were modified between Wave 1 and Wave 2.

MEASUREMENT OF OTHER VARIABLES

A number of other covariates are included in these analyses. It is well known that morbidity can have substantial effects on disability. Respondents were asked a series of questions regarding specific diseases in the following manner: "Has a doctor ever told you that you have . . . hypertension or high blood pressure?" Thus, each response relies on the report of a medical diagnosis. The diseases asked across waves were differentiated into serious or life-threatening conditions and chronic (non-serious) conditions (Ferraro and Farmer 1999). Counts of each type of disease were created at both Wave 1 and Wave 2. Serious illness ranges from 0 to 5 and includes the following conditions: heart problems (including heart attack), cancer, stroke, hypertension, and diabetes. Chronic (non-serious) health conditions range from 0 to 4 and include: arthritis, kidney problems, cataracts, and hip fracture.

Regular exercise is an important health behavior to consider in studies of health, especially when examining the consequences of excessive body weight. Respondents were asked to assess their level of regular exercise, separately for occupational and recreational, at the baseline and at Wave 2 as "a lot, moderate, or no physical activity." Those who had moderate or a lot of physical activity in either their job or in recreation were coded 1; all others were coded 0. To study compensatory mechanisms, change in regular exercise was also determined between baseline and Wave 2, differentiating four categories defined by regular exercise at the two waves: ceased regular exercise by Wave 2, began regular exercise by Wave 2, engaged in regular exercise at both waves, and no regular exercise at both waves. In estimated models, the first three groups were compared with the non-exercisers at both waves to assess the effect of this countervailing mechanism.

Smoking, another important health-related behavior, was measured using self-reports of consumption of cigarettes, cigars, and pipe tobacco at the time of the interview and during one's lifetime. Current and past smokers were identified by binary variables (0, 1). Changes in smoking status were examined in preliminary models, but changes in smoking

status were not consequential to the long-term health outcomes assessed here.

Several variables indicating access to health care were included in the models. Having a regular physician, having private health insurance, and receiving Medicaid were measured with binary variables where 1 equals yes (all others were coded 0).

Socioeconomic status has been shown to be predictive of health in later life; thus, education and income were included in the analyses. Education ranges on a scale from 0 to 7, with 0 equal to less than 8 years of schooling, and 7 representing a post-college education. The scale for income at Wave 1 has 12 categories, ranging from less than \$1,000 (= 1) to more than \$25,000 (= 12). At Wave 2, the income categories were rescaled, ranging from less than \$3,000 (= 1) to more than \$100,000 (= 14).

Several demographic variables were also used in the models. Age was measured in years. Female, black, and "lives alone" were binary variables for which 1 equals yes (all others were coded 0). Other variables were tested in preliminary models and subsequently dropped because they did not significantly predict upper- or lower-body disability. These include living in an urban area, living in the South, and being widowed.

ANALYSIS PLAN

Although case-tracing and re-interview rates were high in the follow-up study, it is always possible that attrition in longitudinal analyses might influence sample estimates of relationships and lead to bias in the estimates. Thus, Heckman's (1979) selection bias models were used to correct parameter estimates for differential selectivity due to death, refusal to participate, or inability to trace (Stolzenberg and Relles 1997). The procedure was to first estimate a probit model to distinguish cases that participated at each follow-up wave from those who did not. The second step was to include in the regression model of interest a selection instrument (λ) based on the inverse Mills ratio of the probit results. This two-step approach has been extended to incorporate two hazard-rate instruments for different forms of attrition (Maddala 1983) and is conveniently handled in Stata.

The results presented below differentiate attrition due to mortality from that due to nonresponse by estimating separate probit equations. The probit model estimating mortality by Wave 2 showed that deaths were more likely among respondents who were black, older, male, those with more physician-evaluated morbidity, and with less income. The probit model estimating nonresponse by Wave 2 showed that the subjects more likely to drop out of the analysis were black, older, male, with less income, and those with more physician-evaluated morbidity based on the International Classification of Diseases (World Health Organization 1967). These two probit selection equations also included instrumental variables—at least one variable not included in the substantive equation for disability. Separate equations for attrition were estimated for Wave 2 through Wave 4 attrition.¹²

The analysis proceeds in two stages. First, a series of models was estimated for each outcome so that the analysis differentiates early versus late obesity and also considers the effects of long-term or chronic obesity. To account for related changes in morbidity and health behaviors, a series of models was also estimated to examine changes in independent variables. Changes in morbidity and regular exercise are presented in the final models.

The second stage of the analysis examines changes in body weight observed during the study period. Several mechanisms for measuring change in body weight are tested, including 10 percent change in body weight and transitions into and out of the defined obese state. For the latter, several different cut-points for obesity were examined, ranging from 27 to 31 BMI, to assure that the ef-

fect of body weight transitions were not an artifact of measurement thresholds. Because the different methods yielded similar results, transitions across the BMI > 30 threshold are presented.

FINDINGS

Table 2 presents tobit models of lower-body disability at Wave 2 and Wave 4. Whereas no disability measures were taken at the baseline survey, the first three equations show the effects on upper-body disability at Wave 2. Model 1 tests for effects due to early obesity, Model 2 tests for Wave 2 (later) obesity, and Model 3 tests for long-term or chronic obesity. There is evidence from Models 1 and 2 of both lagged and immediate effects of obesity on lower-body disability. As displayed in Model 3, respondents who were chronically obese had higher levels of lower-body disability. Respondents who were ever underweight were not more likely to have higher disability at Wave 2. Illness at Wave 1 was associated with more lower-body disability, but respondents who began or continued a regular exercise program had less lower-body disability. Women had more lower-body disability at Wave 2 than men did, and white respondents were more disabled than are black respondents. In addition, the role of attrition due to mortality was important in shaping Wave 2 disability levels. The selection effect for mortality reveals that people who died before Wave 2 were at greater risk of disability. Supplementary analyses comparing the results from Table 2 with those that did not account for attrition revealed that the effect of obesity was overestimated in the latter. It is clear that failure to account for attrition in panel studies where the outcome is related to attrition will lead to biased estimates of the relationships being investigated.

The results for Wave 4 are presented in the last four models. Models 1 through 3 are parallel to those for Wave 2, but add change in serious and chronic illness from Wave 1 to Wave 2. Model 4 is parallel to Model 3, but specifies Wave 2 lower-body disability as an independent variable to measure change in the outcome. Early obesity (Wave 1) was consequential to lower-body disability 20 years later, and there is no evidence

¹² The probit model estimating mortality by Wave 4 showed that deaths were more likely to occur among respondents who were older, male, those with more physician-evaluated morbidity, and those with less Wave 2 income. The probit model estimating nonresponse by Wave 4 showed that subjects more likely to drop out of the analysis were urban, older, those with less Wave 2 income, and those with more physician-evaluated morbidity based on the International Classification of Diseases (World Health Organization 1967). Instrumental variables were physician-evaluated morbidity and urban.

Table 2. Tobit Models Predicting Lower-Body Disability: Adults Aged 45 and Older at Baseline, National Health and Nutrition Examination Survey, 1971 to 1992

Independent Variables	Lower-Body Disability, Wave 2			Lower-Body Disability, Wave 4			
	Model 1	Model 2	Model 3	Model 1	Model 2	Model 3	Model 4
<i>Early Obesity</i>							
Obese at age 25	1.38 (.92)	—	—	2.22 (1.23)	—	—	—
Obese, Wave 1	2.29*** (.65)	—	—	3.36*** (1.00)	—	—	—
<i>Late Obesity</i>							
Obese, Wave 2	—	1.35* (.66)	—	—	2.06 (1.38)	—	—
Obese, Wave 3	—	—	—	—	2.99* (1.27)	—	—
Obese, Wave 4	—	—	—	—	.55 (1.06)	—	—
<i>Lifetime Body Weight</i>							
Chronic obesity (times) ^a	—	—	.73*** (.19)	—	—	1.10*** (.21)	.91*** (.19)
Ever-underweight ^b	.31 (.62)	.06 (.65)	.29 (.63)	2.87*** (.86)	3.07*** (.85)	3.23*** (.86)	3.16*** (.81)
Lower-body disability, Wave 2	—	—	—	—	—	—	1.01*** (.13)
Serious illness, Wave 1	.64* (.28)	.77** (.29)	.65* (.29)	1.48* (.59)	1.38* (.60)	1.28* (.58)	1.24* (.54)
Chronic illness, Wave 1	2.34*** (.39)	2.32*** (.40)	2.32*** (.39)	1.79** (.56)	1.59** (.58)	1.70** (.58)	.95 (.60)
Change in serious illness from Wave 1 to Wave 2	—	—	—	1.60*** (.47)	1.35** (.47)	1.40** (.47)	1.22** (.45)
Change in chronic illness from Wave 1 to Wave 2	—	—	—	1.96*** (.60)	1.81** (.62)	1.90** (.62)	1.29* (.59)
Ceased regular exercise by Wave 2	.79 (1.11)	.78 (1.13)	.79 (1.13)	-1.74 (2.75)	-1.83 (2.73)	-1.75 (2.77)	-1.54 (2.78)
Began regular exercise by Wave 2	-5.32*** (1.33)	-5.42*** (1.37)	-5.26*** (1.36)	-3.84 (2.90)	-4.12 (2.77)	-3.78 (2.86)	-1.55 (2.88)
Continued regular exercise, Wave 1 to Wave 2	-8.04*** (1.08)	-8.25*** (1.10)	-8.09*** (1.10)	-6.49** (2.42)	-6.41** (2.39)	-6.35** (2.45)	-3.36 (2.41)
Age	.06 (.08)	.05 (.08)	.06 (.08)	-.08 (.18)	-.04 (.19)	-.04 (.19)	.06 (.17)
Female	2.90*** (.91)	3.05*** (.91)	2.94*** (.91)	2.25 (1.20)	1.95 (1.21)	2.15 (1.21)	.96 (1.09)
Black	-3.14** (1.04)	-3.12** (1.06)	-3.09** (1.06)	-.003 (1.03)	.03 (1.03)	-.04 (1.04)	.56 (1.00)
Lives alone	.37 (.65)	.35 (.64)	.32 (.64)	-.08 (1.20)	-.23 (1.20)	-.14 (1.13)	.07
Education	-.04 (.17)	-.08 (.18)	-.06 (.18)	-.26 (.22)	-.25 (.23)	-.26 (.23)	-.33 (.20)
Income, previous wave ^c	-.05 (.16)	-.05 (.16)	-.05 (.16)	-.04 (.24)	-.08 (.25)	-.05 (.25)	.01 (.22)

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(Table 2 continued from previous page)

Independent Variables	Lower-Body Disability, Wave 2			Lower-Body Disability, Wave 4			
	Model 1	Model 2	Model 3	Model 1	Model 2	Model 3	Model 4
Regular physician	-.44 (.62)	-.38 (.64)	-.41 (.64)	-.21 (1.25)	-.13 (1.24)	-.12 (1.26)	-.35 (1.15)
Medicaid	1.55 (1.04)	1.61 (1.06)	1.61 (1.04)	-1.44 (2.79)	-.94 (2.76)	-.94 (2.74)	-1.32 (2.70)
Private insurance	.30 (.69)	.29 (.71)	.29 (.70)	.96 (1.14)	.93 (1.10)	1.05 (1.12)	.73 (1.07)
Past smoker	.39 (.60)	.32 (.60)	.37 (.61)	.23 (.87)	.02 (.90)	-.16 (.88)	.36 (.78)
Current smoker	.64 (.49)	.50 (.50)	.61 (.49)	2.31** (.74)	2.19** (.72)	2.34*** (.73)	2.20*** (.66)
Selection effect for mortality (λ)	9.45*** (2.52)	9.41*** (2.51)	9.56*** (2.52)	5.59 (4.91)	5.17 (4.97)	5.47 (5.01)	3.00 (4.51)
Selection effect for non-response attrition (λ)	-.56 (6.11)	.30 (6.17)	-.63 (6.23)	10.03*** (3.40)	9.89** (3.42)	9.51** (3.41)	8.14* (3.20)
Intercept	3.69	4.52	3.69	1.11	-.72	-1.44	-16.51
Number of cases	2,940	2,940	2,940	1,897	1,897	1,897	1,889
F (Degrees of freedom)	27.20 (21, 449)	26.29 (20, 450)	27.37 (20, 450)	13.54 (23, 316)	13.60 (24, 315)	13.79 (22, 317)	14.41 (23, 316)
Sigma	7.57	7.63	7.60	10.35	10.26	10.29	9.73

Note: Model estimates are weighted to U.S. proportions and adjusted for clustering. Unstandardized slopes are presented; except where otherwise noted, numbers in parentheses are standard errors.

^a Variable range equals 0 to 5 for Wave 2 and equals 0 to 7 for Wave 4.

^b Five opportunities to be underweight by Wave 2; seven by Wave 4.

^c Income is lagged in each equation using the income value from the previous wave.

* $p < .05$ ** $p < .01$ *** $p < .001$ (two-tailed tests)

from Model 2 of immediate effects of obesity on lower-body disability. The evidence from Models 1 and 2 suggests the importance of the long-term consequences of obesity as a risk factor—lagged effects are observed in both equations. Obesity at age 25 was not significant by itself in predicting Wave 4 disability, but this may be due to its indirect effect through higher morbidity. Indeed, supplementary analyses regressing the two morbidity measures on the two measures of early obesity reveal that obesity at age 25 is significantly associated with serious illness, independent of Wave 1 obesity. The effect due to chronic obesity by Wave 4 is also clear in Models 3 and 4: Long-term obesity accelerates disability. Respondents who were ever underweight were more likely to have higher disability by Wave 4, probably a manifestation of becoming underweight in advanced age (the youngest re-

spondents by Wave 4 are 65 years of age).¹³ Both types of morbidity, and increases in each type, raise disability risk. Smoking has a lagged effect in raising lower-body disability risk by Wave 4. In addition, the role of nonrandom selection becomes important by Wave 4: Nonresponse attrition is related to higher disability risk.

The results of analyses for upper-body disability are presented in Table 3. Many of the findings are similar to those in Table 2 and do not require elaboration. Nevertheless, there are some noticeable differences in

¹³ A measure of chronic underweight was tested in supplementary analyses, but it did not alter the conclusions presented here. In models that included both chronic underweight and ever underweight, the former was not significant. In models substituting chronic-underweight for ever-underweight, the former was not consistently significant.

Table 3. Tobit Models Predicting Upper-Body Disability: Adults Aged 45 and Older at Baseline, National Health and Nutrition Examination Survey, 1971 to 1992

Independent Variables	Upper-Body Disability, Wave 2			Upper-Body Disability, Wave 4			
	Model 1	Model 2	Model 3	Model 1	Model 2	Model 3	Model 4
<i>Early Obesity</i>							
Obese at age 25	.64 (1.23)	—	—	1.44 (1.31)	—	—	—
Obese, Wave 1	1.44 (.74)	—	—	2.92** (1.01)	—	—	—
<i>Late Obesity</i>							
Obese, Wave 2	—	.28 (.64)	—	—	1.62 (1.33)	—	—
Obese, Wave 3	—	—	—	—	2.06 (1.66)	—	—
Obese, Wave 4	—	—	—	—	.42 (1.16)	—	—
<i>Lifetime Body Weight</i>							
Chronic obesity (times) ^a	—	—	.33 (.22)	—	—	.86*** (.22)	.72*** (.21)
Ever-underweight ^b	-.13 (.63)	-.39 (.64)	-.22 (.64)	2.67** (.96)	2.77** (.96)	2.93** (.97)	2.75** (.95)
Upper-body disability, Wave 2	—	—	—	—	—	—	1.38*** (.21)
Serious illness, Wave 1	.69* (.33)	.79* (.32)	.72 (.33)	1.40* (.61)	1.42* (.60)	1.30* (.60)	1.39* (.57)
Chronic illness, Wave 1	2.68*** (.43)	2.69*** (.43)	2.69*** (.43)	1.71** (.67)	1.62* (.67)	1.67* (.66)	1.00 (.67)
Change in serious illness from Wave 1 to Wave 2	—	—	—	.67 (.64)	.50 (.63)	.51 (.63)	.24 (.61)
Change in chronic illness from Wave 1 to Wave 2	—	—	—	2.79*** (.78)	2.69*** (.79)	2.76*** (.79)	2.27** (.71)
Ceased regular exercise by Wave 2	-.27 (1.21)	-.27 (1.23)	-.25 (1.23)	-3.28 (2.67)	-3.40 (2.68)	-3.39 (2.68)	.37 (2.89)
Began regular exercise by Wave 2	-6.01*** (1.60)	-6.15*** (1.60)	-6.01*** (1.61)	-2.87 (3.06)	-3.21 (2.96)	-2.98 (3.04)	2.05 (3.38)
Continued regular exercise, Wave 1 to Wave 2	-9.31*** (1.33)	-9.44*** (1.33)	-9.35*** (1.34)	-6.54** (2.45)	-6.56** (2.47)	-6.52** (2.48)	-.32 (2.75)
Age	-.13 (.63)	-.15 (.10)	-.14 (.10)	-.09 (.19)	-.07 (.19)	-.07 (.20)	.07 (.19)
Female	3.71*** (1.12)	3.93*** (1.10)	3.81*** (1.11)	3.63** (1.24)	3.42** (1.24)	3.60** (1.24)	2.96** (1.13)
Black	-4.43*** (1.18)	-4.47*** (1.20)	-4.44*** (1.19)	1.14 (1.26)	1.17 (1.27)	1.06 (1.28)	1.46 (1.31)
Lives alone	-.44 (.86)	-.44 (.85)	-.45 (.85)	-1.09 (1.17)	-1.16 (1.15)	-1.11 (1.16)	-.68 (1.06)
Education	-.17 (.21)	-.20 (.21)	-.19 (.21)	-.17 (.23)	-.18 (.24)	-.19 (.24)	-.17 (.22)
Income, previous wave ^c	.03 (.19)	.04 (.20)	.03 (.20)	.13 (.24)	.10 (.24)	.12 (.24)	.15 (.22)

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(Table 3 continued from previous page)

Independent Variables	Upper-Body Disability, Wave 2			Upper-Body Disability, Wave 4			
	Model 1	Model 2	Model 3	Model 1	Model 2	Model 3	Model 4
Regular physician	-.07 (.83)	-.01 (.83)	-.05 (.83)	-.65 (1.38)	-.54 (1.37)	-.56 (1.38)	-.37 (1.39)
Medicaid	.60 (1.30)	.60 (1.30)	.62 (1.30)	-2.24 (1.57)	-1.83 (1.59)	-1.86 (1.53)	-1.98 (1.37)
Private insurance	.82 (.87)	.80 (.87)	.80 (.87)	-.24 (1.03)	-.45 (1.04)	-.26 (1.04)	-.28 (1.00)
Past smoker	.05 (.74)	.04 (.75)	.04 (.75)	1.69 (.98)	1.50 (.99)	1.60 (.99)	1.51 (.92)
Current smoker	.80 (.77)	.69 (.78)	.75 (.78)	.95 (.71)	.84 (.71)	.92 (.71)	1.05 (.65)
Selection effect for mortality (λ)	10.27** (3.87)	10.39** (3.91)	10.38** (3.91)	10.79* (5.24)	10.36* (5.22)	10.75* (5.25)	10.03* (4.67)
Selection effect for non-response attrition (λ)	5.00 (7.65)	5.92 (7.63)	5.32 (7.70)	5.00 (3.45)	5.14 (3.42)	4.82 (3.46)	2.29 (3.59)
Intercept	6.72	7.68	7.05	-1.70	-2.43	-3.05	-26.62
Number of cases	2,939	2,939	2,939	1,906	1,906	1,906	1,898
F (Degrees of freedom)	12.44 (21, 449)	13.18 (20, 450)	12.80 (20, 450)	7.29 (23, 316)	8.07 (24, 315)	7.72 (22, 317)	7.89 (23, 316)
Sigma	8.00	8.02	8.02	9.63	9.60	9.60	9.09

Note: Model estimates are weighted to U.S. proportions and adjusted for clustering. Unstandardized slopes are presented; except where otherwise noted, numbers in parentheses are standard errors.

^a Variable range equals 0 to 5 for Wave 2 and equals 0 to 7 for Wave 4.

^b Five opportunities to be underweight by Wave 2; seven by Wave 4.

^c Income is lagged in each equation using the income value from the previous wave.

* $p < .05$ ** $p < .01$ *** $p < .001$ (two-tailed tests)

comparison with lower-body disability—the pattern here is less compelling. Obesity was not directly associated with upper-body disability in any of the models for Wave 2. For Wave 4, only Wave 1 obesity and chronic obesity are consequential. Obesity’s effect on lower-body disability was both immediate and sustained, but its impact on upper-body disability takes longer to develop. Chronic obesity is consequential but, even here, the effect is observed only at Wave 4.

Although Wave 1 illness was associated with upper-body disability at Wave 2 and Wave 4, change in serious illness was not consequential to upper-body disability at Wave 4. Maintaining a regular exercise program was associated with less upper-body disability; starting an exercise program did not reduce upper-body disability for these respondents. African American respondents had lower levels of disability at Wave 2, but

this advantage diminished by Wave 4 so that their disability resembled the level of white respondents.¹⁴

As a final step in the examination of the role of compensatory mechanisms, Table 4

¹⁴ The baseline sample ranged in age from 45 to 74, and these subjects were followed for 20 years. Thus, the cumulative effect of obesity on disability has a longer time to develop among the oldest subjects. Therefore, we performed supplementary analyses by separating the sample into middle-age persons (ages 45 to 55 at baseline) and older persons (56 or older at baseline) and reestimating the models. Results were consistent across the groups with two exceptions. First, ever being underweight had a greater impact on the disability of the older subsample. Second, in a few cases the early-life obesity measures were stronger predictors of disability in the middle-age subsample. The effects were still lagged, but the less distal measures had greater import.

Table 4. Tobit Models Predicting Lower-Body Disability and Upper-Body Disability for Obesity Transitions between Wave 1 and Wave 2: Adults Aged 45 and Older at Baseline, National Health and Nutrition Examination Survey, 1971 to 1992

Independent Variable	Lower-Body Disability				Upper-Body Disability			
	At Wave 2		At Wave 4		At Wave 2		At Wave 4	
	Coef.	(S.E.)	Coef.	(S.E.)	Coef.	(S.E.)	Coef.	(S.E.)
<i>Body Weight Status</i>								
Obese, Wave 1 and Wave 2	2.17**	(.71)	4.04***	(1.14)	.93	(.76)	3.23**	(1.09)
Became obese by Wave 2	.61	(1.11)	5.54***	(1.27)	-.13	(1.26)	3.98**	(1.36)
Became nonobese by Wave 2	3.14**	(1.07)	5.00***	(1.29)	2.46*	(1.11)	4.69**	(1.51)
Ever-underweight ^a	.31	(.62)	3.12***	(.84)	-.15	(.63)	2.89**	(.93)
Serious illness, Wave 1	.68*	(.27)	1.37*	(.60)	.71*	(.32)	1.33*	(.62)
Chronic illness, Wave 1	2.88**	(.90)	1.71**	(.59)	2.68***	(.42)	1.69**	(.67)
Change in serious illness from Wave 1 to Wave 2	—		1.35**	(.47)	—		.46	(.62)
Change in chronic illness from Wave 1 to Wave 2	—		1.93**	(.62)	—		2.79***	(.79)
Ceased regular exercise by Wave 2	.69	(1.11)	-1.43	(2.74)	-.38	(1.22)	-3.22	(2.68)
Began regular exercise by Wave 2	-5.35***	(1.34)	-3.24	(2.93)	-6.10***	(1.61)	-2.61	(3.11)
Continued regular exercise, Wave 1 to Wave 2	-8.06***	(1.07)	-6.10*	(2.40)	-9.30***	(1.33)	-6.37**	(2.47)
Age	.06	(.08)	-.04	(.18)	-.14	(.10)	-.06	(.19)
Female	2.88**	(.90)	1.88	(1.19)	3.74***	(1.11)	3.42**	(1.23)
Black	-3.06**	(1.02)	.23	(1.04)	-4.35***	(1.18)	1.25	(1.26)
Lives alone	.40	(.64)	-.28	(1.21)	-.44	(.85)	-1.17	(1.17)
Education	-.04	(.17)	-.25	(.22)	-.16	(.21)	-.17	(.23)
Income, previous wave ^b	-.06	(.16)	-.06	(.24)	.03	(.19)	.12	(.23)
Regular physician	-.45	(.63)	-.19	(1.24)	-.06	(.83)	-.67	(1.38)
Medicaid	1.51	(1.01)	-1.22	(2.82)	.50	(1.29)	-2.18	(1.61)
Private insurance	.34	(.70)	.96	(1.11)	.85	(.87)	-.30	(1.04)
Past smoker	.37	(.59)	.03	(.89)	.06	(.74)	1.57	(1.00)
Current smoker	.64	(.49)	2.25**	(.71)	.79	(.77)	.92	(.71)
Selection effect for mortality (λ)	9.34***	(2.53)	4.79	(4.89)	10.20**	(3.86)	10.24	(5.24)
Selection effect for nonresponse attrition (λ)	-.48	(6.10)	9.99**	(3.40)	5.20	(7.63)	5.00	(3.44)
Intercept	3.81		-1.31		7.98		-3.11	
Number of cases	2,940		1,897		2,939		1,906	
F (degrees of freedom)	27.08 (22, 448)		13.31 (24, 315)		12.82 (22, 448)		8.04 (24, 315)	
Sigma	7.56		10.25		7.98		9.57	

Note: Model estimates are weighted to U.S. proportions and adjusted for clustering. Unstandardized slopes are presented; except where otherwise noted, numbers in parentheses are standard errors.

^a Five opportunities to be underweight by Wave 2; seven by Wave 4.

^b Income is lagged in each equation using the income value from the previous wave.

* $p < .05$ ** $p < .01$ *** $p < .001$ (two-tailed tests)

presents results for both disability outcomes where obesity transitions between Wave 1 and Wave 2 are also identified. In this analysis, we test for the simultaneous effects of the two compensatory processes: risk-factor elimination and countervailing mechanism. Imagining a 2×2 table defining obesity at the first two waves yields four categories of weight change. Three categories are listed in Table 4, with persons not obese at either wave serving as the reference group.

The results for lower-body disability reveal that respondents in two of the three obesity-status categories manifested more disability than did those who were not obese at either wave. It is not surprising that persons who were obese at both waves showed greater Wave 2 lower-body disability, but note that persons who became non-obese by Wave 2 also manifested greater disability relative to those who were not obese at either wave. Thus, exiting the risk state of being obese did not reduce disability—a finding that speaks to the enduring effects of long-term risk over the life course. Becoming obese by Wave 2 was not associated with more Wave 2 disability, but it was associated with Wave 4 disability, again suggesting lagged effects.

For upper-body disability, a modest effect was observed for exiting the obese state, but it was not in the anticipated direction. Although the effect is not strong, it likely indicates the long-term and enduring effects of obesity and the sequelae of failing health (Launer et al. 1994). In contrast to Wave 2, each obesity category was associated with increased upper-body disability by Wave 4. If a respondent was ever obese, there is a greater risk of Wave 4 upper-body disability. Maintaining regular exercise was consistent in reducing disability.

DISCUSSION

Cumulative disadvantage theory has garnered considerable attention in recent years, but relatively few tests of it have made use of longitudinal data. This study has the advantage of permitting a long-term, longitudinal examination of how risks can accumulate to accelerate health decline. At the same time, this study does have its limitations. Most notably, the analysis rests on respon-

dent recall of body weight in early life, which may lead to an underestimation of the effect of obesity on disability. Nevertheless, the results provide some support for cumulative disadvantage theory, but also suggest some ways in which the theory may be revised.

Obesity is consistently related to lower-body disability, but is less consistently related to upper-body disability. Lower-body disability is more consequential to ambulation and social activity. In this sense, it is probably the more consequential type of disability, but also because it requires more extensive interventions. Although the results are more substantial for lower-body disability, the findings also show consistent effects of chronic obesity on upper-body disability by Wave 4.

In support of cumulative disadvantage theory, the effects of obesity on health uncovered here are largely long-term, with lags of more than 10, 20, and 50 years. The impact of some risk factors is immediate, but there is extensive evidence from these analyses that obesity is consequential over the long haul. Obesity is associated with more upper-body and lower-body disability, but the effects are, for the most part, long-term rather than immediate. The analyses show that obesity alters the life course in an enduring way—evidence for a scarring effect. Moreover, the effects of chronic or long-term obesity are consistently related to disability. Obesity in adulthood is an important indication of health inequality over the life course, and the results presented here show that exceeding the threshold of 30 BMI is associated with premature disability.

Sociological study of the life course has identified “turning points” that shape socioeconomic achievement, health, and well-being (Elder 1969, 1985; Sampson and Laub 1996). Historical events, such as military service and economic depression, can shape the life course of cohorts in meaningful ways, but obesity represents another turning point for understanding health and well-being. It may at first appear to be a more individualistic experience, but social change in the prevalence of obesity as well as life course patterns for the development of obesity suggest otherwise. Indeed, the current obesity epidemic is socially structured as

evidenced by the food, beverage, and diet industries (e.g., Robertson, Brunner, and Sheiham 1999; Young and Nestle 2002).

Turning points can emerge at any point in the life course. On the basis of cumulative disadvantage theory, infancy and childhood must come into the scope of the sociological study of health. Although the data were limited, we performed supplementary analyses that included measures of childhood body weight to determine whether it was consequential to health. Those analyses revealed that childhood overweight was not, in and of itself, consequential to adult health. The conceptualization of risk over the life course may need to consider childhood risks as distinct from those occurring after adulthood or sexual maturity. Cumulative disadvantage theory anticipates childhood disadvantage to be consequential, but the conclusion may not always be that simple (Ferraro, Thorpe, and Wilkinson 2003).

Although research to date on cumulative disadvantage and health has yielded inconsistent results, our study reveals that obesity in a population is associated with increased heterogeneity among adults, a finding consistent with the work of Ross and Wu (1996). Research like ours situates the experience of disability within a matrix of risk factors, compensating mechanisms, turning points, and chronic stressors over the entire life course. The likelihood of experiencing each of these is embedded in the larger social organization, leading some to consider the life course itself as a structure for the development of inequality (Irwin 2001).

Obesity reveals much about the physiological state of the individual, and findings from the present research show that crossing the 30 BMI threshold has enduring effects on health. More generally, our results add to the growing body of literature showing the importance of using biological information when available in survey research (Booth and Dabbs 1993; Conley and Bennett 2000; Jousilahti et al. 2000). Although debates about the appropriateness of some biological measures in survey research continues (Finch and Vaupel 2001), assessing body mass index is a relatively unobtrusive process that yields much in understanding the life course.

Further, when considering much of the previous work on cumulative disadvantage

and health, we found that obesity is an important mediating variable between socioeconomic status and health. Persons of lower socioeconomic status are more likely to be obese, and the effect of obesity reduces the independent association between socioeconomic status and health measures. Much previous research on cumulative disadvantage and health does not consider obesity as a mediating variable between socioeconomic status and health (e.g., Ross and Wu 1996), but the results from the present analysis demonstrate the importance of considering obesity, or some measure of body mass, in such analyses.

Although cumulative disadvantage theory recognizes the possibility of inexorable effects due to risk factors, we believe more attention should be focused on compensatory processes. Two compensatory processes were considered here. One typically thinks that exiting a risk state will reduce the effects of risk factors. Here, risk-factor elimination would mean exiting the obese state. However, exiting the obese state over the first two survey waves did not undo the health disadvantage due to obesity. For the most part, obesity at any time during adulthood heightened health problems, but becoming non-obese did not eliminate them: People who exited the obese state continued to manifest higher-than-average disability, and their health ratings were fairly similar to those who were obese at Waves 1 and 2.

Beyond exiting a risk state, another compensatory process may occur through countervailing mechanisms. Regular exercise is one example of a countervailing mechanism in our analysis, and it emerged as an important health behavior that shapes disability. Maintaining or beginning regular exercise was associated with less lower-body and upper-body disability. Although risk-factor elimination is often the presumed mode of intervention, these results show that intervention via a countervailing mechanism may be equal or superior to risk-factor elimination. Substantively, the compensatory mechanism occurred via exercise, not through weight loss, and this demonstrates the health benefits of exercise, even among overweight or obese persons (Farrell et al. 2002). Previous research shows that exercise benefits a range of psychosocial aspects of

health, such as energy levels and "feeling good" (Myers et al. 1999), but the present research shows noticeable effects on more physical dimensions of health such as functional disability. It may also be that exercise builds physical strength, and that strength is more important than physical size in shaping disability.

This finding is important for tempering cumulative disadvantage theory. There is some evidence of the permanency of effects through the accumulation of risk factors as manifested in chronic obesity. This finding is consistent with a number of studies on how early disadvantage raises health risks across the life course (e.g., Kuh and Ben-Shlomo 1997). At the same time, we found no discernible compensatory effect from exiting the risk state of obesity. In this sense, applying cumulative disadvantage theory suggests that one anticipate that compensation might arise from a related phenomenon rather than from eliminating the risk state under consideration—what some have referred to as "experience" (Dannefer 1988a; Neugarten 1964). It is important to look beyond the manifest risk factor to related phenomena and life changes that the person is experiencing. For obesity, it is exercise in lieu of weight reduction, but there is applicability for other fields of study. For example, in studies of divorce recovery, exiting the divorced state through remarriage may not completely undo the effects associated with earlier marital troubles (Wilmoth and Koso 2002). One would want to examine the surrounding risk factors and resources to determine if remarriage has the potential to ameliorate some of the earlier problems. If not, the earlier risks may only be exacerbated.

Cumulative disadvantage theory offers sociologists many opportunities to organize both empirical generalizations and hypotheses in studying how inequality develops over the life course. Investigators must consider whether the early formulations of the inevitable consequences of risk are worth retaining in light of mounting evidence that favors compensatory mechanisms (Elman and O'Rand forthcoming). It seems appropriate to modify tenets regarding the inevitable consequences of risk to allow for human agency and the moderation of risk fac-

tors through changing social arrangements. Indeed, failure to consider compensatory mechanisms may lead to overstating the impact of early disadvantage. There are certainly instances of early risk factors that have irreversible effects on life chances and well-being. At the same time, there are enough instances of the effectiveness of compensation to temper cumulative disadvantage theory. The theory should explicitly incorporate compensatory mechanisms and the reversibility of effects due to changes in risk-factor exposure.

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