Journal of Gerontology: SOCIAL SCIENCES
2003, Vol. 58B, No. 2, S110–S119

The Life Course of Severe Obesity: Does Childhood Overweight Matter?

Kenneth F. Ferraro,[1,2] Roland J. Thorpe, Jr.,[2,3] and Jody A. Wilkinson[4]

[1]Department of Sociology, Purdue University, West Lafayette, Indiana.
[2]Gerontology Program, Purdue University, West Lafayette, Indiana.
[3]Department of Veterinary Pathobiology, Purdue University, West Lafayette, Indiana.
[4]Arizona Cancer Center, University of Arizona, Tucson.

Objectives. A life course perspective is used to examine the epidemiology of severe obesity in adulthood, defined as a body mass index $\geq$ 35 kg/m^2.

Methods. Data from adults in the National Health and Nutrition Examination Survey I: Epidemiologic Followup Study, including their reports of childhood overweight, were used to examine the risk of severe obesity and mortality over 20 years (N = 6,767). All multivariate models control for age, sex, race, smoking, and socioeconomic resources.

Results. Childhood overweight was significantly associated with severe obesity for both women and men, although the effect was stronger for men. The prevalence of severe obesity was highest between 45 and 64 years of age than for persons under 45 or over 65, and higher for African Americans than White Americans. Childhood overweight was associated with lower mortality risk for women, but not for men. Mortality risk was higher for persons with severe obesity (relative risk = 1.571, 95% confidence interval = 1.335–1.849, $p < .001$).

Discussion. The findings demonstrate the importance of childhood overweight as a risk factor for severe obesity over the life course. Nevertheless, overweight children who did not become severely obese were not at greater risk of mortality.

AVERAGE body mass index (BMI) has increased substantially in recent years, prompting some to describe the growing prevalence of obesity (BMI $\geq$ 30 kg/m^2) in the United States as an epidemic (Mokdad et al., 1999). Estimates vary across studies of the U.S. population, but between 11%–14% of children (Troiano & Flegal, 1998) and about 18%–25% of adults are now considered obese (Flegal, Caroll, Kuczmarski, & Johnson, 1998; Mokdad et al., 1999; Strauss & Pollack, 2001). The disease burden of obesity is substantial, with the number of deaths attributed to it estimated at 280,000 annually (Allison, Fontaine, Manson, Stevens, & VanItallie, 1999; Must et al., 1999).

Not only is obesity associated with significant health risks, but also the National Heart, Lung, and Blood Institute's (NHLBI's) Guidelines (1998) identified three classes of obesity to reflect varying levels of risk: Class I (BMI $\geq$ 30 and BMI $<$ 35), Class II (BMI $\geq$ 35 and BMI $<$ 40), and Class III (BMI $\geq$ 40). Class I obesity raises the risk of many diseases, including type 2 diabetes, hypertension, and heart disease, but there is emerging evidence that risk for some diseases rises further with Class II and Class III obesity (NHLBI, 1998). Consistent with Allison et al. (1999), severe obesity is defined here as including Class II and Class III obesity (BMI $\geq$ 35). Although the prevalence of severe obesity in the United States has increased (Kuczmarski, Flegal, Campbell, & Johnson, 1994), relatively little is known about the antecedents and consequences of a BMI $\geq$ 35. To illustrate what is meant by severe obesity, a person 68 inches tall would need to weigh at least 230 pounds.

At the same time that the prevalence of both adult and childhood obesity has increased in the United States, there have also been important discoveries regarding a life course approach to the epidemiology of chronic disease (Kuh & Ben-Shlomo, 1997). Research has accumulated in recent years demonstrating the importance of early life for the development of risk factors and chronic disease in adulthood (Blackwell, Hayward, & Crimmins, 2001; Preston, Hill, & Drevenstedt, 1998). The present research builds on this line of inquiry by using a life course perspective to examine the prevalence of severe obesity in adulthood. The risks of both severe obesity and mortality are examined with data from a national sample of adults that includes retrospective data on childhood overweight. Special attention is given to gender differences in the contribution of childhood overweight on the outcomes.

Life Course Approach to Severe Obesity

The life course approach emphasizes that the aging process is closely related to traits or characteristics displayed earlier in the life course. This does not imply stability per se, because human agency operates in light of opportunities and constraints over the life course. Personal change is anticipated over the life course, but the nature of the change is conceptually linked with earlier experiences, abilities, and resources (Dannefer, 1987; Elder 1995). Life-course epidemiologists focus on the concept of a *chain of risk*, noting that adversity in early life alters the life course, most often leading to adverse outcomes (Preston et al., 1998). There is also an acquired-immunity thesis, whereby early exposure to diseases, especially infectious diseases, or adverse conditions may reduce the risk of later illness.

In the context of overweight and obesity, there are two groups of studies that examine chains of risk associated with body weight. The first group of studies examines the link between body weight during infancy and childhood and body weight in

adulthood. The second examines variation in BMI during adulthood.

Infancy and childhood.—Previous research reveals a fairly weak link between infant weight and adult BMI (Parsons, Power, Logan, & Summerbell, 1999). On the other hand, there are fairly consistent findings emerging from the literature showing that childhood overweight heightens the risk of staying that way throughout the life course. Previous research suggests that the link is stronger for children who are overweight during adolescence, compared with early childhood (Guo, Roche, Chumlea, Gardner, & Siervogel, 1994; Serdula et al., 1993) and for those with higher childhood body weight (Troiano & Flegal, 1998). Some studies show that the positive association between childhood overweight and adult obesity is stronger for females than males (Guo et al., 1994), whereas other research finds a stronger link for males (Casey, Dwyer, Coleman, & Valadian, 1992).

These studies have helped greatly to identify the link between childhood overweight and adult obesity, but they contain several methodological limitations that are relevant to the present investigation. First, there are many excellent studies of White subjects (Casey et al., 1992; Guo et al., 1994; Must, Jacques, Dallal, Bajema, & Dietz, 1992), but relatively few that give adequate consideration to the link between childhood overweight and adult obesity in minority populations (Freedman et al., 1987). Second, although there are many excellent studies based on clinical samples (e.g., DiPietro, Mossberg, & Stunkard, 1994), the use of health services is related to minority group and socioeconomic status—groups known to be at higher risk of obesity. Third, Serdula et al. (1993) assert that "longer follow-ups are needed to establish the tracking of childhood obesity into middle-age, when the prevalence of obesity is greatest" (p. 174). There have been some long-term studies, but mostly on all White (Guo et al., 1994) or clinical samples (DiPietro et al., 1994). Fourth, Serdula et al. (1993) point out that most of the panel studies do not systematically account for sample attrition, and this could systematically bias results.

Adulthood.—A second body of literature addresses the prevalence and persistence of obesity in adulthood. It is fairly well known that BMI increases with age through the adult years, except during later life when a leveling or decline occurs. Flegal and colleagues (1998) used repeated cross-sectional surveys to show that both obesity and severe obesity increase sharply from 20–29 to 30–39 and continue to rise until 50–59 years of age. Panel data, including analyses based on the data used for the present study, confirm this pattern (Must & Strauss, 1999). Although the age of the peak prevalence of obesity varies for men and women—women peak later—both sexes show lower prevalence of obesity by 70 years of age. Some of this may be from mortality, and some is probably from incident morbidity (Losonczy et al., 1995).

Most of the research on BMI in adulthood is based on cross-sectional designs, but panel studies show that the correlations between BMI during adulthood are greater than those between childhood and adult BMI (Casey et al., 1992; DiPietro et al., 1994). Also, once a person becomes obese, their likelihood of remaining in that state is greater than exiting it alive, especially among women. The rise in obesity in the 30s is often coincident with exiting the "marriage market." For many women, it also follows childbearing.

Mortality risk.—Examining continuities in body weight over the life course requires simultaneous consideration of mortality. Although there remains some debate on the precise shape of the relationship between BMI and mortality (U- or J-shaped), most studies find that premature death from obesity is more likely for both men and women (Durazo-Arvizu, McGee, Cooper, Liao, & Luke, 1998). Although limited attention has been given to the unique mortality risk associated with severe obesity in adulthood, the growing prevalence of severe obesity and the new clinical guidelines from the NHLBI underscore the need for further study.

The findings are more equivocal when examining the link between childhood overweight or obesity and adult mortality. Several studies show that overweight children manifest higher mortality risk in adulthood, but the subjects on which these findings are based include clinical samples (DiPietro et al., 1994), middle-class children (Must et al., 1992), and Dutch men (Hoffmans, Kromhout, & Coulander, 1989). The chain of risk between childhood overweight and adult mortality is probably because of blood lipids that promote hardening of the arteries. By contrast, other Dutch studies, some done on men only, show that overweight children have *lower* mortality risk, especially after age 30, perhaps suggesting the value of energy reserves during development (Sonne-Holm, Sorensen, & Christensen, 1983; Sorenson & Sonne-Holm, 1977). Inconsistent findings suggest the need for further research, especially with long-term follow-up periods. In addition, several studies fail to control for adult BMI (e.g., Nieto, Szklo, & Comstock, 1992; Sonne-Holm et al., 1983). A life course approach to the subject, however, stresses the importance of linking childhood body weight to both adult body weight and mortality risk.

Research questions.—Although there has long been interest in whether childhood overweight is related to obesity, we are unaware of any study that examines whether childhood overweight is related to severe obesity in adulthood (BMI $\geq$ 35). The present research is designed to provide a long-term view of the antecedents of severe obesity and consequences of it on mortality. The life course perspective suggests three major research questions:

1. Does childhood overweight increase the likelihood of severe obesity in adulthood?
2. At which ages of the life course is severe obesity most prevalent?
3. Do childhood overweight and severe obesity in adulthood independently increase mortality risk in adulthood?

Methods

Sample

Data from the *National Health and Nutrition Examination Survey I* (NHANES I) and its *Epidemiologic Followup Study* (NHEFS) are used in this research (Cox et al., 1997). The baseline NHANES I was conducted in 1971–1975. The sampling design was a multistage, stratified, probability sample

of noninstitutionalized persons ages 25–74 years of age. This study makes use of data from the baseline survey and three follow-ups (1982–1984, 1987, and 1992).

The analyses were completed on the NHEFS subsample that was administered the "detailed component," including the Health Care Needs Questionnaire at baseline ($N = 6,913$). The sample used in this study is composed of 5,955 White and 878 Black respondents at baseline. Women who gave birth 1 year before or after any survey wave were omitted (resulting in a sample of 6,767). The percentage of cases traced through follow-ups was excellent (88% of survivors at Wave 2 and 89% of survivors at Waves 3 and 4). The number of cases lost to mortality, tracing, and refusal to participate is 1,644 by Wave 2 and 2,696 by Wave 4. All analyses presented herein are based on the weighted sample and have been adjusted by Taylor-linearization procedures in Stata 7.0 to account for the multistage sampling design.

Measurement of Body Weight

For the first two survey waves, weight and height were measured by research staff. At the baseline survey, weight was measured by a Toledo self-balancing scale to ¼ pound, with participants wearing examination slippers and gown. Height was measured with a level platform and attached measuring rod. For Wave 2, weight was measured with a Health-O-Meter scale placed on a hard surface after calibration to zero (shoes and heavy outer clothing removed). For Waves 3 and 4 of NHEFS, weight was based on respondent report. Although self-reported weight measures are closely related to physical measurements, they may underestimate the tails of the weight distribution (Bowman & DeLucia, 1992). Some of the analyses presented herein do not require use of the last two weights. For those that do, however, it should be recognized that the relationships considered may be underestimated.

BMI was defined as kilograms/meters^2, and severe obesity was defined as BMI $\geq$ 35 (Class II and Class III obesity according to the NHLBI Guidelines; coded 1 = severe obesity, 0 = otherwise). Greater muscle mass among manual laborers or some athletes can result in a BMI indicative of overweight, but it is unlikely that the definition of severe obesity used here (BMI $\geq$ 35) will be biased by differences in muscle mass. As shown in Table 1, nearly 5% of the respondents were severely obese at the baseline. About 9% were severely obese at least one time during the 20-year study.

Childhood overweight was measured retrospectively by self-report. It is preferable to have measured weight and height, but such measures are not available. Moreover, although BMI is a widely used measure of fatness in adulthood, it is not as reliable for measuring fatness in children, probably because of gender differences in maturation and the fact that BMI is not independent of height (Troiano & Flegal, 1998). Childhood overweight is 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?" Respondents who reported that they were either chubby (10.1%) or very heavy (1.0%) were classified as overweight for the purposes of this analysis. Although the response categories are stated in a fairly nonoffensive way, it is possible that there may be some underreporting of overweight associated with this retrospective question. Fortunately, the question was asked about a specific 2-year age range as opposed to a longer one.

Measurement of Covariates

The remaining independent variables span a broad range of risk factors for obesity, either directly or indirectly. Demographic variables include age, female, Black, lives alone, and widowed. All binary variables were coded zero and one, with one equal to the name of the variable. Variables related to socioeconomic resources included education (8 categories) and household income (12 categories) and three binary variables related to medical care access: availability of private health insurance, Medicaid status, and regular physician. Of all of the socioeconomic resource variables, education is probably most correlated with early life resources (parental education and income were not measured). Supplementary analyses treating each category of education (and income) as a separate binary variable, with alternative reference groups, were performed, but did not alter the conclusions presented herein.

Current and past smokers were identified by self-report of consumption of cigarettes, cigars, and pipe tobacco at the time of the interview and during one's lifetime. Respondents were also asked to rate how much exercise they get "in your usual day" from both recreational and nonrecreational activities. A binary variable for limited activity was created to identify respondents who were quite inactive in both questions (1 = limited activity; 0 = otherwise). Some indicator of physical activity is important for the study of severe obesity, but it should be noted that this measure is a crude index of activity that could be from limited function or lifestyle.

Mortality

Vital status was determined at the follow-up surveys for all traced respondents, and death certificates confirmed death for 98.7% of the deceased. Brief interviews were conducted with proxies of deceased respondents. In addition, matches were made for all participants in the baseline survey to the National Death Index, the Social Security Administration Mortality File, and the enrollment file of the Health Care Financing Administration (Cox et al., 1997). Date of death was obtained for 1,936 decedents, so continuous-time event history models were applied.

For the mortality analyses, morbidity was also included as a risk factor. Morbidity measures were derived from a checklist based on the following question: "Has a doctor ever told you that you have ... hypertension or high blood pressure?" (36 conditions presented). Self-reported measures have been shown to have considerable predictive validity, especially when referencing a physician's evaluation of disease (Ferraro & Farmer, 1999). Each condition was coded as a binary variable (1 = present; 0 = otherwise) and then classified into those that were life-threatening or serious and all remaining conditions. Serious conditions included cancer, diabetes, heart failure (attack or trouble), hypertension, and stroke. Examples of chronic nonserious conditions included arthritis, asthma, cataracts, gout, psoriasis, and ulcer. The serious and chronic nonserious conditions were then summed separately.

Means and standard deviations of all variables for the total sample, and by severe obesity at the baseline, are presented in Table 1. Several additional variables, such as rural residence and women's menopause status, were considered in preliminary

Table 1. Means and Standard Deviations of Variables in the National Health and Nutrition Examination Survey I (1971–1975): Total Sample and by Severe Obesity[a]

Variables	Total (N = 6,767)	Severe Obesity: BMI ≥ 35 at Wave 1 (N = 325)	Otherwise: BMI < 35 at Wave 1 (N = 6,435)
Severe obesity	0.042		
Age (24–77)[b]	46.462 (0.224)	47.806 (0.908)	46.329 (0.232)
Childhood overweight	0.118	0.367	0.107***
Female	0.521	0.731	0.512***
Black	0.102	0.254	0.095***
Lives alone	0.109	0.149	0.108*
Widowed	0.072	0.147	0.068***
Education (7 = graduate school)	3.853 (0.034)	3.346 (0.103)	3.876 (0.035)***
Income (12 = $25,000+)	7.900 (0.062)	6.797 (0.181)	7.954 (0.062)***
Private medical insurance	0.845	0.740	0.850***
Medicaid	0.030	0.059	0.029**
Regular physician	0.848	0.885	0.847
Smoker	0.448	0.377	0.451*
Past smoker	0.256	0.213	0.258
Limited activity	0.077	0.145	0.074***
Serious illness (0–5)	0.340 (0.009)	0.720 (0.050)	0.323 (0.009)***
Nonserious illness (0–4)	0.266 (0.006)	0.417 (0.037)	0.259 (0.006)***
Long-term severe obesity (0–4)	0.201 (0.727)	2.929 (0.106)	0.086 (0.008) ***
Deceased by Wave 4	0.286	0.381	0.282**

Note: BMI = body mass index.

[a]The number of cases varies because of missing data. Severe obesity and all other binary variables are coded 0 and 1 (standard deviations of binary variables are omitted). The mean of a binary variable scored zero and one reflects the percentage of cases with that attribute. Tests of significance are for differences by severe obesity, t-test or chi-square.

[b]The prevalence of severe obesity at baseline is distributed across age categories as follows: <35, 3.68; 35–44, 3.43; 45–54, 4.62; 55–64, 5.59; 65–74, 4.11.

*$p \leq .05$; ** $p \leq .01$; ***$p \leq .001$.

analyses, but deleted from the final analysis because they were nonsignificant in multivariate models.

Analytic Plan

The analysis is divided into three stages; all multivariate models control for age, sex, race, smoking, and socioeconomic resources (see tables for exact specifications). The first was to examine the risk of severe obesity at the baseline interview. Logistic regression models were estimated for the total sample and separately for men and women. These analyses examine the prevalence of severe obesity with special attention to the role of childhood overweight.

The second stage of the analysis was to examine the likelihood of severe obesity across the four waves of the NHEFS. Severe obesity was identified at each subsequent wave, and a count variable was formulated to assess 20-year risk of severe obesity or long-term severe obesity. This variable ranges from 0 (90.7%) to 4 (1.9%), in which 4 indicates the subject was severely obese at all surveys. The logic of this stage of the analysis is to identify the antecedents of chronic severe obesity. [After preliminary analysis with Poisson models, the final estimation was with a negative binomial regression model that is more appropriate for the overdispersion of the long-term severe obesity measure (Long, 1997).]

Although case tracing and reinterview rates were high, attrition in longitudinal analyses may influence sample estimates of predictor variables and lead to biased estimates. Thus, selection bias models were used to correct parameter estimates for differential selectivity because of death, refusal to participate, or inability to trace (Heckman, 1979). The procedure was to first estimate two probit models, distinguishing those who survived from those who died and those who participated from those who were lost to follow-up. (Mortality was predicted by age, gender, race, self-rated health, serious illness, and household income; nonresponse was predicted by age, race, household income, missing on income, rural residence, and depression.) The second step was to use the probit results to create two selection (hazard) instruments based on the inverse Mills ratio, each with at least one instrumental variable (Maddala, 1983). Self-rated health served as the instrument for the probit model of alive ($p < .0001$). Three instrumental variables for the nonresponse equation were: missing on income at W1 ($p < .05$), depression ($p < .001$), and rural residence ($p < .0001$). The selection instruments were then added to the model to differentiate attrition caused by mortality from that caused by nonresponse.

The final stage of the analysis examined mortality during the 20-year study. As shown in Table 1, mortality over the study was higher for persons who were severely obese at the first interview. The mortality analysis uses Cox models to examine the net effects of severe obesity in adulthood and childhood overweight on the risk of death among men and women.

Results

Table 2 presents the findings for the logistic regression of severe obesity at the baseline interview. (Full models are presented in the tables, but several reduced-form models were also estimated to inform the analysis and discussion. The latter are available on request from the first author.) Results for the total sample (two models) are presented first, followed by estimates for women and men separately. Model 1 for the total sample contains all of the independent variables, and Model 2 adds a qua-

Table 2. Logistic Regression Analyses for Severe Obesity (Body Mass Index ≥ 35) in NHANES I (1971–1975): Odds Ratios and Confidence Intervals for Total Sample and by Gender

	Model 1 (N = 6,728)		Model 2[a] (N = 6,728)		Women (N = 3,623)		Men (N = 3,105)		
Independent Variables	OR	CI	OR	CI	OR	CI	OR	CI	Wald χ^2
Age	.998	(.987–1.010)	1.085*	(1.002–1.174)	1.109*	(1.000–1.230)	1.074	(.939–1.229)	.140
Age^2			0.999*	(.998–.999)	.998	(.997–1.000)	.999	(.997–1.000)	.001
Childhood overweight	5.256***	(4.000–6.907)	5.221***	(3.974–6.86)	4.194***	(2.985–5.893)	8.648***	(5.256–14.218)	5.56*
Female	2.169***	(1.479–3.181)	2.133***	(1.455–3.127)					
Black	2.671***	(1.923–3.712)	2.598***	(1.869–3.613)	2.657***	(1.828–3.863)	2.359*	(1.050–5.299)	.069
Lives alone	1.012	(.619–1.653)	1.033	(.638–1.674)	.940	(.512–1.725)	1.361	(.514–3.601)	.401
Widowed	1.345	(.807–2.241)	1.429	(.853–2.393)	1.355	(.776–2.365)	.720	(.150–3.454)	.555
Education	.880*	(.791–.978)	.885*	(.795–.984)	.850*	(.738–.980)	.918	(.736–1.145)	.328
Income	.944	(.891–1.000)	.931*	(.877–.989)	.927*	(.865–.994)	.930	(.817–1.059)	.002
Private medical insurance	.687*	(.500–.943)	.728	(.524–1.010)	.684	(.444–1.054)	1.033	(.419–2.548)	.656
Medicaid	1.076	(.602–1.923)	1.139	(.633–2.050)	1.247	(.658–2.361)	.616	(.118–3.211)	.611
Regular physician	1.457	(.958–2.217)	1.407	(.920–2.150)	1.339	(.845–2.123)	1.722	(.861–3.443)	.352
Smoker	.749	(.537–1.044)	.724	(.521–1.007)	.853	(.580–1.253)	.560	(.295–1.065)	1.213
Past smoker	1.231	(.831–1.824)	1.208	(.811–1.799)	1.461	(.882–2.418)	1.072	(.581–1.981)	.583
Limited activity	1.641*	(1.045–2.577)	1.618*	(1.029–2.544)	1.554*	(1.001–2.413)	1.724	(.673–4.413)	.038
F-value	26.50		24.75		13.28		9.46		
df	14		15		14		14		

Note: NHANES I = National Health and Nutrition Examination Survey I; OR = odds ratio; CI = 95% confidence interval.
[a]Model 2 is Model 1 + the Age^2 term.
*$p \leq .05$; ***$p \leq .001$.

dratic term for age to account for the possibility that the odds ratio (OR) varies across levels of age. Comparing chi-square values for the two models, with 1 *df*, reveals that Model 2 is a better-fitting model. Although age was not related to severe obesity in Model 1, both the linear and squared terms are significant in Model 2, showing that the relationship between age and severe obesity is nonlinear. This reveals that the likelihood of severe obesity increases from early through middle adulthood and then declines slightly in advanced ages, perhaps reflecting disease progression in later life. Supplementary analyses with five categories of age reveal that severe obesity at any of the survey waves is very unlikely after age 75. The peak prevalence for severe obesity is between 45 and 64 years of age.

The effect of childhood overweight is consistently strong across both models. Respondents who were overweight at 12 or 13 years of age were about 5.2 times more likely than those not overweight as a child to be severely obese at the NHANES I interview. The ORs in Model 2 for women [OR = 2.133, 95% confidence interval (CI) = 1.455–3.127] and African Americans (OR = 2.598, 95% CI = 1.869–3.613) were more than double those for men and White respondents, respectively. Severe obesity was also more likely among persons with limited education and income. The education effect is important because it is, in part, an indicator of early life resources, and the effect of childhood overweight is the net of such resources. (In supplementary analyses, we also tested the possibility that childhood overweight is related to adult income, but found the relationship to be nonsignificant.) Limited activity was positively associated with severe obesity.

Separate sample analyses to examine whether the effects of any variables are significantly different for women and men are also presented in Table 2. The Wald chi-square statistic is presented to identify which parameter estimates are significantly different by gender. The results indicate only one instance of statistical interaction. The effect of childhood overweight is significant for both men and women, but its effect on severe obesity is greater for men (OR = 8.648, 95% CI = 5.256–14.218). [Unlike linear regression, coefficients in logit models are confounded with residual variation across the groups. Tests of residual variation, however, show that this difference across gender is not from unobserved heterogeneity across the groups (Allison, 1999)].

The next stage of the analysis is to examine the likelihood of long-term or chronic severe obesity over the course of the study. To begin, it is clear from the NHEFS data that there is considerable stability in body weight after one crosses the 35 BMI threshold: 65% of subjects who were severely obese at baseline were severely obese at the second survey wave. Moreover, 65% of the subjects who were severely obese at Wave 2 were severely obese at the third wave, and nearly 75% of the subjects who were severely obese at Wave 3 were severely obese at final follow-up. By contrast, a small percentage of persons who were not severely obese at baseline became so at later waves. For instance, 1.9% of men and 2.9% of women became severely obese between Waves 1 and 2. Consistent with earlier analyses, incident severe obesity was more likely for persons between 45 and 64 years of age, and those who were overweight during childhood.

Table 3 displays negative binomial regression models for the number of times a person was severely obese across the study duration. The results for the total sample show that long-term severe obesity is related to age in a curvilinear fashion. The simple term for age (b = .235, 95% CI = .174–.296) is positively related to long-term severe obesity, but the quadratic term manifests a negative relationship (b = −.003; 95% CI = −.004, −.002). Long-term severe obesity risk increases through middle age and then declines in later life.

The strongest predictor of long-term severe obesity in the total sample was childhood overweight: Persons who reported that they were overweight when they were 12 or 13 were far

Table 3. Negative Binomial Regression Analysis for Long-Term Severe Obesity (Body Mass Index ≥ 35) in NHANES I: Epidemiologic Followup Study, 1971–1992

	Total Sample (N = 3,875)		Women (N = 2,215)		Men (N = 1,610)	
Independent Variable	B (SE)[a]	CI	B (SE)	CI	B (SE)	CI
Age	.235 (.031)***	(.174–.296)	.257 (.043)***	(.172–.342)	.375 (.041)***[b]	(.293–.457)
Age^2	−.003 (.0004)***	(−.004–−.002)	−.003 (.0001)***	(−.004–−.002)	−.006 (.001)***[b]	(−.007–−.005)
Childhood overweight	1.663 (.155)***	(1.358–1.967)	1.413 (.172)***	(1.075–1.750)	2.189 (.293)***[b]	(1.614–2.760)
Female	.441 (.205)*	(.039–.843)				
Black	.418 (.224)	(−.022–.858)	.631 (.250)**	(.141–1.122)	−.144 (.477)	(−1.080–.790)
Lives alone	−.271 (.375)	(−1.006–.463)	−.121 (.347)	(−.803–.559)	−.234 (.599)	(−1.409–.941)
Widowed	.172 (.192)	(−.203–.549)	.101 (.223)	(−.336–.539)	.993 (.301)***[b]	(.403–1.583)
Education	−.121 (.086)	(−.290–.048)	−.106 (.105)	(−.313–.100)	−.208 (.080)**	(−.365–−.051)
Income	−.049 (.059)	(−.165–.066)	−.093 (.056)	(−.204–.016)	.178 (.111)	(−.039–.396)
Private medical insurance	−.532 (.188)**	(−.901–−.164)	−.674 (.189)***	(−1.046–−.301)	.183 (.207)[b]	(−.222–.589)
Medicaid	−.131 (.452)	(−1.019–.756)	−.644 (.450)	(−1.526–.238)	1.038 (.849)	(−.627–2.704)
Regular physician	.173 (.111)	(−.046–.392)	.048 (.136)	(−.218–.315)	.422 (.168)*	(.091–.753)
Smoker	−.116 (.109)	(−.332–.098)	−.302 (.199)	(−.692–.088)	−.001 (.146)	(−.288–.286)
Past smoker	.0001 (.097)	(−.191–.191)	.277 (.221)	(−.155–.711)	−.019 (.118)	(−.250–.212)
Limited activity	.208 (.128)	(−.043–.460)	.290 (.279)	(−.257–.838)	.263 (.368)	(−.458–.985)
Selection (λ), mortality	1.800 (.804)*	(.223–3.377)	1.201 (1.073)	(−.903–3.305)	6.283 (.454)***[b]	(5.393–7.173)
Selection (λ), nonresponse	1.098 (1.174)	(−1.202–3.400)	−.421 (2.928)	(−6.161–5.319)	4.845 (2.629)	(−.307–9.998)
Intercept	−5.780 (.891)	(−7.528–−4.032)	−5.267 (1.917)	(−9.025– 1.509)	−10.693 (.879)	(−12.416–−8.969)
Overdispersion	9.130 (.864)		7.694 (1.135)		10.191 (1.640)	
χ^2	1261.69		1162.01		844.71	
df	17		16		16	

Note: NHANES I = National Health and Nutrition Examination Survey I; CI = 95% confidence interval.

[a]Unstandardized coefficient [standard error (SE) in parentheses].

[b]Wald χ^2 test indicates coefficients are significantly different for women and men ($p < .05$).

*$p \leq .05$; **$p \leq .01$; ***$p \leq .001$.

more likely to be severely obese throughout the duration of the study. Consistent with the cross-sectional analysis of the baseline survey, women manifest a higher risk of severe obesity over the four measurement occasions of the study. Persons without private medical insurance were also more likely to manifest long-term severe obesity. The mortality selection variable was also significantly related to severe obesity over the 20 years of the study (i.e., persons at risk for mortality were more likely to manifest long-term severe obesity).

The results from the separate sample analyses are also presented in Table 3. In those instances in which the relationship was significant in at least one of the groups, the Wald chi-square statistic was used to test for differences in the coefficients for men and women. The results of those tests revealed several instances of significant differences across gender. First, the age effect was stronger for men than for women. Second, the effect of childhood overweight was significantly different. Although childhood overweight was a significant predictor for both women (b = 1.413, 95% CI = 1.075–1.750) and men (b = 2.189, 95% CI = 1.614–2.760), the effect was significantly stronger for men. Other instances in which the effect of long-term severe obesity was stronger for men than women include widowed and mortality selection. By contrast, the effect of private medical insurance on long-term severe obesity was stronger for women than men; it reduced the likelihood of long-term severe obesity for women, but not for men.

The final stage of the analysis is to examine whether childhood overweight and severe obesity in adulthood are related to mortality risk. Two models for estimating mortality risk in the total sample are presented in Table 4. Given that the relationship between BMI and mortality has been found to be curvilinear, binary variables for underweight, normal weight, overweight, and obesity are used. Model 1 specifies the simplest NHLBI categories—in which obesity refers to people with a BMI ≥ 30—whereas Model 2 distinguishes among the types of obesity. The former is what is commonly done when testing whether obesity is related to mortality, but the latter isolates the impact of severe obesity on mortality.

As expected, Model 1 reveals that mortality risk is greater for older people. Childhood overweight is associated with a decreased risk of death once the other body weight variables are considered. (We performed supplementary analyses with reduced models, but found this relationship in even the simplest of models.) For the binary weight variables, mortality risk is higher for both underweight and obese persons. Being overweight, but not obese, (BMI ≥ 25 and BMI < 30) is associated with slightly lower mortality risk [relative risk (RR) = .875, 95% CI = .784–.976]. The effects for the remaining variables are as expected; mortality risk is greater for those with fewer socioeconomic resources (education, income, private medical insurance), smokers, and persons in poorer health (limited activity, serious illness). Having a nonserious illness is associated with lower mortality risk, likely the result of survival into older ages.

Model 2, substituting the two classes of obesity for the one class, leads to very similar results, but clarifies that persons with severe obesity have an even higher risk of mortality. Thinking of the pattern across the binary variables for BMI, one sees that mortality risk is highest for persons who are underweight (RR = 1.746, 95% CI = 1.268–2.403) and persons with severe obesity (RR = 1.571, 95% CI = 1.335–1.849) in comparison with normal weight persons. Class I obesity also heightens mortality risk (RR = 1.356, 95% CI = 1.159–1.588), but being overweight is associated with the lowest mortality risk

Table 4. Risk Ratios and Confidence Intervals from Proportional Hazards Models of Mortality: National Health and Nutrition Examination Survey: Epidemiologic Followup Study, 1971–1992

	Total Sample		Men	Women
Independent Variable	Model 1 (N = 6,497)	Model 2 (N = 6,497)	Model 1 (N = 3,011)	Model 1 (N = 3,486)
Age	1.096***[a]	1.096***	1.092***	1.102***
	(1.087–1.104)[b]	(1.087–1.104)	(1.081–1.104)	(1.094–1.109)
Childhood overweight	.688***	.684***	0.779	.612***
	(.591–.800)	(.587–.797)	(.595–1.020)	(.518–.723)
Female	.564***	.561***		
	(.512–.621)	(.510–.616)		
Underweight (BMI < 18.5)[c]	1.744**	1.746**	1.628**	1.764*
	(1.264–2.405)	(1.268–2.403)	(1.132–2.342)	(1.146–2.714)
Overweight (BMI ≥ 25 and BMI < 30)	.875*	.875*	.907	.853**
	(.784–.976)	(.784–.976)	(.757–1.087)	(.759–.958)
Obese (BMI ≥ 30)	1.404***		1.557**	1.301**
	(1.227–1.606)		(1.190–2.036)	(1.104–1.534)
Obese, class I (BMI ≥ 30 and BMI < 35)		1.356***		
		(1.159–1.588)		
Severe obesity (BMI ≥ 35)		1.571***		
		(1.335–1.849)		
Black	1.101	1.091	1.058	1.133
	(.867–1.398)	(.863–1.378)	(.739–1.515)	(.919–1.396)
Lives alone	1.103	1.107	1.192	1.027
	(.887–1.372)	(.890–1.376)	(.991–1.433)	(.739–1.428)
Widowed	.991	.992	.797	1.070
	(.760–1.293)	(.759–1.296)	(.571–1.112)	(.750–1.526)
Education	.941*	.941*	.926*	.950*
	(.896–.987)	(.896–.988)	(.868–.988)	(.909–.993)
Income	.973***	.972***	.965**	.980
	(.958–.988)	(.958–.987)	(.943–.987)	(.951–1.011)
Private medical insurance	.789***	.793***	.737***	.858
	(.710–.878)	(.715–.879)	(.654–.830)	(.717–1.027)
Medicaid	1.088	1.087	.876	1.393
	(.841–1.408)	(.841–1.406)	(.637–1.205)	(.991–1.959)
Regular physician	.967	.965	1.100	.811
	(.809–1.157)	(.807–1.152)	(.908–1.332)	(.647–1.015)
Smoker	1.796***	1.792***	1.808***	1.828***
	(1.581–2.040)	(1.578–2.035)	(1.495–2.186)	(1.645–2.032)
Past smoker	1.064	1.063	1.013	1.230*
	(.991–1.144)	(.990–1.141)	(.923–1.113)	(1.013–1.492)
Limited activity	1.423***	1.418***	1.627***	1.316**
	(1.229–1.647)	(1.222–1.645)	(1.357–1.950)	(1.112–1.559)
Serious illness	1.431***	1.431***	1.462***	1.424***
	(1.345–1.522)	(1.345–1.522)	(1.376–1.554)	(1.300–1.560)
Nonserious illness	.877*	.877*	.906	.838*
	(.777–.990)	(.779–.988)	(.718–1.143)	(.733–.959)
−2 Log-likelihood	25278.098	25276.666	12343.926	10593.812
df	19	20	18	18

Note: BMI = body mass index.
[a]Risk ratio.
[b]95% confidence interval.
[c]Normal weight (body mass index ≥ 18.5 and body mass index < 25) is the reference group.
*$p \leq .05$; **$p \leq .01$; ***$p \leq .001$.

(RR = .875, 95% CI = .784–.976). The results for most of the other variables are similar across the two models. In supplementary analyses, interaction terms for childhood overweight and the classes of obesity were tested, but none were significant. (An interaction term for race and gender was also tested, but was nonsignificant.)

The next step of the analysis was to estimate mortality risk separately for men and women. Given that there were only 77 men who were severely obese at the first survey, we performed a test of statistical power based on the noncentrality parameter to determine if Model 2 should be estimated for each group (Neter, Wasserman, & Kutner, 1990). Power was insufficient for estimating Model 2 for men (δ = .137); thus, we present Model 1 only for men and women. Several interesting differences exist by gender.

The separate sample analysis reveals that the lower mortality risk observed for childhood overweight in the total sample is because of this effect among women only. Childhood overweight is not related to mortality risk for men. In parallel fashion, the lower mortality risk observed for adult overweight

in the total sample is because of this effect among women only. The other notable difference in these results is that men's mortality is much more contingent on socioeconomic resources than is the case for women. Men who are disadvantaged in terms of socioeconomic status have considerably higher mortality risk.

Discussion

With nearly a quarter of U.S. adults now considered obese, the present research seeks to better understand the life course of severe obesity: Who becomes severely obese, and what are the consequences of having a BMI $\geq$ 35? Although many previous studies identified a link between childhood overweight and adult obesity (BMI $\geq$ 30), the present research revealed an association between childhood overweight and the development of severe obesity in adulthood (BMI $\geq$ 35). More than 14% of the overweight children became severely obese as adults, but less than 4% of the normal weight children became severely obese. Childhood overweight emerged as a strong and very consistent predictor across models of severe obesity at the baseline survey and long-term severe obesity across the four survey waves.

It should be recalled that the measure of childhood body weight used here is overweight, not obesity. A few previous studies have attempted to assess the link between childhood obesity and adult obesity (e.g., Serdula et al., 1993), but the data used here rely on self-reported childhood overweight (identified by respondents as "chubby or very heavy" at 12 or 13). Thus, childhood overweight—let alone childhood obesity—is associated with a significantly higher risk of severe obesity in adulthood. About one-third of the respondents with severe obesity were overweight as children. Moreover, the link may well be underestimated in the present analysis because of the possibility of underreporting childhood overweight, as well as the fact that overweight's effect on adult obesity is stronger in later adolescence (Must, 1996). Overweight during ages 12–13 is a stronger predictor than overweight at younger ages, but overweight at 15–16 has an even stronger relationship with adult obesity (Guo et al., 1994). The analysis reported here provides an important step for better understanding severe obesity over the life course, but additional longitudinal studies are needed to identify why childhood overweight is linked to severe obesity. It may be from parental behavior, learned behavior, genetics, or some combination thereof.

The age range of 45–64 emerged as the most likely period of the life course for incident severe obesity; severe obesity is rare past age 75 for at least two reasons. First, many people 75 and older lose weight associated with sarcopenia or disease incidence and its treatment (Losonczy et al., 1995). Second, as shown here, severe obesity raises the risk of mortality—people with severe obesity are less likely to live into older ages.

Indeed, the results show that both Class I obesity and severe obesity heighten mortality risk. When comparing different classes of BMI, however, it became apparent that overweight women actually had slightly lower mortality risk. There is an emerging body of literature showing that modest overweight may have protective health effects in women during advanced middle-age and later life (e.g., Farahmand, Michaelsson, Baron, Persson, & Ljunghall, 2000). Findings from the present investigation add to this line of research. Further research is needed on why overweight may be associated with lower health risks for women. Other studies, including the Nurses' Health Study, find no protective effect of overweight (Manson et al., 1995), but NHEFS includes a higher percentage of older women. More research is needed on the effects of overweight on mortality, but there is ample evidence showing that obesity raises mortality risk for both men and women—a matter of sober public health concern.

Previous research manifested inconsistent findings regarding the effect of childhood overweight on mortality. Results from the NHEFS reveal that childhood overweight was associated with lower mortality risk for women after controlling for other factors. Other studies have reported parallel findings for men of comparable age (e.g., Sonne-Holm et al., 1983), but this association was observed among women only in this study. This finding is consistent with the concept of acquired immunity—some disadvantages early in the life course may improve immunity or physiological function (Preston et al., 1998). Energy reserves in childhood may benefit young women during key developmental periods, as well as provide more weight-bearing stimulation for the musculoskeletal system. Whatever mechanism is at work, overweight children who did not become severely obese were not at greater risk of mortality.

Much of the previous research on the relationship between childhood overweight and both adult obesity and mortality relied on data from samples of White (Casey et al., 1992; Guo et al., 1994; Must et al., 1992) or male respondents only (Sonne-Holm et al., 1983). The present investigation used a nationally representative U.S. sample and found that women were more than twice as likely as men to be severely obese at the first interview and slightly more likely to manifest long-term severe obesity. African American adults were about twice as likely as White adults to manifest severe obesity at the baseline survey and, interestingly, this racial difference existed for both men and women. Norms for body weight vary by ethnicity, with African Americans generally more tolerant of higher BMI.

Examining the influence of the predictors across gender revealed that a few variables had different effects on the risk of severe obesity. The effect of childhood overweight on severe obesity in adulthood differed by gender. Although women had a higher prevalence of severe obesity, childhood overweight was more closely related to severe obesity for men, consistent with the research of Casey et al. (1992). The link between childhood overweight and *long-term* severe obesity was also stronger among men. Intentional weight loss among women may help explain why childhood overweight is not as closely linked to severe obesity for women. Nevertheless, childhood overweight was a significant predictor of severe obesity, including long-term severe obesity, among both men and women. Severe obesity in adulthood, in turn, independently increased mortality risk.

An important limitation of these analyses is the lack of data on genetic determinants of obesity. Although unavailable in the data, evidence is accumulating regarding genetic determinants of abdominal obesity-metabolic syndrome (Kissebaugh et al., 2000). Indeed, the chain of risk linking childhood overweight with severe obesity could be from genetic forces. In addition, interventions to interrupt the chain of risk across the life course should be tempered by knowledge of the genetic determinants of obesity.

Finally, the link between childhood overweight and severe obesity observed in the NHEFS needs to be viewed in historical context. Most NHEFS subjects were born between 1897 and 1946, when childhood overweight and adult obesity were considerably less prevalent than today (Strauss & Pollack, 2001; Troiano & Flegal, 1998). Meals eaten away from home, portion sizes, and carbonated beverage consumption have all increased in the past three decades. Given that both childhood overweight and adult obesity have increased in recent years—and show no evidence of decline—it is likely that the prevalence of severe obesity in adulthood will increase unless public health interventions are successful. Indeed, not only has the prevalence of obesity increased, but also average BMI among persons with obesity has also risen (NHLBI, 1998). Given these trends, weight-reduction interventions for children and young adults to break the chain of risk could potentially yield substantial health benefits across the life course.

Acknowledgments

Support for this research was provided by grants from the National Institute on Aging to Kenneth Ferraro (R01 AG 13739, K07 AG01055). We appreciate the assistance of Melissa Farmer and Ya-ping Su with the construction of selected variables and computing assistance. Randall Gretebeck, Kimberlee Holland, Jessica Kelley-Moore, Lucy Martin, Janet Wilmoth, and Yunqing Li provided helpful suggestions on an earlier version of the manuscript. Data utilized in this paper were made available by the Inter-University Consortium for Political and Social Research. Neither the collector of the original data nor the Consortium bears any responsibility for the analyses or interpretations presented herein.

Address correspondence to Kenneth F. Ferraro, Professor of Sociology, 1365 Stone Hall, Purdue University, West Lafayette, IN 47907-2059. E-mail: ferraro@purdue.edu

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Received June 6, 2002
Accepted August 29, 2002
Decision Editor: Charles F. Longino, Jr., PhD