Body Mass Index and Disability in Adulthood: A 20-Year Panel Study

| Kenneth F. Ferraro, PhD, Ya-ping Su, PhD, Randall J. Gretebeck, PhD, RD, David R. Black, PhD, MPH, CHES, and Stephen F. Badylak, DVM, PhD, MD

The link between body weight and both morbidity and mortality has been examined extensively, but relatively little research has investigated the relation between body weight and disability.¹ Among the studies that have investigated this relation, the findings are inconsistent. Some of these studies showed that body weight is related to functional disability, 2^{-8} whereas others found no such link.^{9,10} Although the inconsistency may be a result of different research designs and analytic approaches, the measurement of both disability and body weight may be partly responsible for the discrepant findings.

First, *disability* is defined here as a restriction or lack of ability to perform an activity in any domain of life. $11,12$ Although it may be helpful in some circumstances to consider a global construct of overall disability, most of the literature has moved to defining domains of disability, such as upper- and lower-body disability, to better elucidate the process of disablement. $^{9,13-15}$ In addition, the literature suggests a hierarchy in the disablement process such that many of the lower-body or mobility functions, including balance and lowerextremity strength, are the most difficult to maintain, whereas upper-body functions, such as manual dexterity and upper-extremity strength, are easier to maintain. $13,14$ Thus, studies that combine various domains of disability into a single global construct¹⁰ or that consider only a limited range of activities 9 may not be as likely as other studies to uncover a link. Some evidence indicates that obesity is associated with a greater risk for both lower-body^{16,17} and upper-body osteoarthritis¹⁸ leading to disability, but the relation between body weight and different domains of disability must be systematically and longitudinally studied.

Second, the measurement of body weight may itself contribute to inconsistent findings. One empirical generalization that may be gleaned from the studies that found a link be-

Objectives. This study examined whether body mass index (BMI) or change in BMI raises the risk of disability in adulthood.

Methods. The relation between BMI and upper- and lower-body disability was examined among adult subjects from a national longitudinal survey (n=6833). Tobit regression models were used to examine the effect of BMI on disability 10 and 20 years later.

Results. Obesity (BMI≥30) at baseline or becoming obese during the study was associated with higher levels of upper- and, especially, lower-body disability. In persons who began the study with a BMI of 30 or more and became normal weight, disability was not reduced. Underweight persons (BMI<18.5) also manifested higher disability in most instances.

Conclusions. Disability risk was higher for obese persons, but overweight was not consistently associated with higher disability. (*Am J Public Health.* 2002;92:834–840)

tween body weight and disability is that the relation is not linear. Rather, similar to the findings on body mass index (BMI) and mortality, most studies find that the extremes of the BMI distribution are associated with higher disability.^{2,4,5} If only a linear relation between BMI and disability were considered, then the effects associated with low and high BMI would cancel each other, especially when studying an older adult population.

The reasons that obesity and underweight are related to disability are probably a result of different mechanisms. Obesity in adulthood may lead to disability because of 1 or more biological processes: skeletal stress,¹⁹ protein glycation in connective tissue, $20,21$ or atherogenesis.22 Underweight in adulthood, on the contrary, is more often the result of disability, especially among older people.²³⁻²⁵ In more developed countries, underweight is likely the result of disablement processes whereby disease progression during terminal illness leads to reduced muscle mass and strength.26 (Although weight gain is common in middle age, weight loss is common in advanced ages.⁷)

The current study systematically examined the relation between BMI and upper- and lower-body disability in a longitudinal study. We anticipated that both obese and underweight persons would manifest higher levels of both types of disability but that the relations would be stronger for lower-body disability.8,27,28 Although it is unclear if overweight also raises disability risk, such an effect was tested with the National Heart, Lung, and Blood Institute (NHLBI) guidelines.²⁹ Our findings should enhance epidemiological knowledge of BMI and disability risk, including the utility of the NHLBI guidelines for such analyses, and be helpful for health policy and health education efforts to minimize disability in American society.

METHODS

Sample

Data from the National Health and Nutrition Examination Survey I (NHANES I) and its Epidemiologic Follow-Up Study (NHEFS) were used in this research.³⁰ The baseline NHANES I was conducted from 1971 to 1975. The sampling design was a multistage, stratified probability sample of noninstitutionalized persons aged 25 to 74 years. Data from the baseline survey and 2 follow-ups were used. The second wave of data was collected during 1982 to 1984; the third wave was collected in 1992, resulting in an approximate 20-year observation period.³¹

The analyses were completed with the White and Black respondents of the NHEFS subsample, who were administered the "detailed component," including the Health Care

Needs Questionnaire, at baseline (n=6833). The sample used in this study was composed of 5955 White and 878 Black respondents (12.8%) at baseline; unweighted data were used throughout. $32,33$ The percentage of cases receiving the detailed component at baseline and traced through follow-ups was excellent (92.6% of the survivors at the second wave of data; 96.5% of the survivors at the third wave). The number of cases lost to mortality, inability to trace, and refusals to participate was 1644 by wave 2 and 2696 by wave 3.

Measurement of Disability

Disability was not measured during the baseline survey but was measured at the follow-up surveys. Thus, the logic of the analyses was to use the prospective nature of the data to examine the lagged effect of BMI on disability. For the follow-up surveys, items from the Stanford Health Assessment Questionnaire Disability Index were used. The index asks about very specific tasks with the following question: "Please tell me if you have no difficulty, some difficulty, much difficulty or are unable to do these activities at all when you are by yourself and without the use of aids. . . . Lift and carry a full bag of groceries?" The tasks span a wide range of functions in 8 domains. The original index included 26 items, but several items were either deleted or modified in later interviews.³⁴ Nineteen items (common to both waves) were used. Responses for each item ranged from 1 (no difficulty) to 4 (unable to do).

Consistent with the literature, we developed measures of upper- and lower-body disability.
 $\ensuremath{\mathrm{^{9,13-15}}}$ The upper-body disability index used 9 items (range: 9 to 36) and included items from the following domains: dressing and grooming, eating, reaching, and gripping. The lower-body disability index used 10 items (range: 10 to 40) and included items from the following domains: hygiene (and toileting), arising, walking, and activities (errands). A complete list of the items for each index is presented in Table 1. Reliability coefficients (α) for upper-body disability were 0.91 and 0.97 at the second and third waves, respectively; reliabilities for lower-body disability were 0.94 and 0.93, respectively. Missing data were imputed to group means

defined by age, sex, and race/ethnicity if the subject had missing data on 4 or fewer of the items in each index. If respondents missed more than 4 items, they were treated as missing on the index.³⁵

Body Mass Index

BMI was defined as weight in kg/(height in meters)². BMI was measured continuously for each respondent and was analyzed both continuously and categorically according to the NHLBI guidelines (also adopted by the World Health Organization for international comparisons). For the first 2 waves of the NHEFS, research staff measured weight and height. At the baseline survey, weight was measured by a Toledo self-balancing scale to a quarter pound.³⁶ For the second wave, weight was measured with a Health-O-Meter, Model 32, weight scale. For the third wave of the NHEFS, weight was self-reported. Although self-reported weight is closely related to physical measurement, it may slightly underestimate the tails of the weight distribution.^{37,38} Such bias is probably minimal to the main conclusions of this study, however, because it would exist only at the third wave. (To examine potential bias, we created predicted scores for wave 3 BMI from measured BMI at waves 1 and 2 and selected baseline covariates. The correlation between the third wave predicted and reported BMI values was 0.84, suggesting minimal bias due to reported weight at wave 3.)

Table 1 presents cases in each category of BMI at the 3 survey waves and mean disability at waves 2 and 3. Based on the NHLBI guidelines, 46% of the sample was normal weight at baseline. Fewer than 4% of the subjects were underweight, whereas about 34% were overweight, and almost 17% were obese.

Measurement of Covariates

Health measures were assessed in the baseline interview and were used as control variables. Morbidity was derived from the following question: "Has a doctor ever told you that you have. . . hypertension or high blood pressure?" (36 conditions were presented). Unlike some surveys that ask if a person has a condition, the NHANES question solicited responses based on evaluation by a physician. Each condition was coded as a binary variable (0, 1). The conditions were then classified into those that were life threatening or serious and all remaining conditions.³⁹ Serious conditions included cancer, diabetes, heart failure, hypertension, and stroke. Examples of chronic nonserious conditions included arthritis, asthma, bone fracture, cataracts, gout, psoriasis, and ulcer. The serious and chronic nonserious conditions were then summed separately. Supplementary analyses treated the diseases as separate binary variables. Interactions of several of the diseases (e.g., diabetes and arthritis) also were tested, but none was statistically significant in predicting disability.

Although the NHEFS did not include a measure of disability at baseline, respondents were asked 2 questions about their exercise from both recreational and nonrecreational activities. Respondents who were quite inactive based on their responses to both questions were scored as 1 for restricted activity; all others were scored as not restricted (0).

The remaining independent variables span a broad range of factors related to disability or BMI, either directly or indirectly, and their measurement is summarized in Table 2. These variables include indicators of health risk behaviors, such as smoking—based on self-reported consumption of cigarettes, cigars, and pipe tobacco at the time of the interview and during one's lifetime. Current smokers were classified as light or heavy based on the number of cigarettes smoked per day (<20 and ≥20, respectively). NHEFS respondents were asked at baseline whether they had a regular physician (scored as 0, 1). BMI has been linked with socioeconomic status,⁴⁰ and relevant variables in the NHEFS included education, family income, availability of private health insurance, and Medicaid status. The measurement of the remaining independent variables was fairly straightforward.³ All binary variables were coded as 0 and 1, with 1 equal to the variable name.

Analytic Plan

More than 70% of the respondents reported no upper- or lower-body disability. The skewed distribution of the disability measures created a floor effect—what is often referred to as censoring—and violated the assumptions of ordinary least squares regression. Ordinary least squares regression

TABLE 1—Descriptive Statistics for the National Health and Nutrition Examination Survey I: Epidemiologic Follow-Up Study, 1971–1992: Number of Cases and Upper- and Lower-Body Disability for Waves 2 and 3, by Weight Categories at Baseline

Note. BMI = body mass index.

^aWeight categories defined by the National Heart, Lung, and Blood Institute.²⁹

^bUpper-body disability includes the following 9 items: dress self, including tying shoes, working zippers; comb and shampoo hair; cut meat; lift a full cup or glass to mouth; open a new milk carton;

reach and get down a 5-pound object from above head; bend down and pick up clothing from the floor; open jars that have been previously opened; and use a pen or pencil.

Lower-body disability includes the following 10 items: get into and out of bathtub; wash and dry the whole body; get on and off toilet; stand up from an armless chair; get into and out of bed; walk a quarter of a mile; walk up and down at least 2 steps; get into and out of a car; run errands and shop; and do light chores such as vacuuming.

is designed for normally distributed intervalor ratio-dependent variables. In the case of highly skewed and censored data, ordinary least squares estimates are inconsistent (biased intercepts and slopes). Logistic or probit regression models, whether binomial or ordered, are another option, but they would not make full use of the variability among persons with different levels of disability. (One would need to collapse the original metric of the disability variables—with ranges of 28 and 31 into a small number of categories. This would reduce or potentially eliminate differences among the persons reporting disability.)

Tobit models, sometimes referred to as censored regression models, are specifically designed for variables distributed with a large percentage of cases at the lower or upper limit.⁴¹ Tobit models assume clustering at a limit and simultaneously account for (1) probability of being censored and (2) variability among those at different levels of the outcome. Multivariate tobit models were used in the current analysis to conveniently distinguish the cases with no disability from those with any disability and to account for the considerable variability among the latter. Unlike ordinary least squares or logistic regression methods, tobit estimates are consistent and efficient for censored data. The tobit model uses the same structural form as the probit model but preserves the information within the limit via maximum likelihood.⁴²

Although case tracing and reinterview rates were high in the NHEFS, it is always possible that attrition in longitudinal analyses may influence sample estimates of relationships and lead to bias in the estimates. Thus, selectionbias models, originally developed by Heckman,⁴³ were used to correct parameter estimates for differential selectivity due to death, refusal to participate, or inability to trace. The procedure was first to estimate a probit model to distinguish subjects who participated from those who did not. The second step was to use the probit results to create a selection instrument (lambda) based on the inverse Mills ratio and add the selection instrument to the

regression model of interest.⁴⁴ This 2-step approach has been extended to incorporate 2 hazard-rate instruments for different forms of attrition 45 and is conveniently handled in LIMDEP.⁴⁶

The results presented below differentiate attrition due to mortality from that due to nonresponse by estimating separate probit equations. The probit model estimating mortality during the survey showed that deaths were more likely among Black, older, male respondents who had more physician-evaluated morbidity and less income. The probit model estimating nonresponse during the survey waves showed that subjects more likely to drop out of the analysis were Black, younger, missing on occupational status, urban, and of lower income. These 2 probit selection equations also included instrumental variables (i.e., at least one variable that was not included in the substantive equation for disability). For the nonresponse equation, missing on income, missing on occupation, rural, South, and marital status at wave 1 variables were included; for the

TABLE 2—Tobit Estimates of Disability at Wave 2 of the National Health and Nutrition Examination Survey I: Epidemiologic Follow-Up Study

Note. BMI = body mass index.

Reference group.

P <* .05; *P <* .01; ****P <* .001.

mortality equation, self-rated health worries and 2 measures of physician-evaluated morbidity were included.⁴⁷

Although this study examines the consequences of body weight, women who gave birth 1 year before or after any survey wave were omitted from the analysis. Given the large number of cases available, emphasis was placed on findings based on a more conservative probability level of .01.

RESULTS

Table 2 shows the results of the tobit analyses predicting upper- and lower-body disability at the second wave. The first 2 equations are for upper-body disability and reflect the continuous and categorical forms of BMI, respectively. The simple linear measure of BMI is not predictive of upper-body disability at wave 2, but the NHLBI categories in the second equation (second column) show that both obese and underweight persons had more disability approximately 10 years later. Higher disability also was observed among persons with restricted activity and more morbidity. Supplementary analyses substituting binary variables for the count of chronic nonserious illnesses showed the strongest effects for

arthritis; diabetes and heart trouble emerged as important among the serious illnesses. Upper-body disability also was higher among older people and women. The importance of correcting for selection bias becomes clear when one examines the lambda coefficients in Table 2: those who died and those who were not reinterviewed would have had much higher levels of upper-body disability (i.e., their departure from the study was likely, in most cases, a result of their poorer health status).

The last 2 columns of Table 2 show the results for lower-body disability at the second wave. BMI was significantly associated with

lower-body disability, and most of the effects of the other variables were similar to what was observed for upper-body disability. The effects due to smoking, especially heavy smoking, were stronger in the equations for lower- than for upper-body disability. The supplementary analyses for individual diseases again showed the effects of arthritis, diabetes, and heart trouble, but hip fracture also emerged as important to lower-body disability. The results based on the NHLBI categories again indicated that both obese and underweight $(BMI<18.5)$ persons had higher disability at wave 2. Overweight respondents (BMI≥25 and≤30) did not differ from the normal-weight respondents on lower-body disability.

The results showed that both forms of disability were more likely among persons at the tails of the BMI distribution and confirmed the utility of the NHLBI guidelines for studying disability. Thus, in the analyses to follow, we used NHLBI guidelines to more closely examine changes over the duration of the study.

We next considered the role of BMI change in disability and extended the analysis to the full 20-year follow-up (presented in

Table 3). All of the covariates included in the models for Table 2 also were included in these analyses, but only the BMI categories are shown in Table 3 (an elaborated version of Table 3 that includes the covariates is available from the authors on request).

The first 2 equations in Table 3 examine the association between change in BMI from wave 1 to wave 2 and upper- and lower-body disability at wave 2. These equations include a series of binary variables (scored 0, 1) to capture change in BMI based on the NHLBI guidelines (normal weight serves as the reference group throughout). Underweight was associated with more upper-body disability, but 2 forms of BMI change were statistically significant. First, subjects who had a normal BMI at the first wave and became obese by the second wave also had higher disability. Second, subjects who had a normal BMI at the first wave and became underweight by the second wave also likewise had higher disability, suggesting problems associated with the progression of the serious and chronic illnesses. The results for lower-body disability were similar to those for upperbody disability: being or becoming obese or

underweight was associated with more disability.

The remaining equations (3 to 6) in Table 3 are for disability at the third wave and treat disability at the second wave as an independent variable; thus, these equations report residualized change analyses (interpreted as change in disability). The third equation uses the categorical forms of BMI and BMI change observed by wave 2 in predicting upper-body disability by wave 3. This equation examines the lagged effects of BMI and BMI change on disability. The results showed that obesity at the baseline interview was associated with increased upper-body disability by wave 3. The results also showed that persons who had a normal BMI at baseline but became obese by wave 2 manifested an increase in upper-body disability by wave 3. Underweight at baseline was associated with more upper-body disability, but note that underweight persons who became normal weight experienced a *decline* in disability.

The next equation (4) in Table 3 shows that both being obese at baseline and becoming obese by wave 2 were associated with greater lower-body disability by wave 3. Being underweight at baseline remained a

TABLE 3—Tobit Estimates of Disability at Waves 2 (W2) and 3 (W3) of the National Health and Nutrition Examination Survey I: Epidemiologic Follow-Up Study, by Changes in Body Mass Index

Note. W1 = wave 1. Covariates in each model include those listed in Table 2. The equations for disability at W3 include W2 disability.

P <* .05; *P <* .01; ****P <* .001.

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significant predictor, but becoming underweight was not related to lower-body disability. Once the change variables were considered, baseline overweight was associated with greater lower-body disability.

The final 2 equations in Table 3 include BMI transitions across all waves, and the results showed fairly consistent effects on upper-body disability for both obesity and underweight, whether measured at baseline or moving into these categories during the study. Note also that those who were underweight at baseline and became normal weight by the second wave had a reduction in upper-body disability. The risk of lower-body disability also was higher for obese and underweight respondents, both at baseline and during the course of the study. Note that the consequences of becoming obese by wave 2 and of becoming obese by wave 3 were the same: greater lower-body disability. Again, baseline overweight was associated with greater lowerbody disability.

Finally, we replicated these analyses with alternative categoric forms of BMI and found very similar results. It is clear that the effect due to obesity is robust across various models and that obesity's effect on disability is not short-lived: obesity at the inception of the study was consistently related to higher disability, regardless of subsequent weight loss. Also, persons who began the study with a BMI of 30 or more and lost weight did *not* see a reduction in their disability.

DISCUSSION

The current study systematically examined the relation between BMI and disability in a prospective longitudinal study. The study adds evidence to the accumulated body of knowledge on the health consequences of excess body weight. First, the findings were quite robust across lag times that obesity was associated with higher levels of both upperand lower-body disability and more rapid increases in each over time.2,5,48 Moreover, the findings showed that obesity's effect on disability is not short-lived. Obesity at the inception of the study was consistently related to higher long-term disability, especially lowerbody disability. In addition, persons who began the study with a normal BMI and be-

came obese had higher disability at waves 2 and 3. It is also striking that persons who began the study with a BMI of 30 or more and lost weight did not see a reduction in their disability.

One should not conclude that obesity's effect on disability is irreversible, but the evidence from the NHEFS makes it clear that adult obesity greatly raises the long-term risk of disability. These findings speak to the following research question: What else besides weight loss is required to reverse the longterm effects of obesity on disability risk?

Although obesity was consistently related to disability, overweight as defined by the NHLBI was not. Based on the NHLBI definition of overweight across different lag times, the analysis found only 2 instances in which overweight was associated with higher disability: in both cases, baseline overweight increased lower-body disability. In no instance did overweight increase *upper*-body disability risk. More research is needed, however, on the long-term consequences of overweight, especially as it pertains to overweight associated with a larger waist circumference.⁵

The relations between underweight and disability also became clearer across the models tested with these data. Baseline underweight often was related to disability, and becoming underweight was associated with greater upper- and lower-body disability. The effect due to incident underweight during the NHEFS, however, was not as long term as for incident obesity. For example, becoming obese between waves 1 and 2 was associated with more disability at both wave 2 and wave 3, but becoming underweight between waves 1 and 2 did not regularly influence disability at wave 3. The findings on underweight suggest a mechanism due to the progression of disease: incident weight loss is likely a consequence of incident morbidity and disability.⁷ Further research on the relation between disease progression and weight loss is needed. Shorter-term longitudinal studies may be beneficial for the purpose of identifying rapid weight loss associated with terminal decline.

Research predicting the course of disability has proliferated in recent years, but many of these studies—even fairly recent ones have not examined the influence of BMI. The findings from the NHEFS strengthen

empirical generalizations that a nonlinear relation exists between BMI and various health-related outcomes.^{2,4} It is clear that simply adding BMI as a covariate to studies of disability may be misleading—the relation between BMI and disability is curvilinear. Given the different consequences for overweight $(25 \geq BMI \leq 30.0)$ and obesity (BMI \geq 30) observed in the current analysis, we urge disability researchers to include categorical measures of BMI in analyses of the disablement process.

This study adds to the modest but growing literature on the consequences of BMI on disability and quality of life.⁵ These findings from the NHEFS were quite clear that obesity has long-term consequences on the functional status of adults and showed the importance of avoiding a BMI of 30 or more. Although the current study could not identify the precise etiologic mechanism for the relation between obesity and disability, the consistency of the findings across 2 domains of disability suggests that it merits further attention. \blacksquare

About the Authors

Kenneth F. Ferraro, David R. Black, and Stephen F. Badylak are with Purdue University, West Lafayette, Ind. Yaping Su is with Lighthouse International, New York, NY. Randall J. Gretebeck is with Wayne State University, Detroit, Mich.

Requests for reprints should be sent to Kenneth F. Ferraro, PhD, Professor of Sociology, 1365 Stone Hall, Purdue University, West Lafayette, IN 47907-1365 (e-mail: ferraro@purdue.edu).

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Contributors

K.F. Ferraro planned the study, supervised the data analysis, and wrote the paper. Y. Su performed all statistical analyses and contributed to the revision of the paper. R.J. Gretebeck, D.R. Black, and S.F. Badylak helped plan the study (including instrumentation) and revise the paper.

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