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Original Contribution

Obesity and Mortality Risk: New Findings From Body Mass Index Trajectories

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Little research has addressed the heterogeneity and mortality risk in body mass index (BMI) trajectories among older populations. Applying latent class trajectory models to 9,538 adults aged 51 to 77 years from the US Health and Retirement Study (1992–2008), we defined 6 latent BMI trajectories: normal weight downward, normal weight upward, overweight stable, overweight obesity, class I obese upward, and class II/III obese upward. Using survival analysis, we found that people in the overweight stable trajectory had the highest survival rate, followed by those in the overweight obesity, normal weight upward, class I obese upward, normal weight downward, and class II/III obese upward trajectories. The results were robust after controlling for baseline demographic and socioeconomic characteristics, smoking status, limitations in activities of daily living, a wide range of chronic illnesses, and self-rated health. Further analysis suggested that BMI trajectories were more predictive of mortality risk than was static BMI status. Using attributable risk analysis, we found that approximately 7.2% of deaths after 51 years of age among the 1931–1941 birth cohort were due to class I and class II/III obese upward trajectories. This suggests that trajectories of increasing obesity past 51 years of age pose a substantive threat to future gains in life expectancy.

body mass index trajectories; heterogeneity; mortality; obesity; United States

Abbreviations: ADL, activities of daily living; BMI, body mass index; HRS, Health and Retirement Study.

The rising prevalence of obesity has emerged as a potential threat to overall life expectancy in the future. The extent of this threat, however, is still uncertain, and estimates of the percentage of total deaths due to obesity vary widely, from 5% (1) to 13% (2–4). Although these estimates are all based on measuring the mortality consequences of body mass index (BMI) assessed at baseline (i.e., at 1 point in time), other studies have found that a dynamic measure of weight status (weight or BMI change) is more predictive of mortality than is a static measure of weight status (i.e., baseline BMI), especially among older adults (5, 6). We might expect obesity to increase the risk of death more profoundly when it persists over the life course. Therefore, in order to better assess the rising threat of obesity, it is essential to examine the mortality consequences of BMI trajectories.

Prior studies based on dynamic measures have yielded mixed findings about the mortality consequences of weight change (5–21). Several factors contribute to the mixed findings. First, the association of weight change with mortality depends on baseline BMI status. Weight gain leads to excess death among overweight/obese individuals but lowers the mortality risk among underweight or normal weight people (8). Second, the association differs by the magnitude of weight change. Modest weight gains are associated with a decreased mortality risk, but excessive weight gains predict an increased mortality risk (7, 13). When both initial weight and the magnitude of change are taken into account, small weight gains (1.0–2.9 BMI units) are not associated with excess mortality risk among 50–70-year-old Americans, regardless of their initial BMI levels, whereas large weight gains (3.0–5.0 units) increase the risk of death only when the initial BMI is greater than 35. Moreover, both small weight losses (1.0–2.9 units) and large weight losses (3.0–5.0 units) are associated with an increase in the risk of death among people who are normal, overweight, or mildly obese at baseline (22).

Even though they advanced beyond the use of a static measure of baseline BMI, prior studies nevertheless have suffered from several limitations. First, most studies only considered mortality consequences of weight change between 2 time points, either over the short term (22) or the long term (8). This approach obscures heterogeneity in weight changes that occur after the second time point, as well as weight fluctuations

between the 2 time points. Second, although distinguishing between small and large weight changes is important, the specified cutoffs are necessarily arbitrary and may inadequately represent true variation in the magnitude of weight change. Third, when estimating the interaction effect between weight change and BMI on mortality risk, some studies assumed a linear functional effect; that is, they expect the effect of weight gain or weight loss on mortality to be linear across initial BMI status (22). This functional assumption, however, is arbitrary and may misrepresent the interaction of weight change and initial weight status.

The objectives of the present study were to capture heterogeneity in the entire BMI trajectory after 51 years of age, examine the mortality consequences of this heterogeneity, and calculate the mortality risk attributable to each trajectory using data from the US Health and Retirement Study (HRS). We used a semiparametric group-based trajectory model or the latent class trajectory model (23–26) to capture heterogeneity in weight changes without specifying artificial cutoff points or a strong functional assumption. This strategy can straightforwardly depict how BMI may increase, decrease, or remain stable among various groups with different initial BMI statuses.

MATERIALS AND METHODS

Data and participants

We used data from the HRS, a nationally representative survey of Americans born between 1931 and 1941 (27). HRS respondents and their spouses were initially interviewed in 1992 and were then reinterviewed in 2-year intervals. We used 9 waves of data that spanned 1992–2008. We restricted the analysis to respondents who were 51–61 years of age at the time of the original 1992 HRS sample. Our analytic sample consisted of 9,538 respondents aged 51–61 years in 1992 who were followed until death, exit from the study, or censoring after the end of the 2008 wave. HRS collects data on the vital status and date of death, if applicable, during its attempts to recontact respondents in each wave. HRS also matches respondents to the National Death Index to ascertain the date of death. When available, we used the date of death from National Death Index rather than the date of death from the HRS interviews; for deaths after 2008, we used dates of death from the HRS interviews only. As of September 2011, a total of 2,526 respondents from our analytic sample had been confirmed as deceased by the HRS. The HRS supplied the month and year of death, allowing us to compute time spent at risk. For the 2,526 respondents known to have died, exposure to mortality risk was calculated as the duration from 51 years of age until the date of death (in months). For the 7,012 surviving respondents, we similarly computed exposure to mortality risk as the duration from 51 years of age until December 2009 if they are known or presumed to be alive as of the 2010 wave.

Predictors of mortality

BMI trajectory. Upon entry into the study, respondents contributed data on self-reported height and weight, and they contributed further data on self-reported weight at every successive interview. We used these data to calculate BMI, defined as the ratio of weight in kilograms to the square of height in meters. In waves in which a respondent was not interviewed or did not report his or her weight, we treated BMI as missing. The latent class trajectory model allowed individuals to have incomplete BMI data over the course of follow-up so that they could be retained in the analytic sample. The youngest respondents in the sample were 51 years of age in 1992, and the oldest respondents in the sample were 77 years of age in 2008, so we were able to construct BMI trajectories from ages 51–77 years.

Sociodemographic and behavioral factors. At the 1992 baseline interview, HRS investigators recorded information on sex, race/ethnicity (non-Hispanic white, non-Hispanic black, or Hispanic), marital status (never married, married, separated, divorced, widowed, or living with a partner), and educational level (years completed). We use imputed data files provided by RAND to determine respondents' income in dollars (28). Respondents also reported whether they have ever smoked, and if so, whether they currently smoked. We collapsed these questions into a single measure of smoking, distinguishing among never smokers, former smokers, and current smokers. We include a binary measure of physical activity level, distinguishing between respondents who engaged in vigorous physical activity 3 or more times per week and those who do not.

Health and medical history. At the 1992 baseline interview, the HRS survey included a battery of 5 questions aimed at measuring difficulty with activities of daily living (ADL), including dressing oneself, eating, bathing and showering, getting in and out of bed, and walking across a room (29). The original response categories for each activity consisted of a 4-point scale, from "not at all difficult" to "very difficult/can't do," with an additional category of "don't do." We recoded each ADL item to one if the respondent reported having any difficulty or that he or she didn't do the task and zero if the respondent reported no difficulty. We then summed the items to create a 0–5 scale of ADL limitations. At baseline, ADL items demonstrated good agreement with alternative measures of physical functioning collected in the HRS (30). Respondents also reported at the 1992 baseline whether they had ever been diagnosed with any of the following 7 conditions: angina, heart failure or heart attack; arthritis; bronchitis or emphysema; cancer; diabetes; stroke; or bone fracture. Finally, we include a baseline measure of self-rated general health on a 5-point scale: 1 indicated "excellent," 2 indicated "very good," 3 indicated "good," 4 indicated "fair," and 5 indicated "poor."

Statistical analysis

We use a semiparametric group-based trajectory model to capture unobserved heterogeneity in the BMI trajectories after age 51 years. This model uses a multinomial mixture modeling strategy and identifies relatively homogeneous clusters of trajectories of change over time in the presence of repeated observations on analytic units (23, 25). In other words, this model assumes that the population consists of a mixture of underlying trajectories (31). Web Appendix 1 (available at http://aje.oxfordjournals.org/) provides the technical details of this model. We used the SAS PROC TRAJ package to estimate the model (SAS Institute, Inc., Cary, North Carolina).

As the distribution of BMI was right-skewed, we model the logarithm of BMI (log(BMI)) instead of a linear specification of BMI. After obtaining trajectories of log(BMI), we fitted a multivariate Cox proportional hazard model adjusted for baseline sociodemographic, behavioral, health, and disease factors to calculate the relative mortality risk of each trajectory, using age (months elapsed since age 51 years) to parameterize the baseline hazard function (32). The analyses were performed using SAS PROC PHREG program.

Although the health covariates were assessed multiple times in the HRS, we only used data on these covariates from the 1992 baseline interview. In the latent trajectory model, BMI trajectories were determined using information on BMI only. Time-constant variables can be used to assign trajectory membership (e.g., baseline difficulties with ADL influence the likelihood of entering a certain BMI trajectory) that can then be used to probabilistically assign an individual's BMI trajectory. A time-varying covariate can only shift the trajectory up or down; it cannot retroactively determine trajectory membership. We included health covariates to assess whether the associations of BMI trajectories with mortality risk were confounded by other factors that affect trajectory membership, so we used the baseline measurements of these factors, which may influence both trajectory membership and subsequent mortality risk. Considering baseline health characteristics as potential confounders, we proceeded by fitting regression models both with and without adjustment for these factors.

After obtaining the hazard ratios associated with BMI trajectories adjusted for baseline sociodemographic and behavioral factors from the Cox model, we calculated the population attributable mortality risk fraction using the following formula:

$$\frac{\sum_j (C_j RR_j - C_j^* RR_j)}{\sum_j (C_j RR_j)},$$

where j indexes the category of BMI trajectories, C_j refers to the proportion of j_{th} BMI trajectory in the population, and RR_j refers to the relative mortality risk of j_{th} BMI trajectory compared with the reference trajectory, which can be obtained from the hazard ratios in the proportional hazard model (33). C_j^* is the counterfactual proportion of the j_{th} BMI trajectory in the population when all the respondents in the corresponding j_{th} trajectory are assigned to the reference trajectory.

RESULTS

Table 1 describes the analytic sample. At the 1992 baseline, the mean BMI was 27.2, which is in the middle of the overweight range. The average age was 56, with non-Hispanic whites comprising 73% of the sample, non-Hispanic blacks comprising 17%, and Hispanics comprising 10%. Men accounted for 47% of the sample and women accounted for 53%. A review of the health characteristics revealed a population beginning to experience the maladies of old age: 38%, 16%, and 11% had been diagnosed with arthritis, circulatory problems (angina, heart failure, heart attack, or stroke), and diabetes, respectively. The average respondent reported fewer than 1 ADL limitation, and the average self-rated health was halfway between the very good and good categories.

Figure 1 portrays the 6 trajectories obtained from the latent class trajectory model. Six linear latent trajectories best fit the data (Web Appendix 2 describes the model selection). We defined 4 BMI groups based on World Health Organization guidelines: normal weight (BMI of 18.5–24.9), overweight (BMI of 25–29.9), class I obese (BMI of 30–34.9), and class II/III obese (BMI greater than or equal to 35). Because we modeled the trajectories based on log(BMI), we transformed the cutoff points to 2.92, 3.21, 3.40, and 3.56, respectively. The topmost trajectory (plus signs), which included 3.4% of the sample, started with class II/III obesity at age 51 years (BMI = 40.8) and then increased to a BMI of 42.9 at age 77 years. We referred to this as the "class II/III obese upward" trajectory. The next trajectory (closed diamonds), which included 11.7% of the sample, started with a class I obese status at age 51 years (BMI = 33.1) and increased to a BMI of 34.9 at age 77 years. We referred to this as the "class I obese upward" trajectory. The trajectory marked by the dashed line, which included of 22.8% of the sample, started with an overweight status at age 51 years (BMI = 28.9) and progressed to a class I obese status at age 77 (BMI = 30.6). This is the "overweight obesity" trajectory. The trajectory marked by the solid line started with an overweight status at age 51 years (BMI = 25.8) and slowly increased, but it remained within the overweight category by age 77 years (BMI = 26.9). We call this trajectory "overweight stable," and it comprised 29.5% of the sample. The next trajectory (x's), which accounted for 24.2% of the sample, started with a normal weight at age 51 years (BMI = 23.1) and slowly increased to a BMI of 23.6 at age 77 years. We referred to this the "normal weight upward" trajectory. The trajectory marked by open diamonds, which included 8.4% of the sample, started with a normal weight at age 51 years (BMI = 20.5) and slowly declined to a BMI of 19.4 at age 77 years. We referred to this as the "normal weight downward" trajectory.

Figure 2 shows the Kaplan-Meier survival curves for these 6 BMI trajectories. The overweight stable trajectory, shown as a solid line, is more rectangular and extends further to the right than other trajectories. This means that individuals on this trajectory were more likely to survive to older ages. Close to this survival curve are the curves for the overweight obesity and normal weight upward trajectories, with the former extending further to the right. Class I obese upward and normal weight downward are less rectangular than the above 3 trajectories, implying that individuals on these 2 trajectories died earlier. The survival curve on the far left is for the class II/III obese upward trajectory, which means individuals on this trajectory died even earlier than did those in the other 5 trajectories.

Table 2 presents the adjusted hazard ratios of BMI trajectories from the Cox proportional hazard model with the overweight stable trajectory as the reference group (Web Table 1 includes all coefficients). As expected from Figure 2, the other 5 trajectories are associated with an excess risk of death compared with the overweight stable trajectory. After adjustment for sociodemographic factors, the normal weight downward trajectory was significantly associated with a 112% ($P < 0.001$, 2-sided here and thereafter) increase in mortality risk. The normal weight upward trajectory was associated with an excess risk of 17% ($P < 0.01$). The overweight obesity trajectory was not significantly associated with a greater

Table 1. Baseline Characteristics of Participants in the Health and Retirement Study, United States, 1992

Characteristic	Total ($n = 9,538$)		Men ($n = 4,482$)		Women ($n = 5,056$)	
	Mean (SD)	%	Mean (SD)	%	Mean (SD)	%
Deceased		26.5		31.0		22.5
BMI[a]	27.2 (5.1)		27.3 (4.4)		27.1 (5.7)	
Underweight[b]		1.3		0.5		2.0
Normal weight[c]		34.4		29.7		38.6
Overweight[d]		40.8		48.6		33.9
Class I obese[e]		16.6		16.6		16.5
Class II/III obese[f]		6.9		4.5		9.1
Demographic characteristic						
Age, years	55.7 (3.2)		55.7 (3.2)		55.7 (3.2)	
Non-Hispanic white		73.1		75.2		71.2
Non-Hispanic black		17.4		15.6		19.0
Hispanic		9.5		9.2		9.8
Socioeconomic factors						
Income	46,390 (50,394)		52,059 (55,363)		41,362 (44,946)	
Years of schooling	12.0 (3.2)		12.2 (3.4)		11.9 (3.0)	
Married		73.5		80.6		67.2
Partner		2.5		3.5		1.7
Separated		3.2		2.6		3.6
Widowed		6.2		1.6		10.3
Never married		3.7		3.6		3.7
Divorced		11.0		8.1		13.4
Behavioral factors						
Current smoker		27.4		29.6		25.4
Former smoker		36.3		45.0		28.6
Never smoker		36.3		25.4		46.0
Vigorous physical activity ≥3 times per week		22.3		21.4		23.0
Health and disease factors						
No. of ADL limitations	0.2 (0.7)		0.2 (0.7)		0.2 (0.7)	
Bone fracture		13.8		13.3		14.3
Arthritis		38.1		31.0		44.5
Angina, heart failure or heart attack		12.9		14.9		11.1
Bronchitis or emphysema		8.2		7.7		8.6
Cancer		5.6		3.3		7.6
Diabetes		10.8		10.7		10.8
Stroke		2.9		3.3		2.6
Self-rated health	2.6 (1.2)		2.6 (1.2)		2.6 (1.2)	

Abbreviations: ADL, activities of daily living; BMI, body mass index; SD, standard deviation.
[a] Weight (kg)/height $(m)^2$.
[b] Underweight was defined as having a BMI less than 18.5.
[c] Normal weight was defined as having a BMI between 18.5 and 24.9.
[d] Overweight was defined as having a BMI between 25 and 29.9.
[e] Class I obesity was defined as having a BMI between 30 and 34.9.
[f] Class II/III obesity was defined as having a BMI greater than or equal to 35.

risk. The class I obese upward trajectory had a 25% increase ($P < 0.01$), and the class II/III obese upward trajectory had an even larger increase of 128% ($P < 0.001$). After adjustment for behavioral factors, including smoking status and vigorous physical activity, the excess mortality risks associated with the overweight obesity, class I obese upward, and

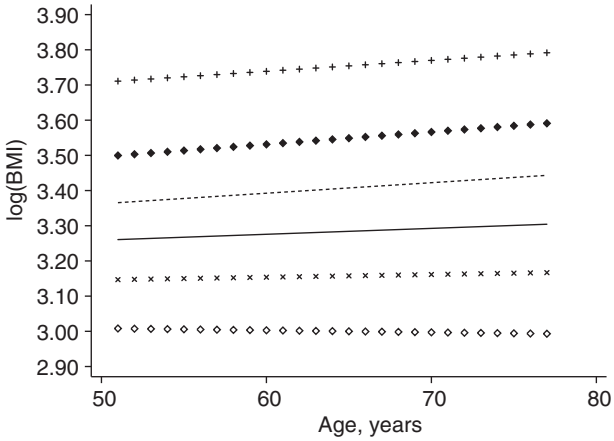

Figure 1. Six latent body mass index (BMI) trajectories after 51 years of age in the 1931–1941 Health and Retirement Study Cohort, 1992–2008. The BMI trajectories are as follows: +, class II/III obese upwards; solid diamonds, class I obese upwards; dashed line, overweight obesity; solid line, overweight stable; x, normal weight upward; and open diamond, normal weight downward.

class II/III obese upward trajectories increased to 3% ($P > 0.05$), 30% ($P < 0.001$), and 147% ($P < 0.001$), respectively, whereas the excess risks associated with the normal weight downward and normal weight upward trajectories decreased to 76% ($P < 0.001$) and 9% ($P > 0.05$).

The next 2 models were adjusted for baseline ADL difficulties, 7 measures of chronic illness, and self-rated health, and the associations of the 5 trajectories with mortality remained significant and in the same direction. Stratifying the analyses by sex returned comparable findings, which are presented in Web Table 2. Finally, we calculated the population attributable mortality risk fraction using hazard ratios from the model adjusted for behavioral factors, as was done in other studies (1, 34). The mortality risks attributable to class I obese upward trajectory and class II/III obese upward trajectory were 3.0% and 4.2%, respectively, when compared with the overweight stable trajectory.

In Table 3, we constrained the analysis to the healthiest subsamples (those with no difficulties with ADL, no preexisting illness, or excellent/very good/good self-rated health) because we might get more accurate estimates of the relationship between obesity and mortality among healthy people who experienced few comorbid illnesses and competing mortality causes (34, 35). We found that the deleterious associations of the class I obese upward and class II/III upward trajectories were generally greater among the healthiest individuals than in the whole sample. The excess mortality risks associated with these 2 trajectories were 16% ($P > 0.05$) and 103% ($P < 0.001$), respectively, among people with no difficulty with ADL; 39% ($P < 0.05$) and 172% ($P < 0.001$), respectively, among people with no preexisting chronic illness; and 24% ($P < 0.05$) and 158% ($P < 0.001$), respectively, among people who reported their health as good or better.

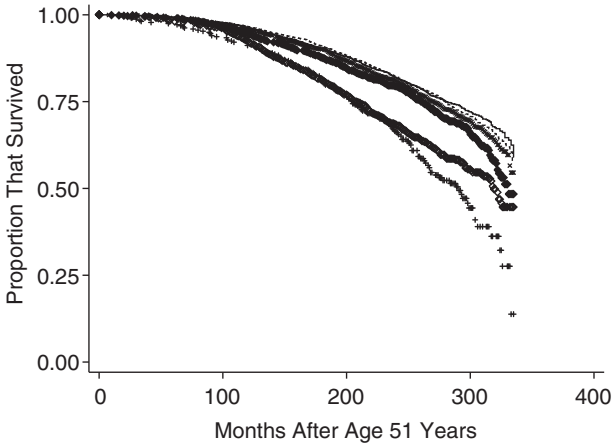

Figure 2. Kaplan-Meier survival curve among persons in 6 body mass index trajectories in the 1931–1941 Health and Retirement Study Cohort, 1992–2008. The body mass index trajectories are as follows: +, class II/III obese upwards; solid diamonds, class I obese upwards; dashed line, overweight obesity; solid line, overweight stable; x, normal weight upward; and open diamond, normal weight downward.

DISCUSSION

Little research has addressed the heterogeneity and mortality risk of BMI trajectories in older populations. The present study focused on BMI trajectories past 51 years of age in the original participants in the HRS who were born between 1931 and 1941. People who were overweight at 51 years of age and remained overweight through age 77 years had the lowest mortality risk. People who were in the class II/III obese category at age 51 years and gained weight through age 77 years had the highest mortality risk. Compared with the overweight stable trajectory, the class I obesity upward and class II/III obese upward trajectories were significantly associated with 30% and 147% increases in mortality risk, respectively, without controlling for confounding health factors. The hazard ratios decreased after controlling for these confounding factors. The deleterious effects of these 2 trajectories are greater among people with no preexisting chronic illnesses or ADL limitations and those who reported their health as good or better at baseline. This is consistent with several studies (35–37) that have found that obesity leads to a higher mortality risk among healthy people.

The differences between the overweight stable and overweight obesity trajectories were not statistically significant. This finding suggests that in people who are overweight at 51 years of age, small weight gains do not lower the probability of survival. By contrast, weight gain in obese people (either class I or class II/III obese) increases their mortality risk. These findings indicate the associations of weight gain with mortality risk depend on baseline BMI status. Many previous studies have found that weight gain was associated with a higher mortality risk in overweight/obese individuals (8); however, we found that weight gain does harm obese individuals but does not harm not overweight individuals. These inconsistencies may result from prior studies using

Table 2. Adjusted Hazard Ratios of Body Mass Index Trajectories From Cox Proportional Hazard Models in the 1931–1941 Health and Retirement Study Cohort, 1992–2008

Covariate	No. of Persons	No. of Deaths	Total		Adjusted for Sociodemographic Factors[a]		Adjusted for Behavioral Factors[b]		Adjusted for Disease Factors[c]		Fully Adjusted[d]	
			HR	95% CI	HR	95% CI	HR	95% CI	HR	95% CI	HR	95% CI
Overweight stable[e] (referent)	2,851	670	1.00		1.00		1.00		1.00		1.00	
Normal weight[f] downward	792	311	1.86	1.63, 2.13	2.12	1.85, 2.43	1.76	1.53, 2.02	1.69	1.47, 1.94	1.64	1.43, 1.88
Normal weight[f] upward	2,312	598	1.12	1.00, 1.24	1.17	1.04, 1.30	1.09	0.97, 1.21	1.09	0.97, 1.21	1.09	0.98, 1.22
Overweight obesity[e]	2,151	504	1.02	0.91, 1.15	1.00	0.89, 1.12	1.03	0.92, 1.15	1.01	0.90, 1.13	0.99	0.88, 1.11
Class I obese[g] upward	1,115	304	1.27	1.11, 1.46	1.25	1.09, 1.43	1.30	1.13, 1.49	1.19	1.03, 1.36	1.11	0.97, 1.28
Class II/III obese[h] upward	317	139	2.28	1.90, 2.74	2.28	1.90, 2.75	2.47	2.04, 2.96	1.97	1.63, 2.38	1.83	1.52, 2.21
BIC			43,944		43,549		43,163		42,824		42,629	

Abbreviations: BIC, Bayesian information criterion; CI, confidence interval; HR, hazard ratio.

[a] Adjusted for sex, race/ethnicity, marital status, educational level, and income.

[b] Adjusted for sex, race/ethnicity, marital status, educational level income, smoking status, and physical activities.

[c] Adjusted for sex, race/ethnicity, marital status, educational level, income, smoking status, physical activities, activities of daily living limitations, angina, heart failure or heart attack, arthritis, bronchitis or emphysema, cancer, diabetes, stroke, and bone fracture.

[d] Adjusted for sex, race/ethnicity, marital status, educational level, income, smoking status, physical activities, activities of daily living limitations, angina, heart failure or heart attack, arthritis, bronchitis or emphysema, cancer, diabetes, stroke, bone fracture, and self-rated health.

[e] Overweight was defined as having a body mass index between 25 and 29.9.

[f] Normal weight was defined as having a body mass index between 18.5 and 24.9.

[g] Class I obesity was defined as having a body mass index between 30 and 34.9.

[h] Class II/III obesity was defined as having a body mass index greater than or equal to 35.

Table 3. Hazard Ratios of Body Mass Index Trajectories From Cox Proportional Hazard Models[a] Among the Healthier Sample in the 1931–1941 Health and Retirement Study Cohort, 1992–2008

Covariate	No Difficulty With Activities of Daily Living[b] (*n* = 8,481)		No Preexisting Chronic Illness Sample[b] (*n* = 3,901)		Excellent/Very Good/Good Self-Rated Health Sample[b] (*n* = 7,389)	
	HR	95% CI	HR	95% CI	HR	95% CI
Overweight stable[c] (referent)	1.00		1.00		1.00	
Normal weight[d] downward	1.77	1.51, 2.07	1.57	1.20, 2.04	1.74	1.45, 2.10
Normal weight[d] upward	1.13	1.00, 1.28	1.09	0.89, 1.32	1.08	0.94, 1.25
Overweight[c] obesity	1.04	0.91, 1.18	1.07	0.86, 1.32	1.06	0.91, 1.23
Class I obese[e] upward	1.16	0.99, 1.35	1.39	1.06, 1.82	1.24	1.03, 1.50
Class II/III obese[f] upward	2.03	1.62, 2.56	2.72	1.78, 4.15	2.58	1.98, 3.36
BIC	33,515		11,047		24,753	

Abbreviations: BIC, Bayesian information criterion; CI, confidence interval; HR, hazard ratio.

[a] All models were fully adjusted for demographic, socioeconomic, behavioral, disease, and health indicators.

[b] The number of deaths were 2,004, 723, and 1,495 among people who had no difficulty with activities of daily living, no preexisting illness, or excellent/very good/good self-rated health, respectively.

[c] Overweight was defined as having a body mass index between 25 and 29.9.

[d] Normal weight was defined as having a body mass index between 18.5 and 24.9.

[e] Class I obesity was defined as having a body mass index between 30 and 34.9.

[f] Class II/III obesity was defined as having a body mass index greater than or equal to 35.

arbitrary cutoff points for weight change or assuming a linear function of the weight-change effect across BMI status, which may have yielded overdeterministic results. Weight loss, even a small one (a decrease of approximately 1 BMI unit), in a person in the normal weight category 51 years of age can potentially have a significant deleterious effect on health. Many previous studies found that even small weight losses can exert a harmful effect on survival, regardless of the initial BMI level (22).

Table 4. Adjusted Hazard Ratios of Baseline Body Mass Index Status From Cox Proportional Hazard Models[a] in the 1931–1941Health and Retirement Study Cohort, 1992–2008

Covariate	Fully Adjusted	
	HR	95% CI
Underweight[a]	2.28	1.78, 2.92
Normal weight[b]	1.08	0.98, 1.19
Class I obese[c]	1.06	0.95, 1.19
Class II/III obese[d]	1.61	1.40, 1.85
BIC	42,634	

Abbreviations: BIC, Bayesian information criterion; CI, confidence interval; HR, hazard ratio.

[a] All models were fully adjusted for demographic, socioeconomic, behavioral, disease, and health indicators.

[b] Underweight was defined as having a body mass index less than 18.5.

[c] Normal weight was defined as having a body mass index between 18.5 and 24.9.

[d] Class I obesity was defined as having a body mass index between 30 and 34.9.

[e] Class II/III obesity was defined as having a body mass index greater than or equal to 35.

The associations of BMI trajectory with mortality are stronger than the associations of initial BMI status alone. Table 4 presents mortality risks by baseline BMI status. Persons in the underweight and class II/III obese categories had increased mortality risk compared with the reference category (overweight). Normal weight and class I obesity were not associated with significant increases in mortality risk. These findings are consistent with those from the analysis by Mehta and Chang (34). The effect sizes of these BMI statuses (normal weight, class I obese, and class II/III obese) were smaller than the corresponding effect sizes of BMI trajectories in Table 2. The Bayesian information criterion statistic suggests the model has a better fit when using BMI trajectories than when using BMI status, which supports previous studies in which it was concluded that weight change is more predictive of mortality than is initial weight status alone (5, 6).

This study has several limitations. First, the BMI measures were constructed from self-reported weight and height and are therefore subject to potential bias. However, self-reported and clinically measured height and weight are strongly correlated (22, 34, 38), although the extent of this correlation among HRS respondents is unknown. Moreover, differential biases in weight reporting may have accumulated over time and further biased our estimates. However, there is no reason to assume that the bias in weight and height reporting varied across any of the 6 trajectories. Therefore, using self-reported weight and height should not have introduced substantial bias to our analysis. Second, we were not able to trace the BMI trajectories to the earlier stages of the life course. It may be important to investigate whether BMI trajectories in early and middle adulthood display similar heterogeneity and whether this heterogeneity has similar implications for mortality risk.

Third, we were not able to differentiate between intentional and unintentional weight changes, particularly weight losses. However, prior studies have found that intentional

weight loss has, at best, weaker detrimental effects on mortality and not the anticipated protective effect (39, 40). Moreover, we have controlled for a wide range of underlying health problems and functional limitations that may lead to unintentional weight change, thereby estimating the net effect of weight change. Fourth, although BMI is the most commonly used measure of adiposity, it has been criticized as not being able to directly measure body fat and muscle composition or distinguish between central and peripheral adiposity (41). Although some datasets (e.g., the dataset from the National Health and Nutrition Examination Survey IV, 1999–2004) have data from more accurate and direct measures of body composition, such as dual energy x-ray absorptiometry, they do not track long-term changes in these measures.

Improving upon prior studies, we investigated the association of dynamic BMI trajectories with mortality risk. In one previous study, investigators examined BMI trajectories over time instead of over the life course in the HRS, but they detected less heterogeneity in BMI trajectories (42) (for a more detailed comparison, please refer to Web Appendix 2). We found people in the overweight stable trajectory had the lowest mortality risk, followed by people in the overweight obesity, normal weight upward, class I obese upward, normal weight downward, and class II/III obese upward trajectories. The lower mortality risk among people in the the overweight trajectories is consistent with the view that extra body weight, including lean tissue mass and fat mass, may provide protection against nutritional and energy deficiencies, metabolic stresses, the development of wasting and frailty, and loss of muscle and bone density caused by chronic diseases such as heart failure (35, 41, 43).

Mortality risks attributable to the class I obese upward trajectory and the class II/III obese upward trajectory were 3.0% and 4.2%, respectively, compared with the overweight stable trajectory. In total, approximately 7.2% of deaths after 51 years of age in the 1931–1941 birth cohort were due to obesity upward trajectories. These estimates are larger than those of Mehta and Chang (34) (5.1% and 4.7% for obese females and males, respectively), who used baseline BMI measures with reference to overweight status in the same dataset and same cohort of respondents. This comparison again demonstrates that BMI trajectories are more predictive of mortality risk than are initial BMI statuses. Our estimates are not directly comparable to those obtained by Allison et al. (2), Mokdad et al. (3, 4) or Flegal et al. (1) because of the different age groups in the samples. Their studies included adults of all ages, whereas ours focused on people 51 years of age or older. Because of the age-dependent nature of the BMI-mortality link (i.e., a stronger correlation among younger adults (44–47)), we might have observed an even larger association for the 1931–1941 birth cohort if we could take into account the risk of dying before the age 51 years. Our study suggests that trajectories of increasing obesity past 51 years of age pose a substantive threat to future life expectancy increases.

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