THE AUTHORS REPLY

Mehta and Stokes (1) urge caution when interpreting the age patterns of the obesity-mortality relationship found in our study (2). They reference findings that suggested that the obesity-mortality association strengthens with time in study (i.e., length of mortality follow-up) and contend that these patterns likely reflect reverse causal pathways. Specifically, they suggest that some survey respondents will have experienced recent weight loss because of a life-threatening disease and, consequently, will also be at a high risk of death during the early period of follow-up. As a result, estimates of the age patterns of the obesity-mortality relationship will be downwardly biased, especially in early periods of follow-up. Mehta and Stokes contend that we missed this biasing factor and that our conclusion that the impact of obesity on mortality remains strong at older ages could be artifactual. We agree that disease-induced weight loss can downwardly bias estimates of the association between body mass index (BMI, measured as weight (kg)/height $(m)^2$) and mortality during the first years of time in study. We disagree that our approach failed to address this bias.

To understand why, we direct readers to Figure 4A and 4B in the article by Zajacova and Burgard (3) that was referenced by Mehta and Stokes. The figure demonstrated that BMI effects that were sensitive to early follow up in the National Health Interview Survey-Linked Mortality Files were largely confined to participants in the low BMI range. In keeping with this observation and cognizant of the potential bias that Mehta and Stokes noted, our design omitted the low BMI group from our analyses. Doing so limited the confounding effects of disease-induced weight loss and increased our confidence that the bias to which Mehta and Stokes point could not be the source of the rising marginal impact of high BMI relative to normal BMI that we reported.

Although we are confident that the bias is the not the explanation for our main findings, we do find the speculations of Mehta and Stokes of interest and wonder if there could be any data to support them. To that end, we reasoned that if the increases in the obesity-mortality association unfold with time in study (as a product of reverse-causation) instead of across increasing age, then the following ought to hold. 1) The rate of compositional change in underlying health must be faster in the normal-weight sample than in the obese sample; that is, attrition among the terminally ill in the normal-weight sample ought to manifest in compositional changes by which the normal-weight sample becomes "healthier" across the first few years of time in study relative to the obese sample. This process can be verified by directly observing the attrition-related compositional change in a prospective data set, such as the Health and Retirement Survey. 2) As a direct result of point 1, the sample ought to become more composed of obese respondents in the first few years of mortality follow up because the terminally ill participants in the normal-weight sample die at earlier times. 3) Finally, limiting model analyses to data that have exposure times greater than 1, 2, or 3 years ought to significantly and substantively affect the rising age patterns of the obesity-mortality association.

As a test of point 1, we provide several figures that illustrate percent changes in the Health and Retirement Survey in a normal-weight sample (Figure 1), a grade 1 obese (BMI of 30–34.9) sample (Figure 2), and a grade 2/3 obese (BMI ≥35.0) sample (Figure 3) that self-reported health as poor, fair, good, very good, or excellent as a result of differential survival across time in study; the percent increases in the number of respondents from each sample who self-rated their health as very good or excellent that resulted from differential survival across time in study (Figure 4); and the percent changes in the number of respondents who reported heart problems (Figure 5). All evidence suggests that compositional changes resulting from differential attrition more strongly affect the obese sample than the normal-weight sample, which is the opposite of what one would expect if the conjecture by Mehta and Stokes was apt. (Other measures of health—for example, number of health conditions and whether or not health limits work—show similar compositional changes.)

Consequently, and to address point 2, the grade 2/3 subsample accounted for a decreasing proportion of the overall Health and Retirement Survey sample across time in study (Figure 6) while simultaneously becoming "healthier" via attrition at a faster rate than the normal-weight sample. Again, these findings are just the opposite of what one would expect based on the ideas presented by Mehta and Stokes. Finally, to address point 3, we provide Table 1, which contains estimated age-specific hazard ratios of US men's mortality risk by BMI group from 3 models (results for US women are consistent). Model 1 limited survival analyses to a time in study greater than 1 year, model 2 limited survival analyses to a time in study greater than 2 years, and model 3 limited survival analyses to a time in study greater than 3 years. Estimates from each model are consistent with the age patterns we presented in our article (2), indicating that the impact of obesity on mortality risk grows stronger with age.

Our empirical exercises performed on data from both the Health and Retirement Survey and National Health Interview Survey-Linked Mortality Files are inconsistent with the contention that the obesity-mortality association changes across time in study but are consistent with the suggestion that the association changes across attained age. Finally, although we agree with Mehta and Stokes that caution is needed, our empirical tests of their idea redirect concern away from our findings to the alternative they proposed.

ACKNOWLEDGMENTS

Conflict of interest: none declared.

REFERENCES

1. Mehta NK, Stokes A. Re: "Obesity and US mortality risk over the adult life course" [letter]. *Am J Epidemiol*. 2013; 178(2):320.
2. Masters R, Powers D, Link B. Obesity and US mortality risk over the adult life course. *Am J Epidemiol*. 2013;177(5): 431–442.
3. Zajacova A, Burgard S. Shape of the BMI-mortality association by cause of death, using generalized additive models: NHIS 1986–2006. *J Aging Health*. 2012;24(2):191–211.

Ryan K. Masters[1], Daniel A. Powers[2], and Bruce G. Link[1] (e-mail: ryan.masters@colorado.edu)
[1] *Mailman School of Public Health, Columbia University, New York, NY 10032*
[2] *Department of Sociology, University of Texas at Austin, Austin, TX 78712*

DOI: 10.1093/aje/kwt118; Advance Access publication: June 11, 2013

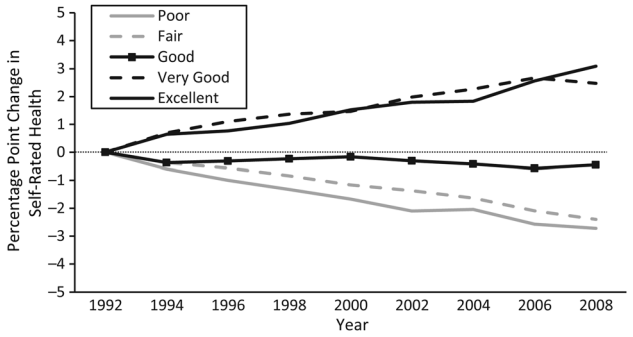

Figure 1. Absolute percentage point change in self-rated health due to respondents' differential survival among respondents with a normal-weight body mass index (weight (kg)/height $(m)^2$) (18.5–24.9), Health and Retirement Survey, 1992–2008.

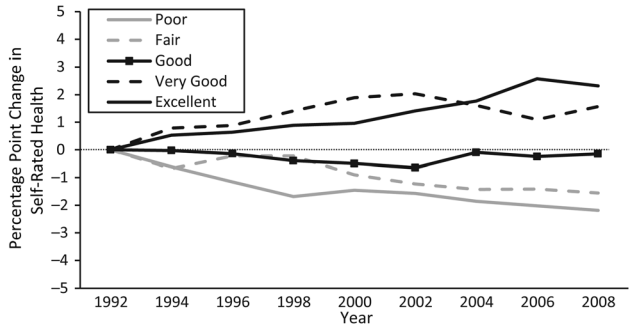

Figure 2. Absolute percentage point change in self-rated health due to respondents' differential survival among respondents with a grade 1 obese body mass index (weight (kg)/height $(m)^2$) (30.0–34.9), Health and Retirement Survey, 1992–2008.

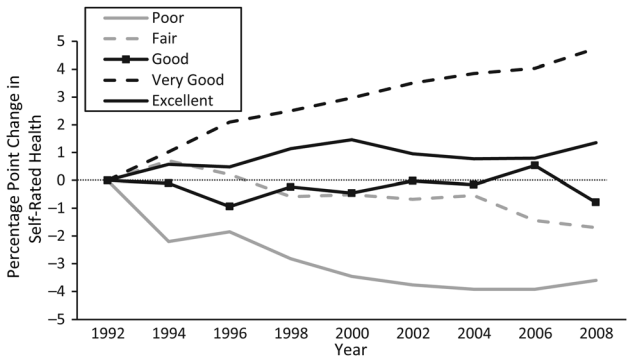

Figure 3. Absolute percentage point change in self-rated health due to respondents' differential survival among respondents with a grade 2/3 obese body mass index (weight (kg)/height $(m)^2$) (≥35.0), Health and Retirement Survey, 1992–2008.

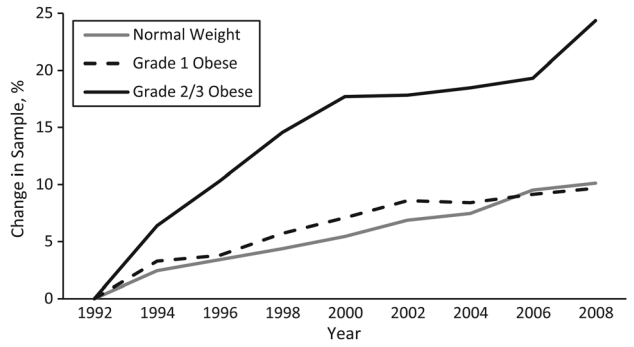

Figure 4. Percentage of change due to respondents' differential survival in the number of respondents who self-rated their health as very good or excellent, by body mass index, Health and Retirement Survey, 1992–2008. Grade 1 obesity refers to a body mass index (weight (kg)/height $(m)^2$) of 30–34.9 and grade 2/3 obesity refers to a body mass index ≥35.0.

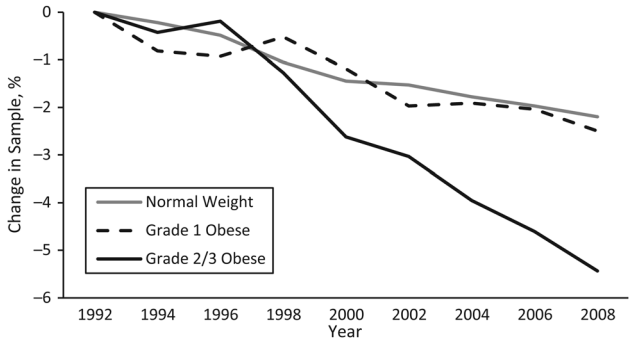

Figure 5. Percentage of change due to respondents' differential survival in the number of respondents who reported heart disease, by body mass index, Health and Retirement Survey, 1992–2008. Grade 1 obesity refers to a body mass index (weight (kg)/height $(m)^2$) of 30–34.9 and grade 2/3 obesity refers to a body mass index ≥35.0.

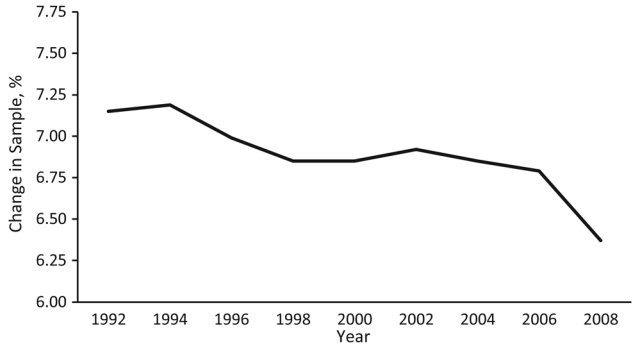

Figure 6. Percentage of change due to respondents' differential survival in the number of respondents with a grade 2/3 obese body mass index (weight (kg)/height $(m)^2$) (≥35.0), Health and Retirement Survey, 1992–2008.

Table 1. Estimated Hazard Ratios From Cox Survival Models[a] That Examined the Association Between Obesity and Mortality Restricted to Time in Study[b] in Adult Men, United States, 1986–2006

Obesity Status by Age Group, years	Model 1[c] (*n* = 757,988)		Model 2[d] (*n* = 682,319)		Model 3[e] (*n* = 602,869)	
	HR	95% CI	HR	95% CI	HR	95% CI
Grade 1 obesity[f]						
25–34	0.76	0.54, 1.05	0.81	0.56, 1.16	0.70	0.46, 1.07
35–44	1.04	0.91, 1.19	1.07	0.92, 1.24	1.13	0.96, 1.32
45–54	1.13	1.04, 1.22	1.17	1.07, 1.27	1.16	1.06, 1.28
55–64	1.27	1.20, 1.35	1.27	1.19, 1.35	1.28	1.19, 1.37
65–74	1.42	1.30, 1.54	1.39	1.27, 1.52	1.41	1.27, 1.56
75–84	1.65	1.47, 1.86	1.56	1.37, 1.77	1.58	1.37, 1.82
≥85	1.53	1.31, 1.79	1.38	1.15, 1.66	1.35	1.10, 1.67
Grade 2 obesity[g]						
25–34	1.43	0.91, 2.25	1.28	0.75, 2.20	1.31	0.70, 2.46
35–44	1.44	1.15, 1.80	1.41	1.12, 1.77	1.46	1.13, 1.88
45–54	1.53	1.33, 1.75	1.48	1.27, 1.72	1.39	1.17, 1.65
55–64	1.63	1.45, 1.84	1.76	1.55, 2.00	1.71	1.48, 1.98
65–74	1.91	1.64, 2.22	2.11	1.79, 2.48	2.23	1.85, 2.69
75–84	2.13	1.73, 2.61	2.37	1.90, 2.94	2.46	1.91, 3.15
≥85	2.09	1.50, 2.90	2.45	1.72, 3.49	2.67	1.77, 4.04
Grade 3 obesity[h]						
25–34	2.56	1.31, 5.01	2.63	1.29, 5.36	2.77	1.26, 6.10
35–44	2.17	1.54, 3.05	2.29	1.60, 3.28	2.39	1.61, 3.53
45–54	2.35	1.92, 2.86	2.41	1.94, 3.00	2.46	1.94, 3.12
55–64	2.87	2.39, 3.43	2.82	2.31, 3.43	2.81	2.26, 3.48
65–74	2.48	1.91, 3.21	2.42	1.78, 3.29	2.19	1.56, 3.08
75–84	3.17	2.10, 4.79	3.18	2.05, 4.92	2.82	1.78, 4.47
≥85	2.14	0.83, 5.54	2.52	0.84, 7.57	1.93	0.59, 6.31

Abbreviations: BMI, body mass index; CI, confidence interval; HR, hazard ratio.

[a] All models were adjusted for educational attainment, income, marital status, region of residence, race/ethnicity, 5-year birth cohort, and age at the time of interview.

[b] Data were obtained from the National Health Interview Survey Linked Mortality Files.

[c] Model 1 was restricted to the sample who had spent more than 1 year in the study.

[d] Model 2 was restricted to the sample who had spent more than 2 years in the study.

[e] Model 3 was restricted to the sample who had spent more than 3 years in the study.

[f] Grade 1 obesity refers to a body mass index (weight (kg)/height $(m)^2$) of 30–34.9.

[g] Grade 2 obesity refers to a body mass index of 35.0–39.9.

[h] Grade 3 obesity refers to a body mass index of 40.0 or higher.