term equals 0 or, in other words, age-at-survey equals mean age. It makes no biological sense to estimate hazard ratios under this assumption for most age groups. For example, it is impossible to interpret hazard ratios for men aged 35–44 years by assuming their age-at-survey is 47.31 years (mean age).

I have recalculated hazard ratios on the basis of all relevant numbers in their final models according to the above formula. These recalculated hazard ratios are approximate estimates because I did not use the original raw data. Nevertheless, the trend is clear (Figure 1 and Table 1). The obesity-mortality association actually weakened substantially with increasing age, which contradicts their conclusion of growing stronger with age. When multiple regression techniques are used to control for confounders, particularly with interaction terms involving the exposure of interest, authors should interpret their findings in the context of complete relevant information generated by the model rather than focusing only on interpreting coefficients of main effect estimates.

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THE AUTHORS REPLY

We thank Dr. Wang (1) for commenting on our paper (2), as well as for raising important points regarding the fitting of survival models and the interpretation of their results. Our response is 3-fold. First, we stand by our use of age-at-survey as a proxy for likely confounders of the US obesity-mortality association (i.e., cohort differences in mortality risk and age-graded survey selection). We accounted for these factors by modeling effects of respondents’ 5-year birth cohort and age-at-survey, as well as 2-way effects between body mass index (weight (kg)/height (m)²) and age-at-survey. Dr. Wang is correct that age-at-survey and attained age are collinear. However, collinearity between age-at-survey and attained age does not preclude partitioning age-related variation into these 2 distinct sources (3, 4). Moreover, if the goal is to separate these sources of variation, it is inappropriate to reintroduce the very source of variation for which one is controlling (i.e., age-at-survey). That said, we recognize Dr. Wang’s point that it is biologically implausible to standardize age-at-survey on the mean (47.31 years) for those participants in the National Health Interview Survey (NHIS) whose attained age was less than the mean.

Second, to address Dr. Wang’s point, we refit survival models on the National Health Interview Survey Linked Mortality Files (NHIS-LMF), stratified by body mass index, for women with attained age of 50–84 years and standardized age-at-survey of 50 years (obese classes 2 and 3 were combined because of small counts of death at individual ages). Consistent with the results presented in our paper (2), Figure 1 illustrates the relative decline in the obesity-mortality association across attained age from survival models that do not account for variation from age-at-survey, whereas Figure 2 illustrates that the obesity-mortality association grows substantially stronger across attained age after standardizing by birth cohort and age-at-survey. Thus, the
than all hazards estimated in the NHIS-LMF data. Furthermore, overweight, and obese populations, yet of the mortality experiences of the underweight, normal weight, NHI-LMF) (5).

Results presented in our paper (2) are replicated on a subsample of data for which the age range appropriately conforms to ages-at-survey and attained ages.

Finally, we end by making an important distinction between 1) fitting models to describe data and 2) fitting models to help describe important processes in the population of interest. We believe that our ultimate goal as mortality researchers is the latter. Dr. Wang, conversely, seems most concerned about the former. To show the importance of this distinction, we compare the smoothed hazards from the NHIS-LMF samples of US women with the $q_x$ in the official 1999 US female life tables (1999 is the average year of death in the NIH-LMF) (5).

Results presented in Figures 3–5 help to illustrate the importance of our distinction between points 1 and 2 above. Specifically, survival models that do not account for age-at-survey estimate hazards that are both 1) unrealistically high for the normal/overweight sample and 2) suspiciously low for the class 1 and class 2/3 obese samples (Figure 3). The estimates are, in fact, mathematically impossible because $q_x$ must be an average of the mortality experiences of the underweight, normal weight, overweight, and obese populations, yet $q_x$ is substantially lower than all hazards estimated in the NHIS-LMF data. Furthermore, the older US female population is overwhelmingly composed of the normal weight or overweight populations (6). Thus, the age-specific hazards for the normal/overweight sample should approximate the official $q_x$. As seen in Figure 3, this is not the case. Further still, the difference between the normal/overweight hazard and $q_x$ grows larger with increasing age (Figure 5), despite the fact that the older population becomes less obese with age. This model, although presumably preferred by Dr. Wang because it most accurately describes patterns in the NHIS-LMF data, poorly describes the population of interest. Contrast these hazards with those in Figure 4, which are based on models fitted to the normal/overweight, class 1 obese, and class 2/3 obese samples while accounting for 5-year birth cohort and standardizing age-at-survey. The estimated hazards from these models are much closer to the empirical patterns in the US female population.

We conclude by emphasizing that our interest should not be in describing the data—especially if we believe those data to be biased—but rather we should use theory, data, and models to accurately describe population processes of interest. Evidence suggests that our models, which are attentive to cohort differences in mortality risk and age-related selection processes, comply more with empirical mortality patterns in the US population than do models that ignore these sources of variation. When we use these models to investigate the US obesity-mortality association, we find no evidence that the obesity-mortality association weakens with age. Rather, evidence points to a stronger association with increasing age.

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