Thanks for your interest in our response to the editorial statement by the American Journal of Public Health (AJPH) and the American Journal of Epidemiology (AJE) concerning the methods used in Masters et al. (2013).

We organize our response by addressing central points about Dr. Hanley's simulation exercises regarding our approach to estimating age-specific hazard ratios in the National Health Interview Survey-Linked Mortality Files (NHIS-LMF) data. By addressing these points, we answer Professor Hanley's central question, "Is the cure worse than the bias?" with a definitive "No". As we'll show using his own example regarding the age-specific male:female (m:f) ratios in mortality risk, the approach we used to estimate mortality hazards in the NHIS-LMF data is preferred to conventional approaches. The exercise is a useful test of our approach to estimating obesity hazard ratios (HRs) in the NHIS-LMF data because we can use the observed population m:f hazard ratios to gauge the accuracy of different models' estimates. We show that our "cure" was the preferred approach for estimating male-female differences in mortality risk in the NHIS-LMF data, which, by extension, provides strong evidence in support of our approach to estimating obesity HRs in the same data.

We also address Professor Hanley's concern about the simplicity of our model assumptions (e.g., centering age-at-survey on the mean value) by presenting results from models that were refitted by centering age-at-survey on more plausible ages in the NHIS-LMF data. Although the age-specific hazard ratios change, the overall results of our paper do not. Our original estimates suggested that 18.2% of U.S. adult deaths between ages 40 and 84 among the non-Hispanic white and black populations in the 1986-2006 period was associated with high BMI levels (95% confidence interval: 9.8-27.1). Results using age-specific hazard ratios from new models put the figure at about 16.8% (95% confidence interval: 7.1-26.0).

Professor Hanley correctly points out that attenuation of HRs between sub-groups often occurs in unselect populations. Indeed, attenuation is actually quite common when sub-groups experience differential risk across long durations of time (Vaupel and Yashin 1985; Vaupel et al. 1979). The implication of this point is that the attenuation of obesity HRs observed in the NHIS-LMF data could, in fact, be true. Thus, our modeling approach used to adjust for health-related selection into the NHIS survey might be unnecessary and even introduce bias into our estimates of obesity HRs. Professor Hanley uses the case of male-female differences in age-specific mortality risk to simulate a scenario of gendered selection in sample data to test whether the approach we used in our paper would accurately estimate the true age-specific differences in male and female mortality risk. That is, could our modeling approach estimate age-specific m:f HRs that attenuate or will the approach estimate biased HRs? Upon first read, this exercise appears to provide clear evidence against the approach we used to adjust for differential selection into the NHIS-LMF data. From Professor Hanley's exercise it appears that the application of our "cure" – adjusting model estimates for NHIS respondents' baseline age-at-survey – is "worse than the bias" stemming from the selection effects. Indeed, when gender differences in age-at-survey are included in the model, Professor Hanley's results show increasing m:f HRs across age when, in fact, the true m:f HRs attenuate to near 1.0. The takeaway is that our modeling approach in the NHIS data is "exchang[ing] one paradox for another" by estimating a rising mortality discrepancy by obesity when, in fact, attenuation might be reality.

This lesson is echoed in Professor Hanley's conclusion when he requests that researchers check their results against "data generated from known parameter values." We fully agree with this sentiment. Therefore, we checked various model estimates against the age-specific mortality rates calculated with official U.S. mortality records and population counts in Census records. Although we didn't use simulations to test model estimates against known data generating parameters, we compared proportionally-weighted estimates of hazards to the known parameter values for age-specific mortality rates in official U.S. population data. That is, we calculated aggregate age-specific mortality rates by combining estimates of age-specific mortality rates from the normal weight, overweight, class 1 obese, and class 2/3 obese samples with the respective age-specific prevalence of each BMI level in the NHIS data. It was through these checks that we decided to use the model that included five-year cohort fixed effects and respondents' baseline age at time of survey. Below, for example, Figure 1 shows the aggregated age-specific mortality rates from different models fitted to the non-Hispanic white women's sample (this figure was provided in a previous exchange with the AJPH editorial office), and Figure 2 shows the estimated amount of bias in model estimates at older ages.

Figure 1. Estimated Age-specific M_x from Survival Models with 2000 Official M_x, US White Women.

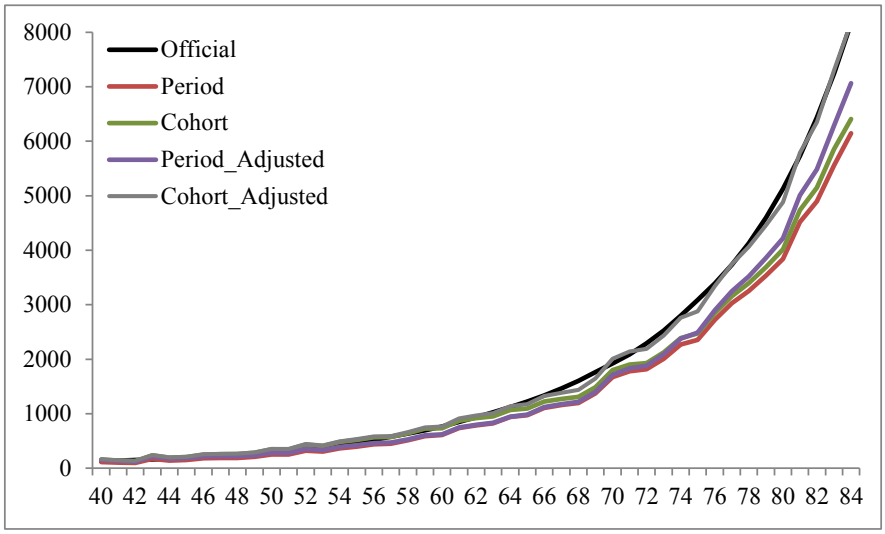
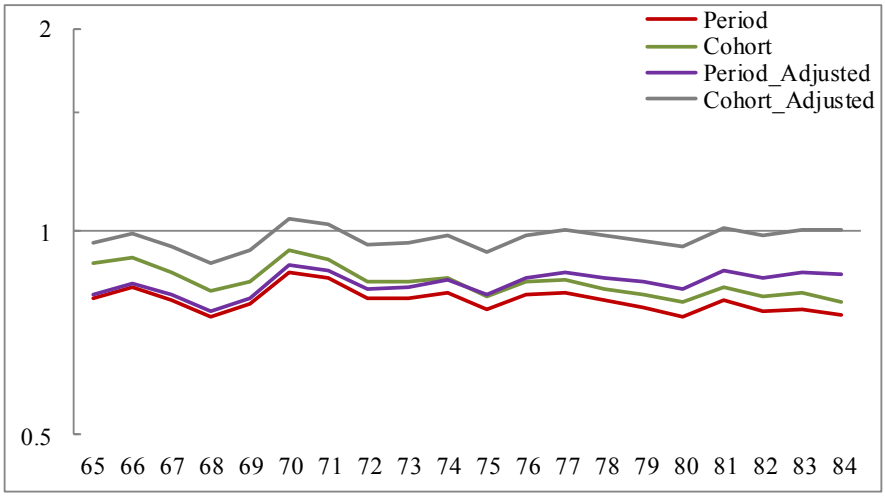


Figure 2. Rate Ratio of Estimated M_x from Survival Models to 2000 Official M_x, US White Women



The models that do not account for age-at-survey (i.e., "Period" and "Cohort" models) both estimate older age mortality rates that are about 20% below the rates observed in the official data. The "Period_Adjusted" model, which accounts for respondents' ages at time of survey, reduces this bias at the oldest ages, but by only a modest amount. The estimated mortality rates from the "Cohort_Adjusted" model, conversely, reveal no pattern of systematic biases at these older ages, and were therefore preferred models for estimating the age-patterns of the obesity-mortality association in the US adult population. Thus, we used the "Cohort_Adjusted" models to estimate age-specific mortality differences by BMI-group in the US non-Hispanic black and white male and female populations, and used the estimated age-specific hazard ratios in the PAF equations.

We therefore used the known parameter values in official mortality rates in the U.S. adult population to guide our modeling strategy by testing different model estimates against true rates. Yet because BMI status is not recorded on official death records, one cannot compare models' estimates of HRs by high BMI status to those observed in the actual population. Thus, Professor Hanley used the case of m:f HRs in real populations to simulate how gender differences in age-specific survey selection might produce biased hazard ratios, and to observe how the approach we used might attend to these biases. To do so, Professor Hanley assumed that male-female differences in sample selection would manifest as differences in mortality hazards during the first *five* years of mortality follow-up. He simulates such a selection process among *men* and not among women, and then refitted survival models controlling for age-at-survey to see how well (or poorly) the model adjusted HR estimates for the selection biases.

However, a more straightforward and realistic test of our modeling approach would be to observe the m:f HRs in age-specific mortality rates in U.S. official mortality data for years 1986-2006, and then to compare these true HRs to the age-specific HRs in the NHIS-LMF 1986-2006 estimated from different models. By comparing the m:f HRs observed in the actual U.S. population to the m:f HRs estimated in the NHIS-LMF data, we can assess the size and direction of discrepancies between sample estimates of HRs and the known true HRs. We could then directly test how our approach might adjust estimates to match the true HRs. Such a comparison

is very useful because *it removes the need to impose simple and possibly incorrect assumptions about the size, direction, and duration of selection biases in simulated data*. For example, in his simulation exercise, Dr. Hanley assumes positive-health selection among men when, in fact, it is more likely that positive-health selection exists among older women in the NHIS. We know, for instance, that living arrangements in the United States differ substantially for older men and women, with older men much less likely to live alone. In terms of the time range that we analyzed in the NHIS data (1986-2006), Current Population Survey (CPS) data show that 31.5 percent of women over the age of 65 lived alone in 1990 compared with only 8.4 percent of men, and that respective percentages in 2000 were 27.8 compared with 8.8 (Vespa, Lewis, and Kreider 2013). If we look at the NHIS data themselves, we see large gender differences in marital status, especially for currently married and widowed (see below in Figure 3).

Figure 3. Married and Widowhood Status by Age for Female Respondents (Left Graph) and Male Respondents (Right Graph) of NHIS, 1986-2004.

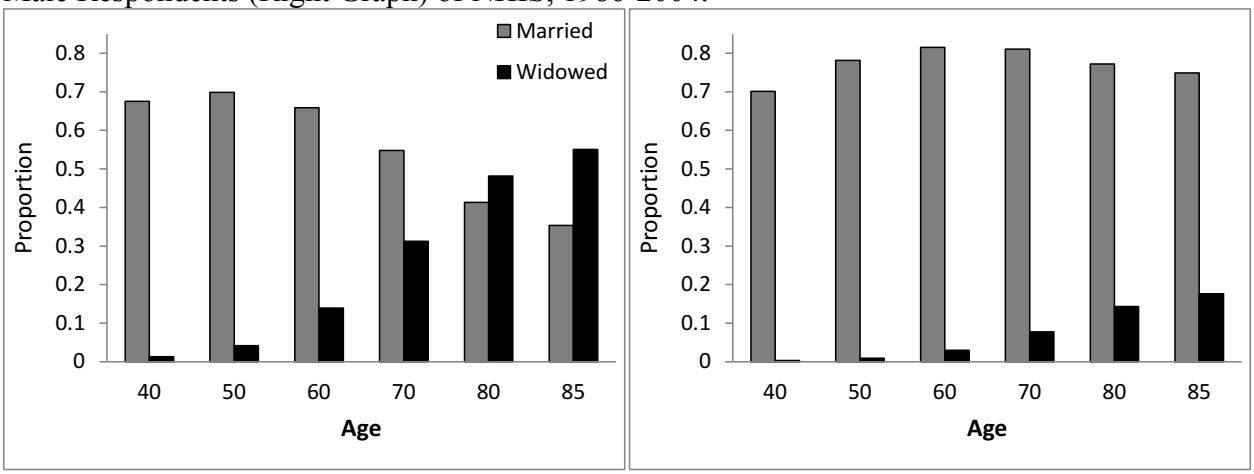


Figure 4. Male-to-Female Relative Rate Ratio of NHIS Reporting Status, 1986-2004.

We also see that men in the NHIS are much more likely than women to have their information provided by a proxy, or to self-report only some information in the survey, and that these gender differences grow larger with age (Figure 4). The point in showing large gender differences in

marital status and reporting status is to provide empirical support for the argument that health selection at older ages is likely operating more among women than among men. That is, older men in poor health are likely still included in the NHIS sampling frame via their spouses' proxy reporting on their behalf. Older women in poor health, conversely, are more likely to reside alone and/or in institutionalized settings that preclude them from participating in the NHIS. As such, those older women who participated fully in the NHIS were likely much more select on health than were men of comparable age. Consequently, *there likely exists gender-based health selection in the NHIS data, but this evidence strongly suggests that works in the direction opposite to the selection assumed to exist in Professor Hanley's simulation exercises*.

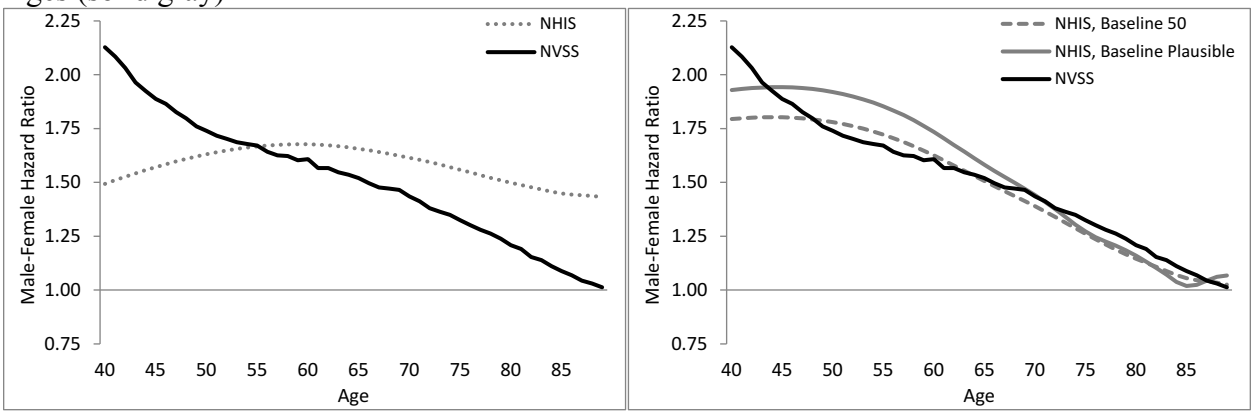
Further, Professor Hanley's simulation assumes that differential survey-selection affects men's mortality risk during only the *first five years* of mortality follow-up, which is consistent with a "reverse causality" explanation (Mehta and Stokes 2013). But we have shown elsewhere (Masters, Powers, and Link 2013) that the time metric over which the obesity-mortality association changes is likely age, not calendar time of mortality follow-up. Further, and consistent with this point, our exploratory analyses showed that the obesity-mortality association grew stronger across *all 19 years* of mortality follow-up in the NHIS-LMF data, inconsistent with both a "reverse causality" explanation as well as with Dr. Hanley's exercise. Again, it is fortunate that Professor Hanley used m:f HRs as an example for his simulation exercise, because in this case we can actually *see* the true m:f HR in U.S. official mortality data (National Vital Statistics Systems [NVSS]). We can then compare different models' (i.e., models fitted using a conventional approach vs. our approach) estimated HRs in the NHIS-LMF data to the actual age-specific HRs in the NVSS data to determine if and how the sample estimates are biased.

In Figure 5 below, we contrast estimated age-specific m:f HRs in the NHIS-LMF data with corresponding HRs calculated with NVSS data. The left panel of Figure 5 shows the age-specific m:f HRs in the official U.S. mortality data for years 1986-2006, which were estimated from a single-year piecewise-constant exponential model controlling for five-year birth cohort (solid black line). Also included in the left panel of Figure 5 are the age-specific m:f HRs in the NHIS-LMF, 1986-2006, which were estimated from a Royston-Parmar survival model that controlled only for five-year birth cohort and an attained age-varying coefficient of male (dashed gray line). In short, this survival model fitted to the NHIS-LMF data allows the m:f HR to vary across age. If the NHIS-LMF data are not biased, then the estimated age-specific m:f HRs in these data should match closely the age patterns of the HRs observed in the NVSS data. The age patterns of the m:f hazard ratios observed in the U.S. official mortality data between 1986 and 2006 show strong attenuation from a relative risk over 2.0 at age 40 to 1.50 at age 60 to almost 1.0 by age 89. In striking contrast, the age-specific m:f HRs estimated from a conventional survival model show no significant attenuation across age in the NHIS-LMF data. Through this simple exercise we see that estimates of age-specific m:f HRs in the NHIS-LMF are severely biased.

The right-hand panel of Figure 5 shows the age-specific m:f HRs in the U.S. official mortality data for years 1986-2006 (solid black line), as well as age-specific m:f HRs in the NHIS-LMF, 1986-2006, estimated from a Royston-Parmar survival model controlling for five-year birth cohort, a time-varying coefficient of male, age-at-survey (centered on age 50), and a two-way interaction between male and age-at-survey (dashed gray line). This model matches closely the approach we used in our paper by adjusting model estimates of age-specific HRs for

respondents' ages at time of survey (centering all ages at survey close to the mean, 50 years). The right-hand panel also plots age-specific m:f HRs in the NHIS-LMF, 1986-2006, estimated from a Royston-Parmar survival model controlling for five-year birth cohort, a time-varying coefficient of male, age-at-survey (centered on plausible ages-at-survey depending on attained age), and a two-way interaction between male and age-at-survey (solid gray line). This model also matches closely the approach we used in our paper, but instead of simply centering all baseline ages on the same value of 50 years, we centered age-at-survey on plausible baseline ages depending on respondents' attained ages (i.e., centered on age 45 if attained age 40-50, centered on age 50 if attained age 50-60, centered on 55 if attained age 60-70, centered on age 60 if attained age 70-80, and centered on age 65 if attained age 80-89).

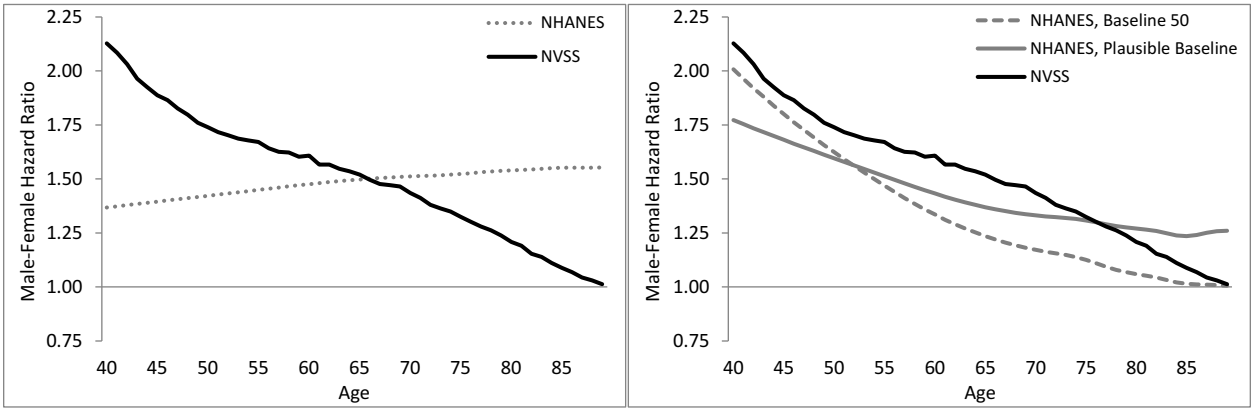
Figure 5. NHIS Male:Female Mortality Hazard Ratio by Age, 1986-2006, vs. NVSS Male:Female Mortality Hazard Ratio, 1986-2006, controlling for five-year birth cohort
Left Panel: No control for NHIS respondents' Baseline Age at time of survey
Right Panel: Center Baseline Age at 50yrs (dashed gray) and Center Baseline Age on Plausible Ages (solid gray)



Unlike the estimated m:f HRs in the NHIS-LMF plotted in the left-hand panel of Figure 5, which are severely biased at nearly all ages, the m:f HRs estimated in the NHIS-LMF from survival models that control for male and female respondents' baseline ages at time of survey match closely the age patterns of the hazard ratios estimated in the NVSS population data. Specifically, the m:f HRs in the NHIS-LMF are estimated to grow significantly weaker with increasing age, and the overall bias in the age-specific HRs is much less than the respective bias in the HRs estimated from the conventional survival model that doesn't adjust for baseline age.

As a sensitivity test of our approach, we performed a similar exercise using a different U.S. survey-based data source, the National Health and Nutrition Examination Survey (NHANES) linked mortality files, 1988-2006. In the left-hand panel of Figure 6 below, we compare m:f HRs in the NHANES-LMF data estimated from conventional survival models (dashed gray line) with the m:f HRs in the U.S. official mortality data, 1986-2006 (solid black line). The contrasting age patterns of the HRs reveal that m:f differences in mortality risk in the NHANES-LMF data are biased in ways similar to the biases seen in the NHIS-LMF data. Estimates from survival models fitted using conventional approaches show m:f HRs that are relatively constant across age, showing no attenuation.

Figure 6. NNANES Male:Female Mortality Hazard Ratio by Age, 1988-2006, vs. NVSS Male:Female Mortality Hazard Ratio 1986-2006.
Left Panel: No control for NHANES respondents' Baseline Age at time of survey
Right Panel: Center Baseline Age at 50yrs (dashed), Center Baseline age at 45 if attained age 40-50; 50 if attained age 50-60; 55 if attained age 60-70; 60 if attained age 70-80; 65 if attained age 80+



When we refitted models to control for NHANES respondents' baseline ages at time of survey – and account for gender differences in mortality variation by age-at-survey – the m:f HRs in the NHANES data were estimated to grow significantly weaker with increasing age. The right-hand panel of Figure 6 plots the estimated age-specific m:f HRs from these models, revealing estimated age patterns of m:f HRs in the NHANES-LMF data that more closely approximate the age patterns found in the actual population.

These exercises clearly indicate that survey-based data are biased sources of mortality differentials in the U.S. adult population. Due to gender differences in living arrangements, sex differences in survival, and/or other possible sex- and gender-based differences in health-related factors, U.S. men's and women's likelihood of being sampled into non-institutionalized survey-based data sources appear to be very different from one another. As demonstrated above, these differences in survey selection result in biased estimates of age patterns of m:f HRs. As further demonstrated, the approach we used in our paper to account for selection into survey data was shown to provide good estimates of the true age-specific m:f HRs observed in the U.S. population. Thus, the adjustment strategy we used to account for differential selection into the NHIS-LMF by obesity status was shown to be quite effective at accounting for differential selection into the NHIS-LMF by gender. These adjustments were also shown to be effective at estimating age patterns of gender-based mortality differences in the NHANES-LMF data as well. Conversely, models fitted to these data that did not include this adjustment were shown to estimate m:f HRs that were badly biased at nearly all ages. Therefore, the evidence from these exercises answers Professor Hanley's question, "Is the cure worse than the bias?" with a clear "No". In both the NHANES-LMF and NHIS-LMF data, m:f HRs estimated from our modeling approach are much less biased than HRs estimated from conventional approaches. This is likely to be the case with obesity HRs in the NHIS-LMF data as well.

Finally, Professor Hanley correctly notes a limitation of our original model, namely that the adjustment for baseline age-at-survey is centered on mean age-at-survey across the full range of ages. By centering all baseline ages on this single value, "the 'adjusted' pattern poses a new question: at *older* ages HRs are raised considerably; but at *younger* ages they are now below 1." This is a fair point, and one raised before (Wang 2014, 2014; Wang and Liu 2014) and discussed before (Masters, Powers, and Link 2014; Masters et al. 2014). We are happy to address the concern here as well, especially as it pertains to the PAF estimates.

Although we observed only minor differences between the estimated m:f HRs from models that centered baseline age-at-survey on 50 years and those estimated from models that centered baseline age-at-survey on plausible ages (see right-hand panels of Figures 5 and 6), the models that center on the single mean age are likely too simple in their attempts to adjust for mortality variation in respondents' baseline ages. As a result, age-specific estimates are set to be conditional upon being surveyed only at age 50, thereby extrapolating the estimates beyond possible ranges of age in the data. We believe it is to this point that Professor Hanley correctly notes that models be "as simple as possible but not simpler".

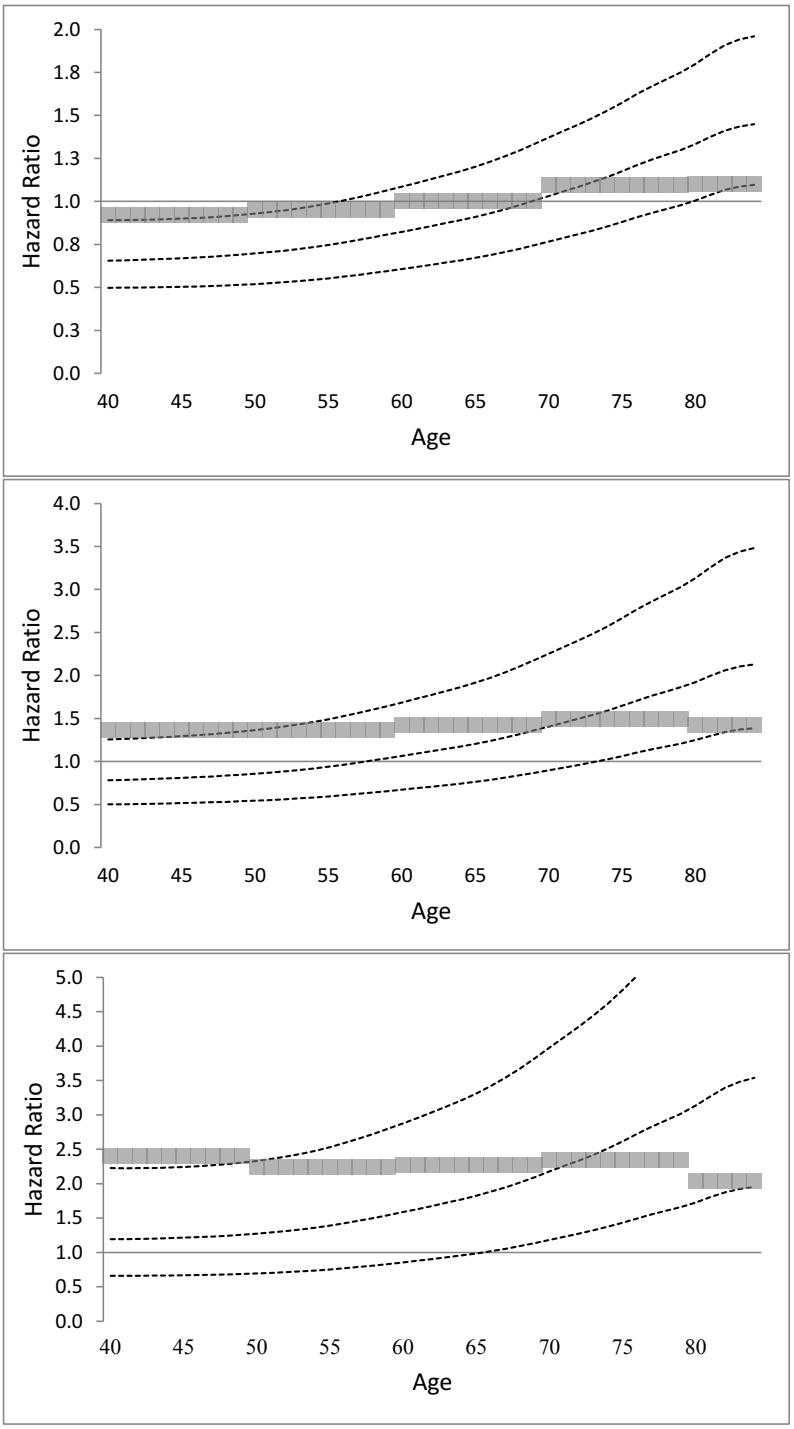
We have addressed this point more fully in a previous exchange with AJPH, but will provide a summary of our approach here. We first estimated age-specific HRs for overweight, grade 1 obese, and grade 2/3 obese by refitting survival models that allow baseline age to vary. To simplify the models, we used 10-year age groups to model age-based variation in the obesity-mortality association: [40-50), [50-60), [60-70), [70-80), and one five-year age group [80-85). We then held constant the baseline age *within* these grouped ages, but allowed the baseline age for each group to differ from one another. Thus, the models fit age-variation in the BMI-mortality association in the following way:

for attained ages 40-50, hold constant baseline age at 30;
for attained ages 50-60, hold constant baseline age at 35;
for attained ages 60-70, hold constant baseline age at 40;
for attained ages 70-80, hold constant baseline age at 55; and
for attained ages 80-85, hold constant baseline age at 60.

We therefore attended to Wang's (2014, 2014), Wang and Liu's (2014), and Professor Hanley's concerns by (1) allowing baseline age to vary by thirty years (30 to 60) and (2) the grouped age-specific HRs are estimated on plausible baseline ages within the NHIS-LMF data. That is, the model estimates are not severely extrapolated beyond possible baseline ages, therefore estimating plausible age-specific HRs in these data. At the same time, the modeling approach holds constant baseline age within the grouped ages to estimate overweight, grade 1 obese, and grade 2/3 obese HRs, thereby still controlling for the fact that age-specific estimates of high BMI HRs are conditional on NHIS respondents' ages at time of survey.

To show an example of the changes in model estimates, we plot the grouped age-specific HRs and the HRs from the original RP models for non-Hispanic white male sample in Figure 7 below. The gray bars indicate grouped age-specific HRs from the new models, and the dashed black lines are the HRs from the original RP survival models holding baseline age constant at -1 standard deviation (SD), the mean, and +1 SD.

Figure 7. Dashed black lines are estimated age-specific HRs from the original RP survival models, holding baseline age = 33 (-1SD), 49 (mean), and 63 (+1 SD). Gray bars are estimated 10-year age-specific HRs from survival models with baseline age held constant at 30, 35, 40, 55, and 60.



At young ages, the estimates of grouped age-specific HRs reflect the survival of those respondents who entered the sample at younger ages (baseline ages=30, 35, 40) whereas at older ages the estimates reflect those respondents who entered at relatively older ages (baseline ages=55 and 60). This approach, therefore, accommodates the concerns that the age-specific mortality estimates need to reflect reasonable baseline ages, but also partly controls variation in baseline age. Thus, we're no longer estimating age-specific HRs conditional on baseline age of 50 years at time of survey, but rather estimating age-specific HRs conditional on reasonable baseline ages for different attained ages. Overall, the estimated age-specific HRs for these 10-year age groups move consistently within the "normal" range of the HRs estimated from the RP models fitted in our paper. That is, at younger ages, the 10-year age-specific HRs are close with the age-specific HRs from the models fitted at -1 SD baseline age (33 years). The HRs then move closer to the RP estimated HRs for mean baseline across mid-adulthood, and then at older ages are pulled downward to reflect older ages at baseline (e.g., 55 and 60 vs. the 63 +1 SD estimates). As a result, as opposed to the estimated age-specific HRs from our original RP model, which estimated low HRs at young ages and estimated high HRs at older ages, these grouped age-specific HRs move consistently with the estimated HRs for appropriate baseline ages.

We refitted these survival models for the non-Hispanic black and white male and female NHIS-LMF samples and used the respective HRs to recalculate the PAFs. Estimates of non-Hispanic black and white men's and women's PAFs for overweight, grade 1 obesity, and grade 2/3 obesity as causes of US death are displayed in Table 1 Modified below.

Table 1 Modified. Estimated % of U.S. Deaths between Ages 40.0 and 84.9 for Birth Cohorts 1900 to 1960 Associated with Overweight, Grade 1 Obesity, and Grade 2/3 Obesity, NHIS-LMF 1986-2006.

	Overweight		Grade 1 Obesity		Grade 2/3 Obesity		Total
Black Women	-2.6	(-11.2,4.9)	8.7	(-1.5,14.3)	15.3	(6.3,19.4)	21.4
White Women	.6	(-3.4,4.7)	11.0	(8.1,14.0)	7.7	(7.0,8.4)	19.3
Black Men	.1	(-7.8,8.3)	5.0	(.6,10.1)	4.8	(2.0,8.6)	9.9
White Men	2.4	(-1.6,6.5)	7.1	(4.9,9.4)	5.2	(3.9,6.6)	14.7

Abbreviations: NHIS-LMF, National Health Interview Survey Linked Mortality Files.
Numbers in parentheses indicate 95% confidence intervals.

Together, these estimates suggest that 16.8% (95% CI: 7.1-26.0) of US deaths to black and white men and women aged 40-84 between 1986 and 2006 were associated with high BMI in these populations. This overall estimate, which attends to the concerns raised by Wang (2014, 2014), Wang and Liu (2014), and Professor Hanley is neither significantly nor substantively different from our original estimate of 18.2% (95% CI: 9.8-27.1). Thus, using an approach that is "simple but not simpler" than it ought to be, we find that high BMI imposes a considerable mortality burden on the US adult population.

Conversely, if we estimate age-specific obesity HRs in the NHIS-LMF using conventional survival models (i.e., that do not attempt to adjust for differential selection into the NHIS-LMF), we would estimate a PAF that suggests that high BMI levels in the U.S. adult population *reduced* mortality by 9.6% between 1986 and 2006. That is, the estimated PAF is -9.6%. This estimate is implausible not only because of the high prevalence of obesity and its known health

consequences, but also because it's derived from a modeling strategy that was shown to estimate severely biased age-specific m:f HRs in the NHIS-LMF (left-hand panel Figure 5).

We conclude by thanking Professor Hanley for the helpful comments, cautions, and exercises included in his comment. Professor Hanley's pleas about model diagnostics are helpful and we agree with his sentiments in spirit and in practice. We reiterate the fact that we used graphs, drawings, and diagnostic comparisons with population mortality rates to guide our modeling strategy of the obesity-mortality association in the NHIS-LMF data. We also affirm that our adjustments did not "exchange one 'paradox' for another." Rather, as we've shown in our examination of m:f HRs above, our approach estimated age-specific HRs in the NHIS-LMF that matched closely the known parameter values for age patterns observed in the official NVSS data. In contrast, conventional survival models estimated m:f HRs that were severely biased. We therefore started with a paradox – no attenuation of m:f HRs when we know attenuation exists – and we advanced both theoretical and empirical reasons for suspecting gender-based differences in health selection into the NHIS. When we used our approach to adjust estimates for this differential selection we ended not with another paradox, but rather with age-specific m:f HRs that matched closely the HRs observed in the population. We therefore defend the use of this approach, albeit with the recognition of Professor Hanley's plea "that models be 'as simple as possible but not simpler.'" Centering baseline age on the mean age at time of survey indeed made the model "simpler" than it should be, yet centering NHIS respondents' baseline ages on plausible values resulted in comparable conclusions to those presented in our paper.

Thanks,

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