

REPLY TO SCHMID, SNYDER, AND GELMAN AND AUERBACH:

Correlates of the increase in white non-Hispanic midlife mortality in the 21st century

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We welcome the letters of Schmid (1), Snyder (2), and Gelman and Auerbach (3), each of which usefully extends our original analysis (4), and points directions for further exploration. Three main findings of our study are useful to recap: first, contrary to previous trends in the United States, and contrary to current mortality trends in other industrialized countries, there has been an increase in all-cause mortality among white non-Hispanics (WNHs) aged 45-54 in the United States since 1999; second, the increase in mortality is largely among those with a high-school education or less; and third, the most rapidly rising causes of death are accidental and intent-undetermined poisonings, suicides, and alcohol-related diseases. There are also three important topics that we did not address, at least adequately: (i) We present a mortality breakdown by broad census regions, and we note that the deterioration in self-assessed health we report is observed in each state when analyzed separately, but we did not undertake further geographical analysis. (ii) Apart from noting parenthetically that the patterns of increase in these causes of death were similar for men and women, we did not focus on gender differences more broadly; in retrospect, the similarity between the sexes is itself surprising, and should have been investigated further and indeed, many press reports of our work incorrectly assumed that our results applied only to men. (iii) As noted in the Commentary by Meara and Skinner (5) that accompanied our article (4), we did not emphasize that the causes of death on which we focused are not, in and of themselves, sufficient to account for the increase in all-cause mortality in midlife WNHs.

Snyder (2) takes up the first topic (i) we did not address and shows the need to disaggregate all-cause mortality simultaneously by urbanization and by education. That the increase in all-cause mortality falls steadily as we move to more urban settings is consistent with previous work that focused on the increase in drug overdoses in rural areas (6, 7). If we look only at the deaths from poisonings, suicides, and alcoholic liver disease and cirrhosis, the increases in mortality

for WNHs aged 45-54 from 1999-2014 are 31.9 and 34.6 for large-central and fringe metro areas, 43.6 for medium metro, and 47.1, 46.2, and 48.6 for small metro, micropolitan nonmetro, and noncore nonmetro areas. Aside from the large fringe metro, there was almost complete convergence between urban and rural areas in death rates from suicide, drugs, and alcohol over this time period. The levels of mortality are not monotonic in urbanization; in 2014 they were highest in small metro, and lowest in large fringe metro. Snyder (2) also notes, in accord with point (iii), that changes in mortality from cancer and heart disease are also more favorable in urban areas. As a result, all-cause mortality in the most heavily urbanized areas fell for WNHs aged 45-54 over the study period. Even so, the decline of 7.6 per 100,000 is much smaller than those seen in other countries (see figure 1 in ref. 4), or even in the United States in the 1990s, so even those midlife WNHs living in the most urbanized areas are not fully sharing the international decline in mortality.

Schmid (1) takes up this point (iii), and notes, as in our report (4), that the increase in all-cause mortality cannot be accounted for by increasing mortality from external causes, so that other causes must be involved. One of these causes, as argued in Gelman and Auerbach (3), is an increase in the average age of the age group 45–54 as the baby boom generation passes through those ages. Other causes have been extensively investigated in ref. 8, which examines the slower growth in life expectancy in the United States compared with other countries in the past 25 y. Much of this relative slowing can be attributed to women aged 50 and over, where smoking behavior has been a key factor. American women started smoking much later than did men, and quit much later, so that although lung cancer mortality among age 45-54 WNHs has been falling for men and women taken together (see figure 2 in ref. 4), it continued to rise for women through 2009 but fall for men throughout. For the age group, two important drivers of all-cause mortality, other than external causes, are the passing

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through the age band of the baby boom generation—so that the group is aging, by about 0.4 y—and the passing through of the age band of a still-increasing cohort of female smokers or ex-smokers. Of course, these two factors are not likely to be exhaustive and, as emphasized in ref. 8, a full accounting is a major task, and would include factors such as stroke, diabetes, and mental disorders.

Although we accept the importance of the age and sex factors emphasized by Gelman and Auerbach (3), we dispute the claim that there is an "aggregation bias" in our analysis or that the increase in mortality that is headlined in our study does not exist. The mortality increases for WNHs aged 45–54 that we show come directly from the vital registration system, and are as correct as those counts. There is no bias here; we simply reproduce the CDC's counts. Gelman and Auerbach's (3) statement "Contrary to Case and Deaton's figure, we find that there is not a steady increase in mortality rates for this age group" is false. Figure 1 in ref. 4 is about mortality rates, and is correct as presented; it claims nothing about age-adjusted mortality rates. Adjusted and unadjusted mortality rates are different things, and serve different purposes. Our choice was neither erroneous nor accidental.

One way of summarizing our study is to say that for WNHs aged 45–54, the increases in mortality from poisonings, suicides, and alcohol-related diseases have been large enough to increase all-cause mortality for the group as a whole, given the existence of other background factors that have been slowing mortality decline, of which age and smoking among women are two of the

most important. One might reasonably argue that all-cause mortality plays a minor role in our study, which is fundamentally about the rise in poisonings, suicide, and alcohol-related diseases. However, that would miss the importance of flagging these deaths by their extremely unusual and salient effect on all-cause mortality for the group as a whole.

There is more than semantics involved here. The practice of mechanical age-adjustment, although common, is dangerous in the presence of other causal factors that are related to age. As we have argued above, smoking among women is such a factor, so that to age-adjust without taking smoking into account risks confounding the effect of smoking with age and with sex. When we try to tease out the effects of multiple variables on mortality, we must treat them symmetrically and not privilege one, however important its influence. Additionally, without further analysis, it is impossible to tell how much of the effects that Gelman and Auerbach (3) attribute to age and sex are in fact attributable to smoking, or to something else.

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