Session 2B Discussant Comments

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Gaille and Sherris

Cause of Death Mortality: What Do We Know of Their Dependence?

Estimates of the rise in life expectancy with the hypothetical elimination of various diseases have been a part of standard actuarial/demographic analysis for decades. The premise behind cause-deleted life tables is straightforward—people hypothetically saved from dying from one disease are placed back into the mortality risk pool under the assumption they will be exposed to the unaltered risk of death from all other causes. Anyone versed in the use of these tables knows that the underlying assumption of disease independence is unrealistic, in exactly the same way we all know the underlying assumption behind the calculation of period life expectancy, that age-specific mortality risk will not change, is equally untenable. We recognize the limitations of the calculations, perform them anyway, and accept the results for what they are worth—approximations of mathematical truth about a phenomenon, mortality, we should all know by now is driven by a combination of biology, environment and personal choice.

Estimating the magnitude and direction of dependence between diseases has also been discussed for some time. One hypothesis suggests that because diseases are related **to each other**, cause-elimination life tables **overestimate** gains in life expectancy. This would occur because dependence, by definition, means that competing causes exist within bodies at the same time, so those hypothetically saved from dying are not moving back into the risk pool unaltered—they are in fact placed back into the risk pool **with** other competing risks in their bodies.

The opposite hypothesis about the effect of dependence involves the reason death rates decline. If death rates are declining due to improved risk factors, and fatal diseases are dependent, then cause-deleted life tables **underestimate** the gains in life expectancy because risk-factor modification would have an amplified effect by favorably influencing multiple diseases simultaneously.

So which is it? Do cause-elimination life tables underestimate or overestimate gains in life expectancy, or is it even possible that both risks are present at the same time—yielding a reasonably good approximation of what would happen if a disease were cured. One solution is to abandon the use of the **elimination** component of the calculation since medical technology has only eliminated one disease in human history—smallpox. It seems unrealistic to expect this will ever happen, although it is worth noting that **multiple diseases** would have to be eliminated, or the equivalent achieved, to yield a life expectancy of 100 or more as some predict. The solution is partial cause elimination—that is, what happens to life expectancy if, say, 25 percent of deaths from a given cause are effectively eliminated?

Another alternative is to find a mathematical model that estimates the dependence between diseases —this is where the paper by Gaille and Sherris comes in. Using a vector error correction model (VECM) developed in the field of econometrics to assess relationships binding economic variables, they applied this approach to causes of death in various countries across time. I particularly appreciate interdisciplinary approaches just like this to understand human mortality, and this model fits the bill. The elegance and novelty of the VECM is its primary appeal, and I look forward to the publication of this paper. This kind of work is important in the field. However, for reasons I'll explain in a moment, it also exemplifies exactly what can go wrong when pure mathematical models are applied to biological events such as human mortality and longevity. Another example of this problem is described in detail in a paper I published last year, "Zeno's Paradox of Immortality," where I demonstrate that purely mathematical models which predict large increases in longevity and make these predictions while ignoring human biology, very quickly lead to untenable conclusions.

Gaille and Sherris demonstrate convincingly that their model of dependence yields dependence between diseases, which of course should not be surprising. What should have caught your attention was that the dependence did not operate in the same way in different countries—leading the authors to conclude that cause-elimination models should probably not be used to extrapolate across national populations. Once the model is further refined to address age and cause-specific death rates, we'll be able to say much more about its utility.

Here's the problem. Conventional wisdom suggests that diseases are dependent, not independent, **and that they are dependent or related to each other**. We've known independence is not reasonable, and we've known it for decades, and this paper further confirms this belief. While this is

most certainly true at one level, the correlation or dependence exists not because they are biologically dependent on each other, but because they are all together influenced by a common risk factor—the biological aging of the body. This looks to me like the logical fallacy of causation in epidemiology. By way of example, people who wear XXX size clothes tend to die from cardiovascular diseases at younger ages; some might conclude that death is dependent on the choice of clothing size. People who wear XXX size clothes do so because they are obese, and it is the obesity causing the higher mortality, not the clothes. In this case, the dependency is not so much between the various lethal diseases, it's that they all have a common risk factor, which is neither discussed nor measured in this paper. I would strongly encourage the authors to spend time trying to understand the underlying biology of aging and it's linkage to the expression of chronic fatal conditions and include that discussion in any future papers on this topic. While it is not yet possible to measure biological aging with any degree of accuracy, that day may be forthcoming, and when it happens, models like this might prove to be particularly useful for developing a more accurate and thorough understanding of exactly how and why disease dependence occurs.

Sam Gutterman

Obesity and Mortality

Sam Gutterman set out to answer a question in his own mind about the relationship between obesity and mortality, and quickly discovered it's not only extremely complicated, it also requires an understanding of a broad literature involving measurement, reporting, reliability, biology, biochemistry, etc. So, Gutterman did what he usually does when faced with a task like this—he wrote the definitive article after reading everything, and I mean everything, about obesity. I can't really provide a commentary on the book he wrote, although I would encourage all of you to keep a copy of Gutterman's paper on your desktop as a reliable reference when discussing obesity and mortality. What I will do is address the fundamental linkages and the future—which is really where I think our attention should be focused.

On the linkage between obesity and mortality, it is a tradition to point to the decadence and decay of our modern world as the primary reason behind the rise of obesity. Although true at some level to be sure, the problem may not boil down to a simple calories-in-and-calories-burned equation.

Gutterman alludes to this briefly, but there is emerging evidence to suggest that fundamental changes in the microbiota of the human gut may have contributed to this, and this would explain, in part, the extreme resistance we face in trying to lose weight. This is why diets may not work well. Efforts to repopulate the gut with microbes that were present before the introduction of certain antibiotics in the 1970s offer a unique opportunity to attack the problem in a different way, but only time will tell whether this intervention works.

I remember back in 2005 when my colleagues and I suggested that the negative health and longevity effects of the obesity epidemic will only get worse because of latent cohort effects. The response from some well-respected demographers was that the problem can be easily fixed—all we have to do is eat 100 fewer calories a day and the problem will disappear. What happened since 2005? The obesity epidemic accelerated even more rapidly than we anticipated eight years ago, and the negative effects of health and mortality at middle ages expressed itself a few years earlier than the 10-year latent effect we projected. While eating 100 fewer calories per day would work in theory, in the real world it became much easier to consume an extra 100 calories per day.

Some in the media have noted that things seem to be improving because the rate of increase in obesity has decelerated, in some cases it's leveled off, and in some places and for some subgroups there may be a decline in incidence. I've found some amusement at this conclusion, for what I really think is happening is that we're approaching a saturation point. After all, 100 percent of the population is unlikely to become obese, so at some point the rise has to decelerate. A similar problem will soon occur with the crude death rate, which will inevitably rise due to population aging—at the same time life expectancy is also rising. The media will no doubt greet this event with some alarm, until they are told that a rising crude death rate is linked more to a shifting age structure than to worsening health conditions.

Where are we headed with regard to obesity and mortality? Gutterman addresses this briefly and directly at the end of his paper, but the place where he really gets to the point is when he reviews the literature on cohort effects. Current linkages between obesity and mortality are calculated using data on people who acquired their obesity in adulthood, but in the future, the cohorts exhibiting obesity-related mortality, disability and frailty will have acquired their obesity in childhood—decades earlier than current middle and older age generations. Evidence has already emerged that these younger generations are in trouble—some have even been documented to have the cardiovascular system of

middle-aged men—at the age of 10. They are certainly not in the same category as obese adults, and mortality extrapolation models that fail to consider the health of the living and the importance of cohort effects are naive at best.

Sam Gutterman

Smoking and Gender: Population Mortality Trends

One of the fundamental public health interventions in the 20th century was the discovery that smoking is harmful and leads to premature death. Demonstrating this relationship was challenging at a time when smoking was popular, and many simply didn't want to believe that most smokers would eventually pay a price for their habit. The central problem was the lagged effect. For someone who begins smoking at age 15, for example, the negative health consequences for most won't be realized for decades, and some will die from causes unrelated to their habit. Gutterman's paper is devoted to describing the historical literature designed to estimate the effect of smoking on mortality, with particular attention paid to sex differences in exposure and mortality.

I don't see a need to summarize the paper—Gutterman's done that well enough. What I want to do is place the problem into perspective, especially given the first two papers I've already discussed on dependence and the obesity/mortality linkage. In fact, I'm going to make the case that virtually the exact same issues which arose with the previous two subjects are front and center when dealing with linkages between smoking and mortality, and in particular, the use of this relationship to generate forecasts of life expectancy—exactly what some researchers have done.

Gutterman indicated appropriately at the end of his paper that linkages between smoking and mortality are fraught with uncertainties. Included among them are changing dynamics of contributions to mortality, data quality, reference population issues, confounding, secular changes in smoking pattern, and the long lag time between exposure to smoking and death, among others. This doesn't invalidate the linkage—it just means the results should be used with great caution. The primary use of these data, in my view, is to encourage people to avoid smoking if they haven't started, and quit smoking if they have. That's it! Decompositions are fine if used for informative purposes, but serious problems emerge when these linkages are used to generate forecasts of total mortality and life expectancy for national populations. Again, the problem is not with exploring the

negative effect of smoking on life expectancy, or with generating smoker and nonsmoker life tables; it's with efforts to precisely determine how secular trends in smoking will influence **future mortality**. A classic example of the problem appears in table 11 of Gutterman's paper, which shows a Lee-Carter linear extrapolation of survival projected out to 2034 with and without smoking. The issue here is identical to the one I raised earlier about dependency.

All smokers and nonsmokers eventually die; one group on average dies sooner than the other. That much we know with certainty. However, a death averted from one cause must eventually lead to a death from another cause, so the concept of "deaths averted" by avoiding smoking, or by avoiding any other behavioral risk factor, needs to be accompanied by caveats. The Lee-Carter type linear extrapolators work only with mortality statistics—the numerators and denominators required to generate death rates; they completely ignore the most important critical confounders. This is also central to the issue of dependency, biological aging and the health status of living populations. Aging is the elephant in the room, and I simply don't understand how it can be ignored given decades of research now culminating in the conclusion that aging is the primary risk factor for most of what goes wrong with our bodies with time. The longer we live, the greater the aging risk factor becomes and the less we get from risk-factor modification. This is exactly why linear extrapolation, which turns a blind eye to our biology, overestimates future mortality reductions and life expectancy increases. The fact that biological aging cannot as yet be precisely measured at the individual level does not mean it should be ignored. That would be like saying air does not exist because we can't see it with the naked eye.

Gutterman's second paper is once again a thoroughly researched manuscript that should serve as a reference paper for anyone interested in the linkages between smoking and mortality. The actuarial profession is lucky to have someone like Gutterman in their discipline. In fact, all three papers in this session are extremely well written. All should be published, and we should all be reminded that while our actions during life lead to statistically measurable outcomes, there are fundamental biological processes which envelop all we see and measure.