Informal Discussion Transcript
Session 2B–Behavior and Causes of Death: Impact on Mortality and Mortality Modeling

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FROM THE FLOOR: What I have to say is quite positive. These are tremendously good papers. I would like to point out some very broad issues, such as competing risks of death and the fact that your body never forgets. If you currently smoke, quit. But your body still remembers. There are no conditions that are ever cured. They're always in your body, even if they're clinically not measurable. So these are two common problems that we're facing today that will have long-term effects. Regarding Sam Gutterman's paper in particular, Sam mentioned several paradoxes. Two that you triggered in my mind that you didn't mention include the fact that Mexicans in poverty have had bad nutrition. They're the ones that come to the United States and live the longest! How do we explain that? The other paradox involves the overlap between obesity and smoking. Aren't many of these individuals the same people? So if you compound or add their effects on mortality, are you overestimating the effect of each of these conditions? Of course, the big problem in all of our work is, if you don't take a position of mathematical determinism, such as the view Jim Vaupel expressed this morning, then you have to come face-to-face with making projections for real life situations that may occur, the results of the joint effect of which is unknown. So all of these projections have very broad ranges of error.

SAM GUTTERMAN: The Mexican issue is a very difficult one,

particularly because the immigrants from Mexico tend to be much younger than the overall population and therefore have been exposed to obesity risks for a shorter period because of their relatively young ages—so, I think only time will tell. We have started to see adverse trends among Hispanic-Americans in terms of adverse obesity levels, especially in children, which may have consequential adverse mortality as the years go on.

WARD KINGKADE: This is a question for Jay Olshansky. Isn't it possible that what you're calling biological aging is time-dependent?

JAY OLSHANSKY: All right, you mean age-dependent or ...

WARD KINGKADE: Time-dependent.

JAY OLSHANSKY: Maybe I don't understand the difference between age and time, but …

WARD KINGKADE: And to say that there is biological aging sort of postulates that human beings are going to have this biological aging over time, over any period of observation you're talking about.

JAY OLSHANSKY: Yes, of course. Cumulative damage to the mass of building blocks of life. You push out the envelope of survival. You push people out into older and older ages and aging itself or senescence itself becomes an increasingly more important risk factor for the things that go wrong with us. Which is why in practice you would get less and less from behavioral risk factor modification the longer we live. That's why you're only seeing,

hypothetically, a relatively small gain in life expectancy with 100 percent elimination of cancer or 25 percent elimination of cancer in the population. The gains in longevity are very small.

WARD KINGKADE: So you would agree that this mass of building blocks of life is dynamic and how it responds to different things could change over time? And that this might affect the conclusion of various studies? Seems like something that's in principle hard to measure.

JAY OLSHANSKY: Senescence in principle, it's not yet measurable. We can't measure aging yet. We have Len Hayflick in the back of the room, I think, one of the individuals who is capable of addressing this issue possibly better than anyone else.