Commentary

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A general theory of population aging sounds attractive and even necessary, but we must ensure that we are moving toward it rather than moving further away. The construct embraced by Robine and Michel (1) envisions a transition model between four stages: 1) improved survival of sick persons, 2) control of chronic diseases, 3) compression of morbidity, and 4) an epidemic of frailty. These authors note that the literature that they review contains inconsistencies between data sets as well as differences in interpretation of the same data. Their plea for more consistent, better quality, more standard, and more plentiful data is of course useful. Much currently available data is of dubious validity, and a coherent synthesis of uncertain data is a daunting task.

I do have fundamental issues with the endeavor itself, and with the proposed construct. Populations do not age in the most literal sense—people do. Population data, particularly when the population is equated with a political state, represent summary averages of diverse individuals and subgroups, and interpretation of population data in the context of individual variability is difficult. A general theory of biological aging, I would think, needs to precede a general theory of population aging, which could then be derived from it, and the inconsistencies and contradictions explained. Strehler and Mildvan (2) attempted such a synthesis in 1960 with considerable success, and their insights continue to represent a standard for understanding declines, variability, and the biologic inevitability of frailty. Any proposed general theory, I would think, must, at a minimum, have more explanatory power than the Strehler-Mildvan hypothesis, which has a very different conception of frailty than that suggested by Robine and Michel.

A problem with transition or "stage" models, demonstrated in the discussions of Robine and Michel, is that they imply progression from the first toward the higher numbered stages by a series of transitions followed by new stable states. These transitions, however, actually are very gradual, vary from one population to another, are not in synchrony, are not linear, and vary with different subgroups within the population and with individuals within the subgroups. Moreover, the movement can be backward as well as forward. Mortality rates can and do go up and down, and disability (morbidity) can increase as well as decrease. I believe that it is preferable to conceptualize and quantify positive or negative changes in continuums of mortality and morbidity rather than to attempt to fit data into more artificial "stages."

I am gratified, of course, that the compression of morbidity (3) is now regarded as an accepted paradigm and even given a whole "stage" in the proposed general theory; it was not always so. The evidence that morbidity compression can occur has become overwhelming over recent years as longitudinal data on morbidity and mortality became available. In the United States, disability has declined at a rate of 2% to 2.6% per year from 1982 to 1999, while mortality rates declined at a rate of about 1% per year; disability rates declining more rapidly than mortality rates is the formal requirement for compression of morbidity. However, compression of morbidity is not inevitable; its achievement requires substantial postponement of disability (greater than concurrent increases in longevity), which in turn requires preventive efforts by individuals, organizations, and governments (4).

My own "general theory of aging" considers two major elements, mortality and morbidity, which have a relationship with each other because most perturbations that reduce one will reduce the other, and most that increase the one will increase the other, although often not to the same degree.

If morbidity is postponed more than mortality, then there is compression of morbidity, and this is desirable. If mortality and morbidity curves grow apart, there is expansion of morbidity, and this is not desirable. There are some perturbations that increase morbidity, some that do not affect it much, and some that decrease it; the same is true of mortality.

The relative effect of a particular input on morbidity and mortality is not always obvious. Decreases in a nonfatal diseases, such as osteoarthritis, postpone morbidity and do not affect mortality. Decreases in sometimes fatal diseases, such as coronary artery disease, depend upon the distribution of outcomes between sudden death (increases that reduce morbidity and increase mortality) and chronic congestive heart failure and its complications (increases that increase morbidity and decrease mortality). In a global pandemic of depression predicted by some, morbidity would increase but mortality would change little.

In a society where everyone smoked cigarettes, morbidity would be increased from present levels; if no one smoked, it would be decreased (5). If a nation has a pandemic of obesity, disability will be increased. If everyone exercised regularly, there would be less disability. As is occurring in the United States, it is possible for cigarette smoking to go down and obesity to go up at the same time, with uncertain effects on health (6). It is possible for smoking to go up in some subpopulations and down in others. An individual may both smoke and exercise. Mortality rates can change differently in different ethnic subgroups. This is a complex business. Population effects are ultimately the result of the balance between the forces and events that expand morbidity integrated across individuals and those that compress it.

Perturbations to the individual health state may be classified quantitatively as increasing or decreasing morbidity and as increasing or decreasing mortality. The individual is subject to many perturbations, and it is usual for some to have positive and some to have negative effects. Population morbidity and population mortality are the integrated sums of the positive or negative effects of perturbations on individuals.

The ability to compress morbidity by healthy lifestyles has been well shown in longitudinal studies of aging individuals, and the effects are large. Nonsmoking, lean body mass, and exercise in seniors postpones disability by 5 to 8 years (6). Regular vigorous exercise is associated with postponement of disability of 8 to 12 years (7). Should we want a society with expansion of morbidity, we would start with obese, cigarette-smoking, and nonexercising children and end with heavy use of life-extending technology.

Decreases in mortality rates continue at all ages in the low-mortality countries. Yet, changes in life expectancy from higher ages are much more modest than at birth. From 1980 to 1998 in the United States, for example, life expectancy from birth increased by 0.15 of a year per year. The more relevant number, however, is life expectancy from age 65, because this excludes early-life mortality. From age 65, life expectancy increased only 0.066 of a year per year, less than half as rapidly. Assuming that mortality rate declines continue unabated at their historical rates from birth and from age 65, the United States curves of average age at death would intersect at the "point of paradox" at the average age of 87.8 years in the year 2076 (8). Passing this intersection is not possible, since at the intersection point there would be no deaths at all below age 65, and you cannot have fewer than zero deaths. Thus, present mortality trends cannot continue indefinitely although they can continue for quite a long time. "Compression of mortality" is not essential for "compression of morbidity," but slowing rates of increase in life expectancy in mortality would tend to accentuate the compression phenomenon.

Frailty has proven an elusive term, but it is strange to me to see it invoked as a fourth demographic stage resulting in an expansion of morbidity. I do not see any compelling evidence that this stage could occur even theoretically, and a number of reasons to believe that it could not. This stage seemingly envisions a society of increasingly decrepit, extremely old people who just will not die, and it apparently derives from a belief that frailty is not a risk factor for death. Decreases in organ reserve in multiple organs mean that an exponentially ever-smaller perturbation may result in death (2). Medically, we use the term "frailty" to refer to persons who have become very fragile without a dominant chronic illness, and have found frailty to be a strong predictor of mortality. Frailty is the ultimate competing risk for mortality, that of "old age."

I am not impressed by anecdotal or outlier arguments to the contrary. Nor have I seen any surge in supercentenarians beyond that to be expected from larger birth cohorts who have increasingly lived longer lives; as the area of a biologic distribution increases, the tails extend and the absolute numbers of outliers increase. When I first began to write on these subjects, the oldest American had died at 113 years and was the second oldest worldwide, with a surviving Japanese man older (9,10); at present, the oldest living man is said to be a Japanese man at 113 years, and the oldest living woman, a Japanese woman at 115 years, with two prominent outliers between, a French woman and a Japanese man, at 122 and 120 years, respectively (11). Anecdotes about extreme longevity have often proven to involve some form of age exaggeration, and with the possibility of fraud always present, even carefully authenticated isolated extremely rare cases are not compelling.

For the proposed fourth stage to occur, one would expect that centenarians would be more disabled than those younger at the age of death, that the oldest-old (over age 85) would be expanding their morbidity, that disability in the year prior to death would be greater in those with good health risks and longer survival, and that medical care costs, used as an index of disability and frailty, would be highest in those dying at very advanced ages.

To the contrary, centenarians in general exemplify the compression of morbidity rather than its expansion (12), the decrease in disability in the oldest-old exceeds the decrease in oldest-old mortality rates (8,13–16), disability in the year before death is lower in those with good health habits despite their greater longevity (17), and cumulative lifetime Medicare costs in the United States do not increase with increasing age at death (18). All of these observations

support the view that frailty compresses morbidity rather than extends it.

We all agree on the need for better data. This needs to continue to include the relatively weak endpoints of many past studies to allow trend analyses. We need serial studies with the same sampling techniques, and we need longitudinal studies of the same individuals over time. We need similar research designs in different populations, subpopulations, and subgroups. The endpoints, especially including disability, must be reliable, valid across different languages and cultures, and responsive to change. Disability is not a present or absent variable; it is a continuous variable and must be measured as such. These methodologies are available (6,7,19), and with better data we will better be able to identify and quantitate the determinants of successful aging.

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