A Realist View of Aging, Mortality, and Future Longevity

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THE MODERN ERA has seen a continuing, unparalleled rise in human longevity. A few countries now have average life expectancies at birth around 80 years.[1] Numerous other countries are rapidly approaching those levels. A recurring question, then, is how much higher that average can get. This is the subject of intense debate and wide popular interest, with potentially profound implications for health policy and public finance. As research on human longevity has proceeded, it might have been thought that views on the future course of life expectancy would converge to a consensus position. Thus far that has not happened: views remain far apart, expressed across a diverse spectrum of professional journals and scientific disciplines. In a simple three-way classification of the major positions that is used by the present authors (Olshansky and Carnes 2007; Olshansky 2007), there are those who believe that immortality is within reach (*Futurists*); those who believe life expectancy will rise to 100 years or more in this century (*Optimists*); and those who believe that life expectancy is unlikely to exceed an average of around 85 years in the absence of radical advances in the control of the aging process—and could even decline in developed countries in this century (*Realists*).[2]

In this note, we describe our own views on aging, mortality, and longevity, which fall in the "realist" category. In doing so we also identify and seek to correct several misconceptions about that position—and, more generally, about human longevity—that recur in the scientific literature: 1) that realists (or, as often described, "pessimists") believe old-age mortality is intractable and hence (for example) that efforts to prolong the lives of the elderly are likely to be futile; 2) that schedules of age-specific death rates from intrinsic causes of death are immutable; 3) that age patterns of senescence and lon-

gevity are biologically determined, dictated by genes that evolved for that purpose; 4) that there is a fixed upper limit to human longevity; and 5) that average life expectancy cannot exceed 85 years.

"Pessimism" and the intractability of old-age mortality

Every generation speculates on limits to human longevity, and yet no generation believes that those limits have been attained. Early in the twentieth century, the view emerged that there are biological limits to life (an echo of the biblical span of three score and ten years), and that humans were approaching such limits. In the United States, this presumption led the actuaries responsible for making official forecasts for the Social Security Administration to project a decelerating rise of life expectancy during the latter half of the twentieth century (Olshansky 1988). Other population scientists shared the belief in limits, leading them to predict that life expectancy at birth would never exceed approximately 85 years (Bourgeois-Pichat 1978; Fries 1980).

The danger in specifying a limit for human life expectancy is, of course, that it may soon be broken. History provides a number of illustrations. Thus far, at least, that is not true of the 85-year limit.[3] In our original calculations in support of that figure (88 years for women and 82 for men) for the United States and other developed countries, we demonstrated that in order to reach it, death rates would have to decline at every age (including extreme old age) and from every cause by about 55 percent from levels existing in 1985 (the year of the study). As a way to visualize the magnitude of such a mortality reduction, we showed that it would be equivalent to the elimination of both cancer and heart disease (Olshansky, Carnes, and Cassel 1990). Such a prediction hardly seems to warrant being characterized as pessimistic. Labels, of course, are by their nature oversimplifications but some of those applied to schools of thought on mortality and longevity have created and perpetuated misconceptions.

There is, we believe, a broad consensus in the scientific community that anticipated advances in medical technology and improvements in public health will continue to drive down death rates at all ages. The competing schools of thought do differ, however, on the anticipated pace and magnitude of those mortality trends (Olshansky and Carnes 1994), on the influence of biology on those trends (Carnes and Olshansky 1993; Carnes et al. 2006), and on the potential of recent trends in health attributes of the population (e.g., the rise in childhood obesity and the re-emergence of HIV/AIDS) to slow or even reverse the mortality declines (Olshansky et al. 2005).

A charge leveled against advocates of the realist position is that they believe that old-age mortality is resistant to intervention. Vaupel (1997a) says as much, and draws some serious corollaries:

> The belief that old-age mortality is intractable remains deeply held by many people. Because of its implications for social, health, and research policy, the belief is pernicious. Because the belief is so prevalent, forecasts of the growth of the elderly population are too low, expenditures on life-saving health-care for the elderly are too low, and expenditures for biomedical research on the deadly illnesses of old age are too low.

Elsewhere, in a prominent publication, Vaupel (1997b: 1799) explicitly associates us with that belief: "…the view that mortality at older ages is intractable leads to the conclusion that health-care resources and biomedical research should not be wasted on hopeless attempts to prolong the lives of the elderly (Olshansky et al. 1990; Lohman et al. 1992)."

The view of old-age mortality as intractable certainly does not follow from our work.[4] We have stated unambiguously that old-age mortality is inherently modifiable (see the following section), and that these modifications have already led to the characteristic mortality patterns observed in low-mortality populations—see Olshansky and Ault (1986) and Olshansky and Carnes (1994: 76). In the latter study, for example, we wrote:

> We are not arguing that diseases and disorders associated with senescence are necessarily immutable. Indeed, it is probable that the onset and progression of fatal diseases will be further postponed (even at older ages), and it may even be possible to do the same for disabling diseases and disorders that are currently immutable.

As to the implications, nowhere in the two studies cited by Vaupel is there a suggestion that health care resources and biomedical research are either wasted on the elderly or should be denied to them. What we and our colleagues have argued is that the primary goal of life extension research should be the preservation and extension of healthy years (Olshansky et al. 1991; Olshansky and Carnes 1994; Carnes, Olshansky, and Grahn 1996). In other words, we have consistently made improving quality of life for the elderly a high research priority.[5]

Our team's publications over the last 17 years provide a documented record of biodemographic research that has placed the future of human longevity within the context of biological realities and observable attributes of populations. Using established biological concepts, our research tells us that bodies are not "designed" to fail, nor to last indefinitely. This lack of design is exceedingly good news because it means that human-devised interventions can continue to extend life at every age. Eventually, these gains will be further augmented as ways are found to alter the genetic programs that inadvertently give rise to intrinsic diseases and disorders. This perspective on human longevity has led us to argue repeatedly for more research aimed at improving the health and quality of life for both current and future generations of elderly.

Intrinsic mortality schedules and longevity outcomes

Central to our biodemographic approach to the analysis and interpretation of mortality data is the concept of intrinsic mortality—the biological component of all-cause mortality.[6] We refer to the schedule of age-specific death rates for intrinsic causes of death as an "intrinsic mortality signature." Contrary to what some have attributed to the realist view (Tuljapurkar and Boe 1998; Carey 2003), these signatures are not immutable. Changes in a signature would occur "when the forces of selection acting to maintain the genetic composition of a population are disrupted (e.g., through environmental challenges) or methods of intervention become available to modify—either positively or negatively—the expression of intrinsic disease processes" (Carnes, Olshansky, and Grahn 1996: 236). As to those methods of intervention, we wrote:

> [M]odification of the signature's expression depends on the extent to which future technologies can: (1) continue to extend survival for individuals who would ordinarily die from genetic diseases before reaching sexual maturity; (2) further postpone mortality associated with intrinsic diseases; or (3) delay expression of and/or eliminate intrinsic diseases by manipulation of the genome (e.g., genetic engineering, controlling gene expression, providing replacement gene products). The recent identification of genes responsible for a variety of diseases and disorders will eventually lead to further modifications to the expression of the intrinsic mortality signature. (ibid.: 252)

Interventions in the expression of the intrinsic mortality signature can continue to yield gains in life expectancy—gains that we have called "manufactured survival time" (Carnes, Olshansky, and Grahn 1996; Olshansky, Carnes, and Grahn 1998). Already many humans may be living on such time, manifested for some as healthy years of life, for others as disabled years. An important health policy issue is whether an intervention influences the underlying etiology of intrinsic disease processes (e.g., delaying the age of onset) or only modifies the expression of intrinsic disease (i.e., delays the age at death).

The mutability of intrinsic mortality signatures became apparent to us in a study (Carnes, Olshansky, and Grahn 1996) comparing age-determined (intrinsic) mortality patterns across three taxonomically diverse species: mice, dogs, and humans. Normalizing the time scales (age at death) of each to the median age of intrinsic death, the age patterns of mortality (expressed as either a survivorship curve or a hazard function) for the mouse and dog were found to coincide. Contrary to our initial expectation, however, the curve for humans exhibited an accelerated failure rate relative to the other two species—a finding we believe is explained by the medical care the humans received. (Our animals were treated humanely but they received no interventions that would delay their deaths.) Medical care inflated the median age at

death for our human population, and, given the mathematics of our scaling, artificially shifted all the failure times to an earlier age.[7]

We believe that, taken together, our publications realistically portray the ability of human ingenuity to have a positive impact on the duration of life of individuals and the life expectancy of populations. We have, however, expressed concern that delaying age at death without also delaying age at onset will convert acute diseases into chronic disease and thereby extend the period of old-age impairment and have an adverse impact on quality of life (Olshansky et al. 1991). This concern over managing rather than curing disease during a period of population aging underlies our advocacy of more research both on interventions affecting the aging process and on the nonfatal diseases of aging that degrade quality of life (Carnes, Nakasato, and Olshansky 2005; Olshansky et al. 2006).

Mendelian predestination?

A common misconception about the realist position is the alleged assumption that longevity, and the mortality that defines it, is programmed within the genome of organisms.[8] Biological determinism has never been part of our biodemographic paradigm: it is totally incompatible with the established evolutionary theories of senescence upon which our work is based. According to evolutionary theory, senescence originates from biological factors that evolved for other reasons (Hamilton 1966; Kirkwood 1977; Medawar 1952; Williams 1957). Returning to first principles, given the inevitability of death in the hostile environments of Earth, both past and present, a defining characteristic of living matter is the ability to self-replicate or reproduce. There is a race between reproduction and death. Prey animals like mice that are under intense mortality pressure must achieve sexual maturity quickly in order to ensure their replacement in the next generation; in contrast, species like humans that experience much lower extrinsic mortality pressures give birth to altricial young that may take many years to achieve sexual maturity. Duration of life is calibrated to the time needed for maturation, reproduction, and nurturing.

From a biological perspective, the race between reproduction and death is so crucial that it cannot be left to chance. As a result, growth and development involve highly regulated genetic programs (Balinsky 1970). Since life is a race against time, there is no question that the tempo of these growth and development programs is governed by extremely precise and highly coordinated biological clocks. It is easy to see how such clocks could be misinterpreted as clocks for longevity. However, genes involved in this exquisitely complex process are designed for health and vigor, not sickness and decay. The duration of the process can be thought of as the biological equivalent of a warranty period where senescence is the unintended byproduct of bodies' surviving up to or beyond their warranty (Carnes, Olshansky, and Grahn 2003).

This line of reasoning suggests that senescence, far from being a manifestation of Mendelian predestination or evolutionary intent,[9] arises from evolutionary *neglect*. The absence of selection in the post-reproductive period of the life span means that there can be no genetic basis for either immortality or senescence produced by the direct action of natural selection. Rather, senescence-related diseases and disorders are an incidental accompaniment of a rare and largely irrelevant event in nature: survival into the post-reproductive period of the life span (Medawar 1952; Hamilton 1966).

Investing physiological capital into maintaining the soma (body) beyond the ages needed for reproduction, nurturing offspring, and contributing to the reproductive success of those (or other closely related) offspring would be an unnecessary and unwise investment of precious physiological resources (Kirkwood and Holliday 1979). Thus, from a biological perspective, aging arises as an *inadvertent byproduct* of fixed genetic programs and biological metronomes that regulate early-life developmental processes. Aging is an inescapable consequence of life's ingenious and necessary response to the inevitability of extrinsic causes of death (Carnes, Olshansky, and Grahn 2003). An assumption of biological determinism, in contrast, would require senescence to be the *intended* product of natural selection.

An upper limit to longevity?

Other than mythological and biblical references to limits to life (Gruman 1966), the most frequently cited reference to a fixed life span is Fries (1980: 130), who based his theorizing about a "compression of morbidity" on what he considered an evidence-based assumption that the "maximum life span [for humans] has not increased." More recent evidence, however, suggests that both the maximum life span and the median and maximum ages at death increase in response to cohort size as well as to modifications of the physical environment (Carey et al. 1992; Fukui, Liang, and Curtsinger 1993). These findings have led some to conclude that either there is no limit to length of life, or, if one exists, it must lie beyond the observed longevity horizon (Wilmoth 2000).[10]

The measure of maximum life span, of course, is like an Olympic record: it can only improve. Olympic records that were often broken by large margins in the past are now improved by only fractions of an inch or second—large improvements are exceedingly rare. Yet there would be near unanimity that all of today's Olympic records will eventually be broken. Similarly, there would be broad agreement that life span and life expectancy records will continue to be broken.[11] Where opinions on longevity limits differ is on the time-course and magnitude of those improvements over the long term and on the question of how relevant maximum life span records are in a genetically heterogeneous population where only a portion of the population has any chance of becoming, say, a centenarian.

One school of thought follows the straightforward and widely accepted demographic approach of projecting mortality trends from the past into the future. Its proponents emphasize continuity with the past—indeed, they would require compelling arguments in order to make any other assumption about the future course of mortality and longevity (Wilmoth 1998, 2000; Vaupel et al. 1998; Oeppen and Vaupel 2002). Linear extrapolations of the dramatic declines in mortality that have occurred over the last century produce forecasts of life expectancies that can only increase—reaching 100 or more within this century (Oeppen and Vaupel 2002).

The other (realist) school, in contrast, argues that it is unnecessary to assume that the future will be like the past because the present patterns of mortality that have yielded gains in life expectancy already depart from earlier experience. The rapid rise in life expectancy during the first half of the twentieth century was primarily achieved by saving children from death caused by infectious disease as well as saving mothers during childbirth. Once those earlier gains were made, there was a biologically significant transition from those sources of mortality to the major causes of death observed today—heart disease, cancer, stroke, and diabetes (Carnes, Nakasato, and Olshansky 2005). While biological differences (i.e., susceptibility) in the risks of acute death from infectious disease exist, the source of those risks (the disease vector) lies outside the individual. By contrast, the major causes of death today involve pathologic changes that originate within the individual and often progress over long periods of time. The mortality trajectories within as well as between calendar years for these two broad categories of death are different (Carnes et al. 2006). As such, the mortality experience of populations today is fundamentally different from that of earlier cohorts. The mortality of the future may well depart from both of these patterns as ways to intervene in the processes of senescence are discovered and implemented.

Observed changes in the trajectory of mortality at older ages have spawned an immense amount of discussion in the scientific literature, much of it expressed as criticism of a time-honored model in the demographic and actuarial sciences: the Gompertz model. Carey et al. (1992), for example, have declared that the Gompertz paradigm has been overturned because the linearity of its hazard function on a semi-logarithmic scale cannot capture the deceleration of death rates they observed at older ages in populations of fruit flies.

A detailed review of the literature on the Gompertz model (Olshansky and Carnes 1997) permits such criticisms to be assessed within a broader historical context. Two relevant facts emerge from that review. First, Gompertz himself warned his fellow actuaries not to use his function to describe mortality at older ages. Second, population scientists dating back to Gompertz recognized that a mixture of Gompertz equations would better capture the dynamics of human mortality (see Carnes and Olshansky 2001 for discussion). For example, Strehler (one of the most influential thinkers in the

history of biogerontology) and his colleague Mildvan (1960) demonstrated that a mixture of Gompertz subpopulations can produce the decelerations in age-specific death rates that give rise to so much discussion today. They show a slowing of the rate of increase in age-specific death rates, and even a decline, as the surviving population becomes progressively dominated by longer-lived subgroups contained within the original population. Thus, contrary to those who have declared its demise, the Gompertz paradigm remains a relevant and valuable quantitative tool for researchers who are interested in age patterns of mortality.

A naive extrapolation model applied to world record times for the one-mile run leads to the prediction that eventually the race will be completed the very moment the starting gun fires. Absurd though such a prediction may be, it forces one to acknowledge that there are biological constraints on how fast humans can run even though the biology responsible for those constraints did not evolve for that reason. Since longevity and mortality are undeniably biological phenomena, the same logic applies to them as well.

Biological barriers

Longevity optimists see no biological reasons why death rates cannot fall to zero (see, for instance, Wilmoth 2001). Realists do see such reasons. They believe there are practical limits on how low death rates can go, on the life spans of individuals, and on the life expectancy of populations. Those limits are not fixed, because senescence is not programmed into the genome. For the same reason, the intrinsic mortality patterns that give rise to them are inherently modifiable. Unlike deterministic limits, practical limits reflect the probabilistic nature of the stochastic components of senescence (Finch and Kirkwood 2000).

A fundamental lesson from the evolutionary theory of senescence can be stated as follows: *while bodies are not designed to fail, neither are they designed for extended operation*. There is no genetic program for senescence, but because bodies are not designed for extended operation, duration of life must be finite. As such, organisms are subject to the biological equivalent of a warranty period. These warranty periods vary not only from species to species, but also from individual to individual. Further, evolutionary biology would suggest that the biological constraints that define the practical or probabilistic expiration dates for these warranty periods were molded by the extrinsic mortality conditions existing at the time the species first formed. If so, there are more insights on the biology of human longevity and its demographic consequences to be gained from examining our remote past than from empirical studies of the recent past or the present. Biological constraints permeate every aspect of the biology of every organism and at every level of organization. They are extensively documented in the biological, biomedical, biogerontological, and medical literatures.

Prospects for life expectancy to exceed 85

Our prediction about the future of human life expectancy is quite specific. We first made it in 1990 and reaffirmed it in 2001. Life expectancy for humans, we assert, is unlikely to exceed 85 years (for men and women combined) unless it becomes possible to slow the rate of aging in a significant fraction of the population. On the prospects for that slowing, we wrote (Olshansky 1997: 8):

> [T]here is a considerable amount of promising research in the fields of molecular biology, genetics, and other disciplines suggesting that the basic rate of aging itself may eventually fall, to some extent, within the control of medical technology. Life expectancy at birth can rise beyond 85 years, but it is suggested here that this would require significant new advances in medical technology that "manufacture survival time" by decelerating the basic rate of aging itself and postponing death through medical intervention.

This view of the future of longevity has not been drawn out of thin air; it derives from three independent but interrelated lines of inquiry. The first of these was a (necessarily subjective) assessment of the likelihood of curtailing major causes of death that exist today (cancer, cardiovascular diseases and diabetes)—see Olshansky, Carnes, and Cassel (1990) and Olshansky, Carnes, and Désesquelles (2001). A second line of inquiry, referred to above, compared age-determined mortality of humans with two other species, mice and dogs. Assuming that the three species share a common pattern of intrinsic mortality (after appropriate scaling) and excluding extrinsic mortality and effects of medical interventions, this study produced a hypothetical life expectancy of 85 years.

Our third line of inquiry on limits was predicated on the evolutionary conclusion that bodies have biological warranty periods and that the expiration date of those warranty periods is linked to the time required to reach sexual maturity, reproduce, nurture young, and (for some species) provide grandparenting (Carnes, Olshansky, and Grahn 2003). Observed age-specific fertility patterns in mice and humans were used to infer median age of death from intrinsic causes for humans on the basis of mouse data.[12] The resulting median age (an approximation of life expectancy at birth) fell within the mid to upper 80s.

These three totally independent approaches (the last one not even involving mortality data for humans) produced nearly identical probabilistic limits for the life expectancy of human populations.

Conclusion

The future of human longevity has been a topic of discussion since antiquity. Research on longevity and the nature of aging reported in the scientific litera-

ture appears almost simultaneously in the public media. This fascination with the subject is particularly well deserved at a time when population aging is sweeping across the globe and awareness of age structure and its implications is becoming widespread. Although the debates on these topics began among population scientists, they have spread to every discipline contributing to research on the health, mortality, and longevity of humans and other species.

Competition among ideas is a healthy and natural component of scientific progress. Rivalries between schools of thought and debates among scientists are normal and to be welcomed. As long as rival positions are accurately portrayed and fairly represented, heated debates and passionately held views can be indicators of a vibrant and healthy field of study.

We have sought here to restate our views on a number of important issues concerned with aging and longevity, correcting what have sometimes been misinterpretations of them. We summarize our realist stance in terms of a number of "myths" and "realities":

Myth #1: Reaching an average life expectancy of 85 years is a pessimistic outlook for human longevity.

Reality: Attainment of that life expectancy would require death rates at every age and from all causes combined to decline by about 50 percent in this century—equivalent to the complete elimination of cancer and heart disease. This is not a pessimistic view of future trends in mortality.

Myth #2: Species possess an intrinsic mortality schedule that cannot be modified by human intervention.

Reality: Intrinsic diseases and disorders and their health consequences are inherently modifiable and have already been modified. The mortality schedule associated with them must therefore also be modifiable.

Myth #3: Realist scenarios of the future course of human longevity are based on notions of biological determinism.

Reality: Senescence and longevity are inadvertent byproducts of evolutionary neglect rather than direct products of evolutionary intent. Biological determinism has nothing to do with the longevity of any species.

Myth #4: Realists assert that there is an age beyond which there can be no survivors.

Reality: Fixed limits to life cannot exist, because neither senescence nor longevity is programmed into the genome.

Myth #5: Hypothesized biological barriers to longer life spans have been scientifically studied and refuted.

Reality: Duration of life is finite, and the constraints that limit the life span potential of an individual are undeniably biological and extensively documented in the biological, biomedical, biogerontological, and medical literature. Most funding for research on aging interventions is aimed at identifying the biological mechanisms underlying these constraints.

Myth #6: Realists claim that life expectancy at birth cannot exceed 85 years.

Reality: Humans have become adept at manufacturing survival time by managing the symptoms of intrinsic diseases such that age at death is delayed. Eventually, disease management may give way to cures for intrinsic diseases. Further, basic scientists who study aging believe that interventions that slow aging processes will be discovered. All three of these human endeavors can permit life expectancy at birth to rise beyond 85 years. This conditionally optimistic view of the future has been a consistent theme in our work and a motivation for our call for more research into the fundamental biology of aging.

In our "realistic" view, however, we have also expressed concern over two disturbing health trends that could impede or even reverse the life expectancy gains made in developed countries. The first is the threat of new infectious diseases and the re-emergence of infectious diseases previously thought to have been eradicated (Olshansky et al. 1997). The second is the rapid rise in childhood obesity occurring around the globe—now appropriately described as a global obesity epidemic (Olshansky et al. 2005). Either of these trends has the potential to affect enough people to cause declines in a population statistic like life expectancy.

The study of mortality has historically been the purview of demography and the actuarial sciences. However, as we have attempted to argue in this note, mortality and longevity are inherently biological phenomena. As with other biological phenomena, evolutionary and comparative biology provide a coherent and indispensable source of explanations of observations on age-determined mortality (Carnes 2007). This conceptual framework transforms demography into biodemography. Although biodemography in its modern form (Carnes and Olshansky 1993; Carnes, Olshansky, and Grahn 1996; Wachter and Finch 1997) is a young discipline, its true fathers—prominent among them Charlesworth (1980), Strehler and Mildvan (1960), Sacher (1956), Clarke (1950), Brody (1924), Pearl (1922), Brownlee (1919), and Makeham (1867)—built its conceptual foundations long ago. Biodemographic research benefits both of its parent disciplines. As scientists from the two disciplines learn each other's language and concepts and engage in collaborative interdisciplinary research, the resulting synergy will greatly enhance our understanding of the health, longevity, and mortality consequences of aging.

Notes

1 According to UN estimates, life expectancy in Japan, the leader, averaged approximately 82.3 years in 2005. (Note that such averages refer to both sexes combined. Female life expectancy is typically several years higher than male—in Japan female life expectancy was 85.7 years, male 78.7 years.)

2 Various other categorizations, and other terms to describe them, are common in the literature. For example, Manton, Stallard, and Tolley (1991) identified three schools of thought that they referred to as "visionary," "empiricist," and "traditionalist." Visionaries were those (e.g., Strehler 1975) who be-

lieved that advances in biomedical research will raise life expectancy to 100–125 years within the next half century (i.e., by 2025). Empiricists anticipated that current mortality declines would produce life expectancies of 95–100 years by 2080 (e.g., Ahlburg and Vaupel 1990). Traditionalists were described as those such as Olshansky, Carnes, and Cassel (1990) who suggest that limits on human life expectancy are not "significantly greater than current life expectancy, namely about 85 years." Wilmoth (1998), followed recently by Bongaarts (2006), relabeled Manton's traditionalists as pessimists and collapsed the visionaries and empiricists into a single category of optimists.

3 Assertions to the contrary that have appeared in the literature (e.g., Oeppen and Vaupel 2002) are in error—typically by misinterpreting 85 years as life expectancy for females rather than for the population as a whole.

4 In similar fashion, Robine (2006: 8) also inaccurately attributed the notion of intractability to us when he stated:

> Will it [average life expectancy at birth] continue to increase by three months every year in the most advanced countries in the demographic transition, […], as suggested by Oeppen and Vaupel (2002); or shall we henceforth measure its increase in *days or hours*, as suggested by Olshansky and his team (Olshansky et al., 2001a)? [Our emphasis]

The correct quote from the original source was: "Unless the aging process itself can be brought under control, the mortality trends observed from 1985 to 1995 remain consistent with the expectation that future gains in life expectancy will be measured in *days or months* rather than years" [our emphasis] (Olshansky, Carnes, and Désesquelles 2001: 1492).

5 Vaupel (1998: 242) more accurately represents our position when he ascribes to us the view "that health-care resources and biomedical research should increasingly be directed toward improving 'the average well being of the population' rather than extending 'the average life span.'"

6 For discussion on intrinsic mortality and its importance for research on senescence, see Carnes, Olshansky, and Grahn (1996); Carnes and Olshansky (1997); Carnes 2004; and Carnes et al. (2006).

7 The human influence on intrinsic mortality schedules and, by implication, on the underlying diseases is discussed in Carnes, Olshansky, and Grahn (1996) and Olshansky, Carnes, and Grahn (1998).

8 For example: "More pessimistic scenarios of the future course of human longevity are based on notions of biological determinism or arguments about practicality, yielding the now-familiar claim that life expectancy at birth cannot exceed 85 years" (Wilmoth 1998: 396); "A canonical gerontological belief posits genetically determined maximum life-span" (Vaupel et al. 1998: 856).

9 A line of reasoning inappropriately attributed to the realist view by Wilmoth (1998: 396) and Vaupel et al. (1998: 856).

10 Probably as a result of findings like this, Fries (2003) has modified his assumption for compression to require that the median age at death need only increase at a faster pace than the increase in the observed maximum life span.

11 Strict "fixed-limit" assertions about longevity tend to be used as straw-man devices to discredit key propositions that the investigator wants to reject. Wilmoth (1997: 60), for example, writes: "Intuitively, the limited-life-span hypothesis is unappealing because it suggests that it is possible to survive to some maximum age, ω, but not to ω plus one day."

12 The mouse studies revealed the process of reproductive senescence (smaller litters, larger parity intervals, increased pup mortality) that occurs over time. From this information, a metric called the "effective end of reproduction" (EER) was constructed. We then regressed the median age of intrinsic death for 22 strains of mice on to the average EER for the breeding females that produced those mice. Age-specific fertility patterns for humans were used to estimate a human EER (ranging from 32 to 38 years). Inserting these values in the mouse equation allowed a calculation of an estimated median age of intrinsic death for humans.

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