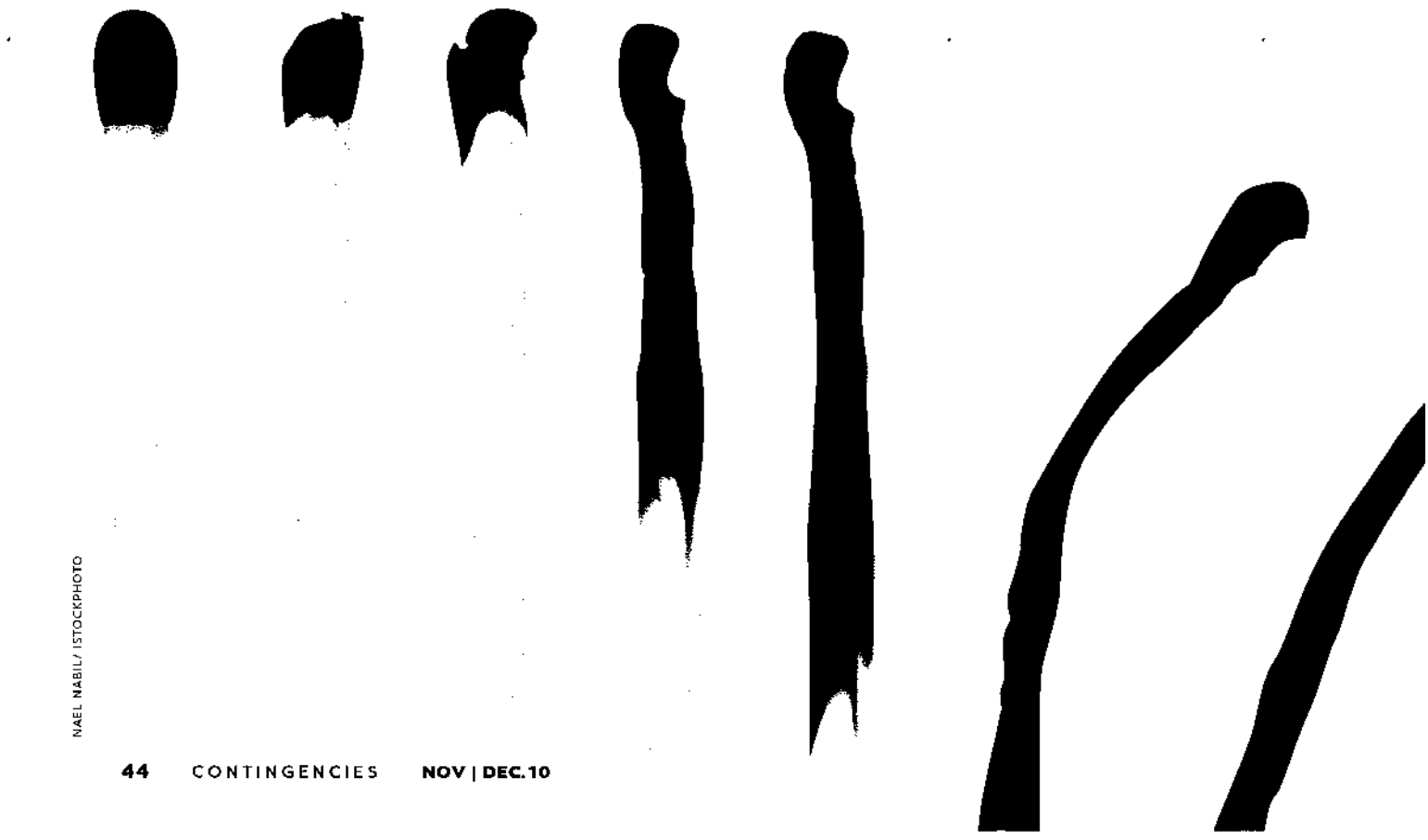
Mortality

By Victor Modugno

Mortality models, with some tweaking, have worked well over time. Are current advances in medicine going to change that?

Driven by public health improvements, rising incomes, and new treatments for disease, Americans experienced a substantial growth in longevity during the 20th century. Life expectancy at birth increased from 49 years to 77 years in that period, and mortality for those 65 and older declined by .7 percent per year.

The increase in obesity in the United States, from 15 percent of adults in 1978 to 31 percent in 2000, has led some to conclude that the trend in improving mortality will end and that life expectancy will decline in the 21st century, much as it did in Russia after the fall of Communism in 1990. At the same time, some biogerontologists claim that in the foreseeable future it may be possible to develop interventions that will reverse the aging process and extend life spans. While others in the field of biogerontology consider this a fantasy, should actuaries consider this possibility when projecting and stress-testing annuity and retirement system cash flows?



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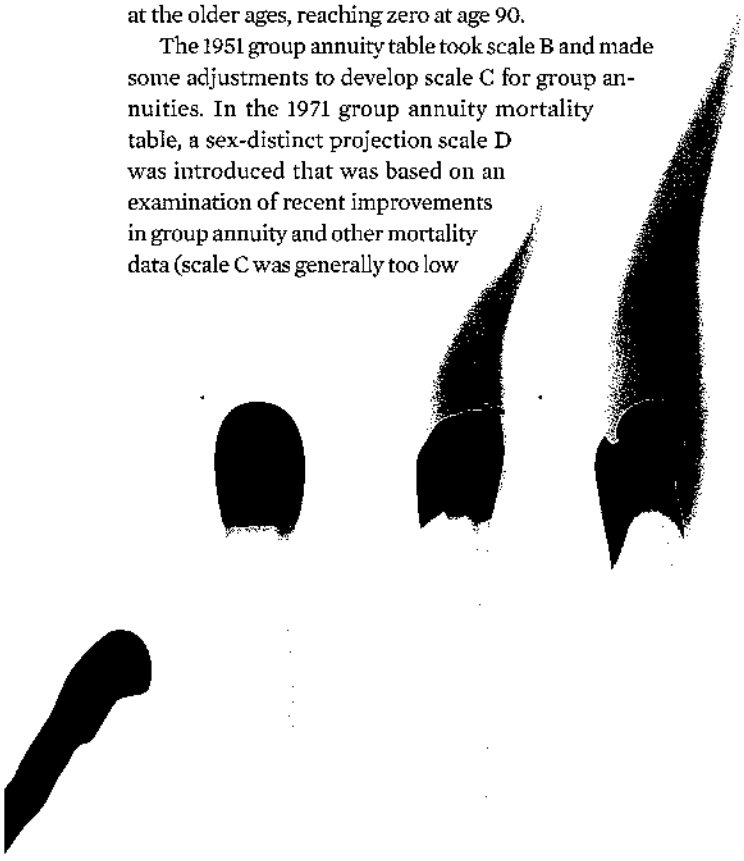
Makeover

Projecting Mortality

Projections for future improvements in mortality were first introduced for use in U.S. annuity pricing with the 1949 mortality table. The method applied an annual rate of decrease to mortality rates that varied by age. The recommended rates of future decrease (scale B) were developed by examining annuity data and long-term historical rates of decrease from a number of sources, including population studies and data from Social Security, civil service, and corporate plans, as well as foreign data. Rates of decrease by cause of death were analyzed, and judgment and adjustments for smoothing were used to develop rates of future improvement by age. Mortality improvement declined at the older ages, reaching zero at age 90.

The 1951 group annuity table took scale B and made some adjustments to develop scale C for group annuities. In the 1971 group annuity mortality table, a sex-distinct projection scale D was introduced that was based on an examination of recent improvements in group annuity and other mortality data (scale C was generally too low for females and could not be used to update the 1951 table). The 1983 table was developed by projecting mortality improvement using population data, after it was determined that scale D did not produce sufficient reduction in mortality. Scale G was developed for individual annuities from population and other data and expert opinion regarding future improvements by cause, similar to scale B. Scale H for future group annuity mortality improvement was developed by modifying scale G at the older ages to equal 0 at 100.



When sufficient group annuity experience became available, a new reserve basis, the 1994 group annuity reserve, was developed. This was the first reserve basis to include mortality projection (in the past, interest rate margins in statutory reserves were assumed to cover future mortality improvement). Dynamic valuation law and lower market rates had reduced these interest rate margins, and a mortality basis that would stay current was needed. The basic table, 1994 group annuity mortality, was reduced 7 percent and then projected using scale AA to the year of valuation. A generation projection was used thereafter in which, for example, 65-year-old mortality for the next year would be the rate for a 66-year-old with one-year projection and for the following year the rate for a 67-year-old with two years projection, etc. Scale AA was based on recent improvement in Social Security and federal civil service data (see Chart 1, next page).

Chart 2 (next page) compares mortality projected to 1994 on earlier tables with the 1994 group annuity mortality (GAM). General U.S. mortality, which is considerably higher, also is shown for comparison. With the exception of scale D, these projection scales generally would have produced adequate reserves, considering the 7 percent margin in the 1994 table. Over time, these scales will result in gradual curve-squaring mortality, in which most live into their 80s but few (albeit more than today) live into their 100s.

Medical Interventions

Anti-aging medicine practiced today generally consists of three kinds of interventions:

■ **LIFESTYLE**—This includes healthful diet; exercise; avoiding smoking, drugs, and other risky behaviors; as well as regular physicals, diagnostic tests, and adherence to medical advice to maintain healthy blood pressure; blood chemistries (sugar, cholesterols, inflammation, and other markers); and weight. Scientists and health professionals are in agreement that this should lead to longer lives. At least regarding weight, however, the U.S. population appears to be moving in the opposite direction.

■ **HORMONES**—This includes replacing testosterone, estrogen, progesterone, human growth hormone (HGH),

CHART 1

GROUP ANNUITY PROJECTION SCALES									
Male					Female				
Age	C	D	H	AA	Age	C	D	H	AA
30	1.25%	0.65%	0.75%	0.50%	30	1.25%	1.30%	1.25%	1.00%
40	1.25%	0.65%	2.00%	0.80%	40	1.25%	1.30%	2.25%	1.50%
50	1.25%	0.65%	1.75%	1.80%	50	1.25%	1.30%	2.00%	1.70%
60	1.25%	0.65%	1.50%	1.60%	60	1.25%	1.30%	1.75%	0.50%
70	1.25%	0.56%	1.50%	1.50%	70	1.25%	1.21%	1.75%	0.50%
80	0.67%	0.36%	1.25%	1.00%	80	0.67%	0.92%	1.50%	0.70%
90	0.00%	0.16%	0.75%	0.40%	90	0.00%	0.38%	1.00%	0.30%
100	0.00%	0.00%	0.00%	0.00%	100	0.00%	0.00%	0.00%	0.00%

SOURCE: AUTHOR

CHART 2:

Male	Projected Mortality to 1994			1994	2000 U.S.	GA51C/	71GAMD/	83GAMH/
Age	GA51(C)	71GAM(D)	83GAM (H)	GAM	Population	94GAM	94GAM	94GAM
40	0.001080	0.001352	0.000878	0.001153	0.002581	0.94	1.17	0.76
50	0.003496	0.004374	0.002895	0.002773	0.005687	1.26	1.58	1.04
60	0.008398	0.010859	0.007083	0.008576	0.013033	0.98	1.27	0.83
70	0.021220	0.030680	0.022230	0.025516	0.030827	0.83	1.20	0.87
80	0.071822	0.078749	0.059810	0.066696	0.071426	1.08	1.18	0.90

SOURCE: AUTHOR

dehydroepiandrosterone (DHEA), melatonin, and thyroid as they naturally decline with age. Supplementing testosterone, HGH, and estrogen has each been shown to temporarily halt certain effects of aging. None, however, has been proven to increase longevity, and to the extent they increase cancer risk, they may have the opposite effect.

■ **SUPPLEMENTS**—These include vitamins A through E, K, CoQ10, TA-65 (astragalus), fish oil, curcumin, herbal remedies, fruit juices, and other food-based antioxidants. Claims are made that these supplements can prevent or cure certain diseases or conditions or boost the immune system. To the extent that they cure or prevent age-related diseases, they could increase longevity. Most of these claims have not been subjected to rigorous scientific testing in humans, and, to the extent that they have, the results have been mixed. The exception is resveratrol, which is thought to be the source of the healthy benefits of wine.

Cosmetic procedures—such as facelifts, liposuction, and Botox injections—that have no effect on longevity sometimes are considered part of anti-aging medicine. To increase longevity, effective interventions would need to be adopted by many in the population, which seems unlikely. Even if that were to happen, the mortality improvement would be gradual and fit within the actuarial model. One result could be greater discrepancy between population and annuitant data, in cases in which annuitization is voluntary.

Resveratrol and Caloric Restriction

Caloric restriction without malnutrition has been shown to increase significantly both average and maximum life spans in laboratory rodents and other short-lived animals. Human studies have shown that caloric restriction greatly improves health, postpones age-related diseases, and increases life expectancy. But there is no evidence that it increases maximum life span. A six-month control study and data from the Calorie Restriction Society show significant improvement in blood pressure and blood chemistries, when compared with control groups, as well as the levels of those in the study before starting to restrict calories. Studies of centenarians in Okinawa, Japan, whose diets were close to caloric restriction diets, show that more reached age 100 than in any other society, but there was no increase in maximum life span—they did not live longer than centenarians living elsewhere.

A study of long-term caloric restriction in adult rhesus monkeys, begun in 1989, has reached the average life expectancy of these animals. Half the control group has died, compared with 20 percent of those eating a calorie-restricted diet. The calorie-restricted group is healthier both in appearance and when metabolic, cardiovascular, and brain functions are measured. Deaths from cardiovascular disease and cancer are 50 percent lower for the calorie-restricted group, and diabetes is nonexistent in the calorie-restricted group, while 42 percent of the

control group is either diabetic or pre-diabetic. It will be another decade or so before researchers can determine whether calorie restriction increases the maximum life span of long-lived primates (which, for rhesus monkeys, is about 40 years).

How long calorie restriction extends human life would be of academic interest in population mortality, given rising obesity levels. But resveratrol, which is in the skins of grapes and is thought to be the source of the cardio-protective effects of wine, appears to mimic the effect of calorie restriction in animal studies. Resveratrol extends the life span of worms, fruit flies, and fish. It lowered mortality 30 percent in mice fed a high-fat diet. A study of low-dose resveratrol in mice reported a striking overlap of calorie restriction and resveratrol in heart, skeletal muscle, and brain. It concludes, "Resveratrol, at doses that can be readily achieved in humans, fulfills the definition of a dietary compound that mimics some aspects of CR (calorie restriction)."

A Massachusetts-based pharmaceutical company, Sirtris Pharmaceuticals, is developing drugs based on the effect of resveratrol and calorie restriction on sirtuins, the enzymes associated with aging. It currently has drugs in clinical trials for Type 2 diabetes, heart disease, and cancer. To the extent these drugs are better or safer than existing drugs, they may offset the expected increase in mortality from rising obesity. In fact, because several new drugs are being tested for diabetes and other diseases, it's probable that some eventually will result in improved treatments that will offset the higher mortality associated with growing obesity in the general population.

Engineered Negligible Senescence

Aging can be thought of in two ways: primary aging, the increasing frailty and susceptibility to age-related diseases, and secondary aging, the pathology that ends life. While all warm-blooded animals age, primary aging may not be inevitable. Engineered negligible senescence was used by the author and gerontological theoretician Aubrey de Grey to describe interventions that will reverse accumulated metabolic damage in cells, thereby postponing aging indefinitely. The SENS (Strategies for Engineered Negligible Senescence) website describes therapy, possible with current or foreseeable biotechnology, that addresses seven major categories of aging damage:

1 CELL LOSS WITHOUT REPLACEMENT—This affects brain and heart cells, among others, and can be fixed by stimulating cell division (by exercise or injection of growth factors and hormones) or by introducing new ones through cell therapy. Stem cell research should provide for this.

2 NUCLEAR MUTATIONS (CANCER)—This could be cured by a process, referred to as whole-body interdiction of lengthening of telomeres, in which cells would have their telomerase genes removed, thereby limiting the number of times they could divide and preventing cancer from growing. Stem cells that need to divide would be replaced by cells with restored telomeres every 10 years or so.

3 MITOCHONDRIAL MUTATIONS—Mitochondria are parts of cells that produce energy. The process can damage some DNA, which plays an important role in aging. The solution—termed "allotropic expression"—involves the use of gene therapy to introduce copies of the 13 unique mitochondrial genes into the nucleus.

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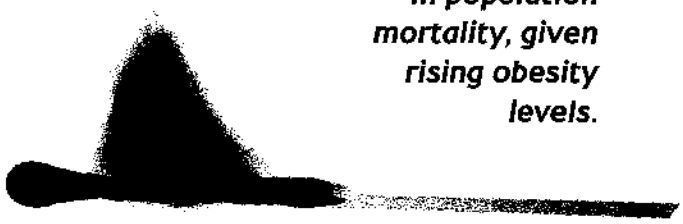

4 DEATH-RESISTANT CELLS—These include visceral fat cells and senescent cells that have lost the ability to reproduce and that accumulate in the cartilage in joints. These also include immune cells that lose their effectiveness over time and don't make room for other kinds of immune cells, resulting in immune system decline in the elderly. The solution is to use distinctive molecules on the target cells for the injection of a drug to kill the cells or have the immune system kill the cells.

5 TISSUE STIFFENING—Caused by extracellular cross links (proteins outside the cells that form chemical attachments), tissue stiffening, such as hardening of the arteries or the loss of flexibility in ligaments, can be solved by finding or engineering enzymes or proteins to break the cross links.

6 EXTRACELLULAR AGGREGATES—Amyloids in between cells cause diseases such as Alzheimer's disease and diabetes. The solution is a vaccine to stimulate the immune system to purge the junk outside of cells.

7 INTRACELLULAR AGGREGATES—There's also junk inside cells, which they can't completely clear. This causes atherosclerosis and is important in several types of neurodegenerative diseases and in macular degeneration. One solution lies in employing microorganisms, present in soil, that have enzymes capable of breaking these aggregates down.

This list is complete based upon current knowledge, but it's possible that some cause of aging is missing. In addition, some of the assumptions underlying the proposed solutions may not be correct. The real issue, however, is the probability of developing safe and effective therapies for all these conditions.

Writing in a 2005 European Molecular Biology Organization (EMBO) report, Huber Warner, associate dean for research at the University of Minnesota College of Biological Studies, points out: "Most therapeutic ideas, even the most plausible, come to nothing—in pre-clinical studies or clinical research, the proposed interventions are found to be toxic or induce unwelcome side effects, are mooted by more successful ideas, or, most

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often, simply fail to work as hoped. Each one of the specific proposals that comprise the SENS agenda is, at our present stage of ignorance, exceptionally optimistic. Therefore, by multiplying the probabilities of success, the claim that all of these proposals can be accomplished, although presented with confidence in de Grey's writings, seems nonsensical."

The response from de Grey, and other scientists who agree with the SENS possibility, is that the success of these therapies can't be categorically ruled out. They argue that within 10 years some of these therapies successfully should extend the life span of mice, and have offered an award to scientists who achieve that. This 10-year time frame was first discussed in 2002 and seems to stay at 10 years in later proclamations. An award was given for the discovery of dwarf mice that live longer because of lack of growth hormone, but the real test is to develop therapies that significantly extend the life of normal mice. According to de Grey, the first human therapies should be available in 25 years. After that, new therapies will develop each year to extend life spans more than one year, which he refers to as "longevity escape velocity."

To the extent these therapies result in improved treatment of age-related diseases such as cancer, they probably fit within the actuarial model of mortality improvement. If these therapies reverse primary aging and significantly extend life span, however, the actuarial model of mortality improvement would be inadequate. Even under the most optimistic assumptions, these therapies are at least 25 years in the future. For well-capitalized and diversified life insurers, the present value of annuity payments beyond 25 years from the present shouldn't be significant.

The Past as Future?

The next 50 years are likely to resemble the previous 50 years. Periods of rapid mortality improvement may alternate with periods of slower improvement or reversal, but this should average to the gradual improvement in the actuarial model.

Terrorism, war, natural disasters, carcinogenic environmental degradation, new drug-resistant microbes, pandemics spread by air travel, and diseases caused by increasing obesity will be offset by new drugs and improved treatments for cancer, Type 2 diabetes, infectious disease, cardiovascular disease, and organ replacement. Safer vehicles should reduce accidents, while growing environmental awareness limits damage and smoking is prohibited in more places.

Then there's obesity. A linear projection of current trends produces 100 percent obesity by the end of this century, an absurd result. A projection of smoking trends at the beginning of the past century might have come to a similar result. Public health is starting to focus on this problem (e.g., Michelle Obama's efforts on childhood obesity). Obesity levels eventually should stabilize, if not decline.

While expected mortality is appropriate for reserves, capital requirements should reflect the worst likely scenario. Should this scenario reflect de Grey's opinion that a future 1,000-year-old man will walk among us? While engineering negligible senescence is improbable, it's possible that some of the SENS agenda could result in a significant increase in the ultimate human life span. Even the most optimistic advocates, however, see the earliest human therapies at least 25 years in the future. The present value of this contingency wouldn't be significant

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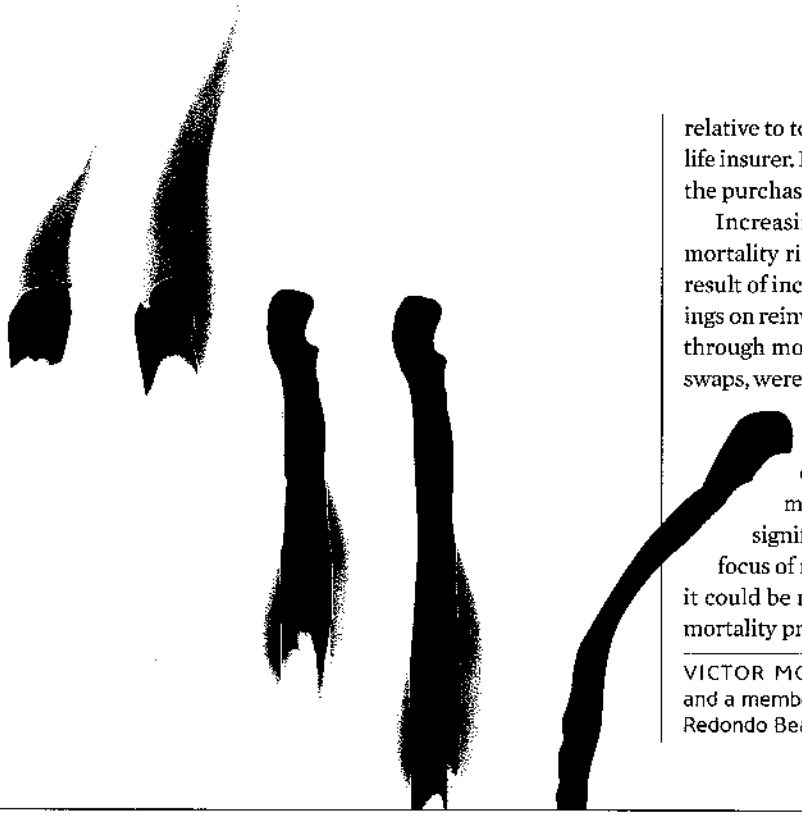

relative to total assets of a safe annuity provider, a highly rated life insurer. In the U.S., pension liabilities are defeasible through the purchase of an annuity from such a provider.

Increasing asset returns should provide an offset to the mortality risk. Inflation and high interest rates are the likely result of increased longevity on entitlements, improving earnings on reinvestment. The separation and concentration of risk through mortality swaps in Europe, which, like AIG's default swaps, were set up without reserves and adequate capital, could be a different story.

While SENS currently doesn't appear to be commercially significant for annuity providers, risk managers shouldn't ignore it. If aging can be reversed significantly in mice through SENS therapies—a current focus of research and prize money—then the possibility that it could be replicated in humans should be taken seriously in mortality projections. □

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