Could Developments in Anti-Aging Medicine Invalidate the Actuarial Model of Mortality Improvement?

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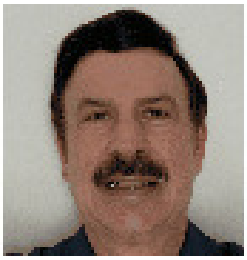

Actuary with Expertise in Pensions and Institutional Products of Life Insurance Companies including GICs, Funding, Agreements, Structured Settlements and Terminal Funding Annuities. Testimony on insurer mortality, interest, and expense assumptions used for terminating pension plans. Cases include In re US Airways (bankruptcy) and Sunkist v. Harding (ERISA multiemployer plan withdrawal liability). Over 20 papers have been published, including one that won a Society of Actuaries award.

Abstract

The actuarial model of mortality improvement used in pricing annuities and projecting cash flows for retirement systems assumes a gradual, "curve squaring" decline in mortality rates. There is a scale where mortality decreases by a small percent each year (depending on age and gender) and a final age where morality reaches 100%. The model worked reasonably well, although age/gender specific rates of improvement had to be tweaked over time. There were no developments affecting the ultimate survival age. Advances in procedures and pharmaceuticals took time to be accepted and adopted. There were offsetting trends – decreased smoking/increasing obesity, new drugs and treatments/ new diseases and drug resistant microbes – that moderated the pace of improvement. Drugs based upon Resveratrol, featured on 60 Minutes as mimicking caloric restriction and possibly increasing life span by 20%, are in clinical trials. That program has also featured research of Dr. Aubrey de Grey of Cambridge University who has contended that life spans of 1,000 years may be possible in the near future. This paper examines research in anti-aging medicine to ascertain the likelihood of a paradigm shift that would invalidate the actuarial mortality model and concludes that this will not happen in the commercially significant future but that biogerontology research should be monitored.

Introduction

The 20th century saw substantial declines in mortality and increases in life expectancy in the United States driven by public health improvements, rising incomes, and new treatments for disease. Life expectancy at birth increased from 49 years to 77 years during that century. Mortality for those 65 and older declined .7% per year during the same period [23]. While this trend has been generally favorable for life insurers, increases in annuities and retirement funds after World War II led to the development of projection scales for future improvement in annuitant mortality.

The increase in obesity in the United States, from 15% of adults in 1978 to 31% in 2000 have led some to conclude that the trend in improving mortality will end and life expectancy will decline in the 21st century, much as it did in Russia after 1990 [17,18]. On the other hand, there are some biogerontologists who are claiming that it may be possible to develop, in the foreseeable future, interventions that will reverse the aging process itself allowing a long lifespan [8]. Other biogerontologists consider this to be fantasy [29]. In projecting and stress testing annuity and retirement system cash flows, these possibilities need to be considered.

Actuarial Models of Mortality Improvement

Mortality projection for future improvement was first introduced for use in annuity pricing in the U.S. with the 1949 Annuity Table. The method was to apply an annual rate of decrease that varied by age to the mortality rates. The recommended rates of future decrease, scale B, were developed by looking at recent and long term historical rates of decrease from a number of sources, in addition to annuity data, such as population, social security, civil service, corporate plans, and foreign data. Rates of decrease by cause of death were analyzed. 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 [15]. The Group Annuity 1951 table took scale B and made some adjustments to develop scale C for group annuities [21].

The 1971 GAM (Group Annuity Mortality) introduced a sex distinct projection scale D. This was based upon examination of recent improvements in group annuity and other mortality data. Projection scale C was generally too low for females and could not be used to update the 1951 table [11]. The 1983 GAM was developed by projecting mortality improvement using

population data, after it was determined that scale D did not produce sufficient reduction in mortality. Scale H for future mortality improvement was developed by modifying scale G developed for individual annuities at the older ages to equal 0 at 100. Scale G was developed from population and other data and expert opinion regarding future improvements by cause, similar to scale B [6,15].

When sufficient group annuity experience became available, a new reserve basis, 1994 GAR (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 have reduced these interest rate margins and a mortality basis that would stay current was needed. The basic table, 1994 GAM was reduced 7% and then projected using scale AA to the year of valuation. A generation projection was used thereafter where for example 65 year old mortality for next year would be the rate for a 66 year old with 1 year projection and the following year a 67 year old with 2 years projection, etc. Scale AA was based upon recent improvement in Social Security and Federal Civil Service data [26].

Group Annuity Projection Scales

Male				
Age	C	D	H	AA
30	1.25%	0.65%	0.75%	0.50%
40	1.25%	0.65%	2.00%	0.80%
50	1.25%	0.65%	1.75%	1.80%
60	1.25%	0.65%	1.50%	1.60%
70	1.25%	0.56%	1.50%	1.50%
80	0.67%	0.36%	1.25%	1.00%
90	0.00%	0.16%	0.75%	0.40%
100	0.00%	0.00%	0.00%	0.00%

Female				
Age	C	D	H	AA
30	1.25%	1.30%	1.25%	1.00%
40	1.25%	1.30%	2.25%	1.50%
50	1.25%	1.30%	2.00%	1.70%
60	1.25%	1.30%	1.75%	0.50%
70	1.25%	1.21%	1.75%	0.50%
80	0.67%	0.92%	1.50%	0.70%
90	0.00%	0.38%	1.00%	0.30%
100	0.00%	0.00%	0.00%	0.00%

The following table compares mortality projected to 1994 on the earlier tables to the 1994 GAM:

Age	Male Projected Mortality to 1994			1994 GAM	2000 U.S. Population	GA51C / 94GAM	71GAMD / 94GAM	83GAMH / 94GAM
	GA51(C)	71GAM(D)	83GAM(H)					
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

What this table shows is that with the exception of scale D, these projection scales would generally have produced adequate reserves, considering the 7% margin in the 1994 table. US population mortality, which is considerable higher, is also shown for comparison [1]. These scales over time will result in gradual curve squaring mortality – where most live into their 80s but few (albeit more than today) live into their 100s.

Anti-Aging Medicine

Anti-aging medicine practiced today generally consists of three kinds of interventions [4,30]:

- Lifestyle – this includes healthy diet, exercise, avoiding smoking, drugs, and other risky behaviors. It also includes regular physicals and diagnostic tests and following medical advice to maintain healthy blood pressure, blood chemistries (sugar, cholesterols, inflammation, and other markers), and weight. It is widely agreed by scientists and health professions that this would lead to longer life expectancy – but at least regarding weight, as previous obesity statistics cited demonstrate, the U.S. population is moving in the opposite direction.

- Hormones – this includes supplementing testosterone, estrogen, progesterone, human growth hormone (HGH), Dehydroepiandrosterone (DHEA), melatonin, and thyroid to the extent they decline with age. Testosterone, HGH, and estrogen have temporary anti-aging effects. However none have 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, TA65 (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 they cure or prevent age related diseases, they could increase longevity. Resveratrol, which is thought to cause the health benefits of wine and is touted as a longevity supplement is discussed in the next section. Most of these claims have not been subjected to rigorous scientific testing in humans. To the extent they have, the results have been mixed.

Cosmetic procedures that have no effect on longevity are sometimes considered part of anti-aging medicine. In order to increase longevity, interventions that are effective would need to be adopted by many in the population, which seems unlikely. Even if happened, 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 where annuitization is voluntary.

Resveratrol and Caloric Restriction

Caloric Restriction without malnutrition (CR) has been shown in to significantly increase both average and maximum life span in laboratory rodents and other short lived animals. Human studies to date have shown CR greatly improves health, postpones age related diseases, and increases life expectancy – but there is no evidence it increases maximum life span. A six month control study and data from CR Society (a group practicing CR for about 6 years) shows significant improvement in blood pressure and chemistries, compared to control groups and their own levels prior to starting CR. Studies of Okinawan centenarians, whose history was close to CR, shows that more reached age 100 than any other society, but no increase in maximum lifespan – they did not live longer than centenarians elsewhere [10,12].

A study of long-term CR in adult rhesus monkeys starting in 1989 has reached the average life expectancy of these animals, and half the control group has died, compared to 20% of the CR group. The CR group is healthier both in appearance and measurement of metabolic, cardiovascular, and brain function. Deaths from cardiovascular disease and cancer are 50% lower for the CR group. Diabetes is not existent in the CR group while 42% of the control group is either diabetic or pre-diabetic [5]. It will be another decade or so before it can be determined whether CR increases the maximum lifespan of long-lived primates, which for rhesus monkeys is about 40 years.

How long CR extends human life would be of academic interest in population mortality, given rising obesity levels. However, resveratrol, which is in grape skin and is thought cause the cardio-protective effects of wine, appears to mimic the effect of CR in animal studies. Resveratrol extends the lifespan of worms, fruit flies, and fish [19]. It lowered mortality 30% on mice fed high fat diet [3]. A study of low dose resveratrol in mice reported: “a striking transcriptional overlap of CR and resveratrol in heart, skeletal muscle and brain,” and concludes: “Resveratrol, at doses that can be readily achieved in humans, fulfills the definition of a dietary compound that mimics some aspects of CR.”[2]

Sirtris™ Pharmaceuticals is developing drugs based upon effect of resveratrol and CR on sirtuins, which are enzymes associated with aging. They currently have drugs in Phase IIa clinical trials for type 2 diabetes, heart disease, and cancer [24]. To the extent these drugs are better or safer than existing drugs; they may offset the expected increase in mortality from rising obesity. Indeed there are several new drugs being tested for diabetes and other diseases, and eventually some will result in improved treatments that will offset obesity driven diseases. Thus they are unlikely to impact the validity of the actuarial model for mortality improvement.

Engineered Negligible Senescence

Aging can be thought of in two ways – primary aging, the increasing frailty and susceptibility to disease and secondary or age related diseases, the pathology that ends life. While all warm-blooded animals age, primary aging may not be inevitable [20]. Engineered Negligible Senescence was used by Dr. de Grey to describe interventions that will reverse accumulated metabolic damage in cells thereby postponing aging indefinitely [8]. The SENS (Strategies for Engineered Negligible Senescence) website has the most current information. It describes seven major types of therapy possible with current or foreseeable biotechnology that address seven major categories of aging damage [22]:

1) Cell loss without replacement – this effects 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) – A cure for cancer referred to as “WILT” (Whole-body Interdiction of Lengthening of Telomeres). Cells would have their telomerase genes removed 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. They have some DNA that can be damaged by this process, 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.

4) Death-resistant cells – These include visceral fat cells, senescent cells that have lost the ability to reproduce and accumulate in the cartilage in joints, and immune cells that lose their effectiveness over time and do not make room for other kinds of immune cells, which results in the decline of immune system in the elderly. The solution is using distinctive molecules on the target cells to inject a drug to kill to cells or to have the immune system kill the cells.

5) Tissue stiffening caused by extra-cellular cross-links (proteins outside the cells that form chemical attachments) – for example hardening arteries and loss of flexibility in ligaments. This can be solved by finding or engineering enzymes or proteins to break the cross-links.

6) Extracellular aggregates – amyloids in between cells causing diseases such as Alzheimer's and diabetes. The solution is a vaccine to stimulate the immune system to get rid this junk outside of cells.

7) Intracellular aggregates – junk inside the cells that they cannot completely clear. This causes atherosclerosis and is important in several types of neurodegenerative diseases and macular degeneration. The solution is finding micro-organisms present in soil that have enzymes capable of breaking these aggregates down.

This list is complete based upon current knowledge, but it possible that some cause of aging is missing. Also some of the assumptions underlying the proposed solutions may not be correct. However the real issue is the probability of developing safe and effective therapies for all these conditions. As critics of the SENS agenda point 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 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."[29]

Dr. de Grey's response is the success of these therapies cannot be categorically ruled out – he has a list of scientists who agree with SENS possibility. In 10 years some of these therapies should successfully extend the life span of mice with an award offered for this [9]. This ten year timeframe was first discussed in 2002 and seems to stay at 10 years in later work [8]. An award was given for the discovery of dwarf mice that live longer due to lack of growth hormone, but the real test is to develop therapies that will significantly extend the life of normal mice [16]. According to Dr. 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".[22]

To the extent these therapies result in improved treatment of age related disease such as cancer, they probably fit within the actuarial model of mortality improvement. However, if these therapies reverse primary aging and significantly extend lifespan, the actuarial model of mortality improvement would be inadequate. Even under the most extreme, 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 now should not be that significant.

Conclusions

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, 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 environmentalism limits damage and smoking is prohibited in more places. Regarding obesity, a linear projection of current trends produces 100% obesity by the end of this century, an absurd result. A projection of smoking trends at the beginning of the last century might have come to a similar result. Public health is starting to focus on this problem (e.g., Michele Obama's efforts on childhood obesity). Obesity levels should eventually stabilize, if not decline.

While expected mortality is appropriate for reserves, capital requirements should reflect the worst likely scenario. Should this scenario reflect Dr. de Grey's opinion that a future 1,000 year old man walks among us? While engineering negligible

senescence is improbable, it is possible that some of the SENS agenda could result in significant increase in the ultimate human life span. Even the most optimistic advocates see the earliest human therapies at least 25 years in the future. The present value of this contingency would not be significant relative to total assets of a safe annuity provider – a highly rated life insurer. In the U.S., pension liabilities are defeasible through hard dollar annuity purchase from such a provider. The assets 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 could be a different story [13], like AIG's default swaps, which were set up without reserves and inadequate capital.

While SENS does appear to be not commercially significant at this moment for annuity providers, risk managers at these firms should monitor developments in this area for changes. In particular, if aging can be significantly reversed in mice through SENS therapies – a current focus of research and prize money – then the possibility it could be done in humans should be taken seriously in mortality projections.

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