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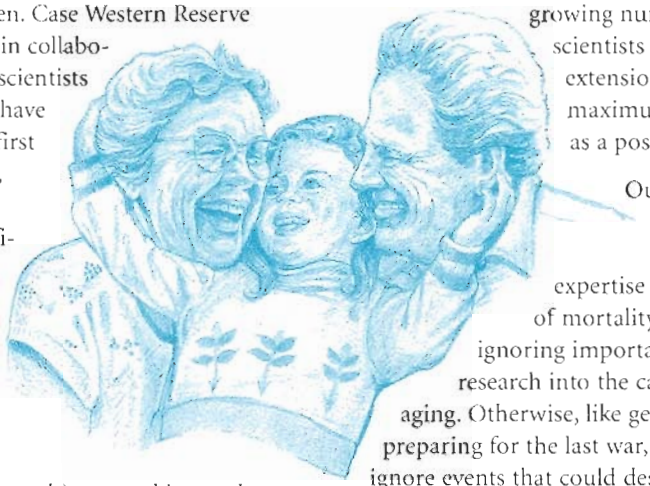
Biology, aging, and the actuary

by Gene Held

Developments in biology during the last decade border on the surreal, far outstripping Hollywood's most ambitious science fiction. With each day, we learn that a new clone has been created, that stem cells hold the promise of curing still another disease, or that some new method has been developed for fighting cancer. Pharmaceutical companies insert genes for desirable drugs into livestock in order to harvest products from the animals' milk, urine, or blood (a process known as "pharming"). Scientists grow new blood vessels from a few cells in a test tube, and researchers from around the world are collaborating to grow an entire human heart within the next decade. Cornell scientists have embarked on a program to create artificial wombs to help women with damaged uteruses have children, and last year a woman served as the surrogate mother for her daughter, thereby becoming the "mother" of her own grandchildren. Case Western Reserve researchers, in collaboration with scientists at Athersys, have created the first functioning, replicating, human artificial chromosome. And scientists at TIGR (The Institute for Genome Research) are working to determine the minimum set of genes necessary to sustain life, a feat which, if successful, will provide a de facto definition for one of mankind's oldest questions: "What is life?" Once achieved, they propose a still more audacious endeavor—to use those genes to create life. The revolution in biology is not only mind-bending, it is quite simply the most important thing taking place on the planet today. If you have not yet familiarized yourself with the technology, do so. Ignoring it would be like sleeping through the Renaissance.



The spin-off from the Human Genome Project is resulting in a rapidly escalating base of knowledge about life processes at their most fundamental level, and knowledge gleaned from that endeavor may offer the prospect of slowing the aging process. Dr. Francis Collins, Director of the National Human Genome Research Institute, has predicted, "By 2030, major genes responsible for the aging process in humans will likely have been identified, and clinical trials with drugs to retard the process may well be getting underway." A growing number of scientists recognize extension of the maximum life span as a possibility.

Our profession cannot lay claim to expertise in the area of mortality while ignoring important scientific research into the causes of aging. Otherwise, like generals preparing for the last war, we may ignore events that could destroy the assumptions underlying our projections of the future. Scientific research into the aging process began long before the Human Genome Project, but it has benefited greatly from the powerful tools and techniques spun off from that effort. Some 7,000 papers are published each year reporting on some aspect of the aging process.

Modern efforts at understanding aging can be traced to the landmark 1961 paper by Leonard Hayflick and Paul Moorhead, in which the two scientists demonstrated that normal human embryonic cells are capable of dividing only 40–60 times before becoming replicatively senescent. That overturned nearly half a century of dogma maintaining that, *in vitro*, cells were capable of dividing indefinitely. In the mid-1990s it was determined that the senescent

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editorial

Morris Fishman
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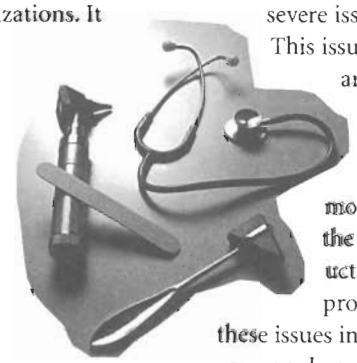
Searching for Omega

by Morris Fishman

Living to 100—who really cares? Well, I do, for one. It has been one of my personal goals for some time now. Having reached the halfway point last year, the subject took on special meaning for me. Who else cares? At least one hundred more people, based on attendance at the Symposium "Living to 100 and Beyond—Survival at Advanced Ages."

This Symposium was sponsored by the Society of Actuaries and co-sponsored by fourteen additional organizations. It was chaired by Bob Johansen and Tim Harris, who recruited experts from around the world to speak at this event. The multi-disciplinary sessions brought together some of the leading minds from fields related to advanced age research, including Professors of Anatomy, Demographic and Aging Research, experts from the Bureau of the Census and Social Security Administration, and actuaries with particular interest in the subject.



What came out of this meeting? For one thing, living to 100 may be too modest a goal, as it has become a routine occurrence. One must become a supercentenarian, 110 years old, to be considered exceptional in some circles. Second, aging is not a disease, and it's not contagious.

One major area of disagreement is human life span. Despite the incredible increases witnessed in life expectancy, we haven't seen much movement in Omega. One leading researcher stated his belief that the best the human race can hope for is 125, a feat that has not yet been credibly accomplished. The Plastic Omega theory suggests that the maximum life span may be susceptible to extension. Another theory has humans wearing out similar to machines. Maybe someone will come along and build a better human!

Why are these issues important to actuaries? The problems associated with age—such as poverty, frailty and other health issues—have not really changed. What has changed is the number of people that reach this stage in life. What once were severe issues for only a handful of people will become severe issues for a much larger group. This issue crosses over several actuarial specialties. How many times have you thought about the liabilities represented by the tail of the mortality table you chose for the task at hand? Do the products you design and the programs you recommend take these issues into account? Are there things you can do as an individual to improve the situation?

Data quality has always been an issue at the higher ages. What will be the significance of data quality when there are sixty times more centenarians than there are today?

There is good news in all of this. Although we haven't done much to eliminate aging or improve lifespan, there have been great strides to improve the quality of the years we do have. Studies of extrinsic mortality show that the health of those occupying age groups today are healthier than ten years ago, and the trend is expected to continue. Commercials with 90-year-old water skiers will not be as surprising as they once were.

Should I revise my goal to 110? What is your goal, and what are you doing to achieve it?

Morris Fishman can be reached at morris.fishman@prodigy.net.

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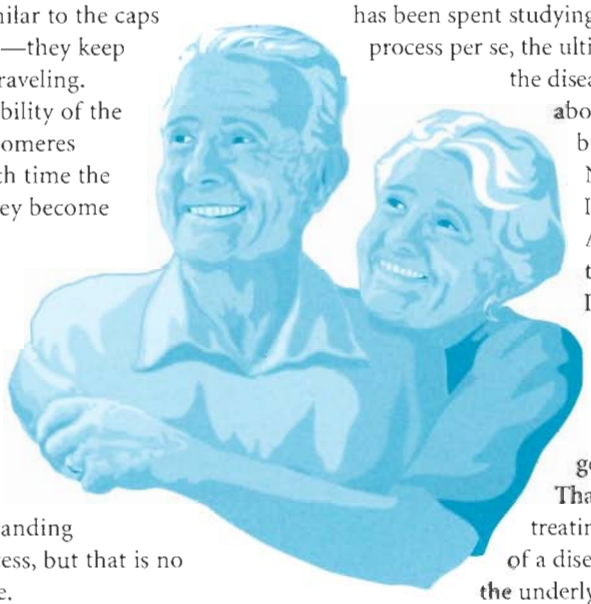
state of the cells was caused by telomere shortening. Telomeres are structures at the ends of chromosomes whose function is similar to the caps on shoestrings—they keep them from unraveling. Due to the inability of the cell to copy telomeres completely each time the cell divides, they become shorter and shorter until eventually cell division ceases. At first it was thought that telomeres might be the key to understanding the aging process, but that is no longer the case.



There are many competing theories of aging, over 300 in fact. It is helpful when studying these theories if one keeps a few key distinctions in mind. First, geriatric medicine describes in great detail how we age—that is, the changes that take place to our bodies over time. But currently we don't have a clear idea as to what causes us to age. That's the focus of ongoing research, which is aimed at explaining the cellular, genetic, and molecular events resulting in the gradual decline of function we call aging. Evolutionary theory, on the other hand, explains why we age quite nicely. The general idea, proposed by Sir Peter Medawar, will sound familiar to actuaries: the force of natural selection declines with time.

It should also be noted that aging is not a disease—nor are the diseases of old age part of the normal aging process. Rather, they are a result of aging. They are a consequence of the body's reduced capacity to fend them off. To date, much scientific time, effort, and money has been invested studying those diseases. Yet relatively little has been spent studying the aging process per se, the ultimate cause of the diseases. Oddly, only about 5% of the budget for the National Institute on Aging (part of the National Institutes of Health), is spent on true aging research. The rest goes to geriatric research. That's like a doctor treating the symptoms of a disease rather than the underlying cause. This is an unfortunate state of affairs, because it represents a lost opportunity. Geriatric medicine is something that can be carried out by the consortium of interests currently serving the public. True aging research, however, is too long-range and too risky for a private enterprise to gamble its financial existence on. However, that's just the type of fundamental research the government has traditionally funded in the past—long range, over-the-horizon science that would provide enormous benefits if successful. (Obviously, in this case it would also create a vast new set of issues to be addressed.)

Oddly, only about 5% of the budget for the National Institute on Aging (part of the National Institutes of Health), is spent on true aging research. The rest goes to geriatric research.

A distinction should also be made between aging and longevity, which represent two sides of the same coin. Aging is a stochastic process of deterioration that occurs after reproductive maturity. It results from a degradation of cellular processes that has multiple etiologies, such as reactive oxygen species, glycosylation, etc. As such, it is not a phenomenon that could be selected by evolution. That is, there are no genes for aging. Hayflick argues that the reason we live as long as we do is because physiological reserves, or redundant capacities, were evolutionarily selected because they enhanced the probability of survival to reproduce. These excess capacities only incidentally permit us to live for some time past that. Longevity, on the other hand, depends on both genetic and environmental influences. Caleb Finch and Robert Ricklefs, professors at the University of Southern California and the University of Pennsylvania, respectively, comment, "Aging itself may not be caused by our genes, but genes may nonetheless regulate its expression and rate."

Finally, we should also recognize that an increase in life expectancy is not the same thing as an increase in life span. This is the reason for the "squaring" of the survival curve. The nation experienced an increase in life expectancy of over 50% during the last century. But the maximum age to which we can live has not changed. (Jeanne Louise Calment, a Frenchwoman, lived 122 years and 164 days. She was born February 21, 1875 and died August 4, 1997.) In order to demonstrate a slowing of the aging process, you must demonstrate an increase in life span—not life expectancy.

Once you jump into the field of aging research, you begin to discover all sorts of oddities. For instance, bacteria and many other single-celled organisms don't age. (That doesn't mean they don't die, just that they are capable of dividing indefinitely. They still may be eaten, starve to death, or succumb to environmental factors.) And some animal

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species do not seem to age at all. Those that reach a fixed size in adulthood, age. These include mammals and birds. But some animals do not reach a fixed size and do not age. Examples are some lobsters, reptiles, and many fish. Non-aging animals simply grow larger, without experiencing a decline in physical capability or reproductive function.

As has already been mentioned, there are a multitude of theories attempting to explain the aging process. There are wear-and-tear theories, immune system and caloric restriction theories, theories that point to the brain or the effect of hormones, programmed aging and waste product accumulation theories, and a host of others. Oxidation and glycosylation have been pointed to, also, along with the actions of certain genes. Research into the genes involved in determining longevity is one of the more interesting and promising areas of research right now. (More about that, plus additional details and references, can be found in the paper "Plastic Omega," which was presented at the SOA's Living to 100 And Beyond conference in January, 2002. Papers presented at the conference are now posted on the Society's Web site. "Plastic Omega" was chosen as the paper's title to denote the possibility that the maximum life span may be malleable and susceptible to extension.)

Will we really be able to stop the aging process? Given time and the funds necessary for research, most scientists seem to agree that we will influence the aging / longevity process to some extent. However, there is a great difference of opinion as to the degree and the point in time at which we will be able to do so. In fact, a major battle is shaping up between the different factions. Some scientists feel the potential has been greatly overplayed, doing a great disservice to serious scientists who have spent a major portion of their careers putting the science of aging on a respectable footing. They see the overzealous claims of some researchers as bordering on "snake oil" remedies and fear a withdrawal of already scarce funds through destruction of the "Credibility = Reputation = Funding equation." Others, at the opposite end of the spectrum—who tend to be the "gene jockeys" involved in cutting edge research aimed at uncovering the mysteries of cellular processes—are supremely confident that we will not only find what causes us to age, but will do so soon, and will be able to develop methods of slowing the process greatly. Whatever your own conclusion, if you choose to research the aging process you will discover an incredibly rich and deeply fascinating field of inquiry—one that younger actuaries may someday have to deal with on a professional level.

Gene Held, FSA, MAAA, is Vice President, Pricing for SCOR Life Re in Addison, Texas. He can be reached at gheld@scor.com.

Living to 100 and beyond: Why do we care?

by Anna M. Rappaport

The "Living to 100 and Beyond" Conference focused on understanding issues and in improving research with regard to high age mortality. There is a lot of disagreement among experts about the future, and about whether developments in biotechnology could lead to major change. This disagreement leads to uncertainty. There is also a lot of difficulty in securing data, as the data at very high ages is scattered. In addition, there are major issues of reliability with regard to this data. While it is possible to verify birth records for some people who are at high ages, this does not occur very often, which leads to the importance of using models to fill in the end of the mortality table. The models depend on the data, the understanding of present state, and our ideas about the future. From a research point of view, this is a fascinating topic.

Alan Parikh and I wrote a paper for "Living to 100" because we see profound implications for our society as it ages, regardless of whether there is much change in age spans. Based on today's life spans and the historical patterns of birth, the average age of our population and the age distribution of our population will continue to change—and that change will create substantial opportunities for business, as well as presenting challenges to all of us. Our paper focuses on the implications of this change rather than on the research. It helps us to focus on why this research is so important, and how the work that actuaries will do is important to our society. We also note that the issues are global. The populations in European countries and Japan are aging earlier than those in North America. Many less developed countries will age later, but the speed of change in some of them is much more rapid.

Living longer has a bright side, but there is also a dark side to aging. As we are living longer, we are remaining healthier longer. Some experts state that the period of added health is about equal to the period of added life. But for many people,

there is still a period of decline and frailty when they need a certain amount of help. Our interest in working, as well as our ability to work, changes as time goes on, but often not as fast as people expect us to leave the labor force. Our activity patterns, such as travel, volunteer work and community service, also change, and, eventually, these patterns are often focused on survival and getting the care we need. All of this creates opportunity for business and changes in housing needs. Our paper focuses heavily on several areas of opportunity arising from an aging society. We will review these here.

Business needs to focus on the aging society from two vantage points—that of employer and that of marketer and producer of goods and services. As employers, one of the key questions will be: "Are people exiting the labor force at the right time, and if not, how do we change that?" Should we be offering new work options to help people move out gradually? This is already happening, but mostly as the result of individual initiative. For all businesses, the potential customer base is changing. In some cases, needs are different, and in others, preferences may simply change.

Housing is an area where there are clear and important differences. Provision of care is merging with housing for the elderly, and there is a range of options including, in some cases, no care, and, at the other extreme, full nursing care. We will explore issues related to housing, in order to provide an example of what we might think of:

- As people age and they become empty nesters, their housing needs change.
- People with hobbies need space to accommodate them at home or near their homes.
- Retirement communities that include activities are welcomed by some and shunned by others.
- Access to appropriate exercise is important.
- Preferences for location may change, particularly if access to public transportation, shopping and certain activities is important. In many cases, access to special transportation becomes important.
- Some people will choose to do more gardening as they age, and prefer large yards. However, others may find them a burden, and choose to move away from homes requiring a lot of upkeep and yard work. Snow shoveling is a problem for some.
- Steps become a problem for some people, and others need homes where they can use a wheelchair.
- Proximity to family is important.
- For people needing a small amount of care each day, it is much more efficient if they live in a place where the same caregivers can care for several people.

While many people will choose to live in some sort of retirement community, others will not. Many of them will still have issues related to their interests, capabilities and housing.

While we have illustrated housing, the aging society is also a major issue for other industries such as travel and health care. Many older persons, particularly those who are phasing out of the workforce or who have retired recently, travel extensively. They may have different interests than other audiences. Some travel, such as Elder Hostels, is particularly designed for them. They are a major part of the customer base for many cruises. In health care, older persons need more care and their needs are different from a younger population. They have more chronic illnesses, which require regular care.

Financial services are of particular interest to actuaries. There are opportunities as the population aging relates to the full range of post-retirement risks. More opportunities are also created, as employers are shifting more responsibility to individuals and away from employee benefit plans. The authors are particularly concerned that there is not a more general focus on outliving assets. This is a bigger issue for women than for men, as women live longer, and often live out their last years alone as widows or divorcées.

The future issues related to increasing life spans are heavily related to women. It should be of particular concern to us that we address them effectively since many of the population over age 85 are women living alone. Women living alone are much more likely to be in poverty than are married couples. In addition, if a person living alone needs assistance, there is no spouse to help, and often children are not available either. This group is the most likely to end up in a nursing home.

Families will also have an important role to play. They can serve as caregivers and financial providers In addition, they can help with decision making and chores, and act as support for family members. Many times elderly women who are alone have fewer resources than they otherwise would because when they were younger, they served in caregiving roles.

We do not know what the future of longevity will be, but we do know that our societies are aging, and the coming changes will affect all of us. Actuaries have a major role to play in helping us understand how change is unfolding and also in providing data to help our business and government institutions adapt to the changing society. Actuaries also have a role to play in supporting many of these institutions as they build financial security programs to meet the needs of an aging society.

Anna M. Rappaport can be reached at anna.rappaport@us.wmmercer.com.

Detection and significance of frailty in elderly insurance applicants

by Robert J. Pokorski, MD

Frailty may be described as a geriatric syndrome of advanced age that leaves a person vulnerable to falls, functional decline, morbidity and mortality.[1] Identification of people who are frail and at risk for functional decline is an essential part of geriatric assessment. It is also becoming a more important aspect of risk selection and classification for insurers that sell life and long-term care insurance to older applicants. This article reviews the geriatric literature to identify risk factors that could be used by insurers to identify existing or incipient frailty.

Frailty syndrome

Because of the absence of a generally accepted definition of frailty, geriatric specialists have chosen to describe the components of this syndrome. These descriptions have two underlying themes: (1) loss of functional reserve in multiple domains (areas), such as strength, balance, flexibility, reaction time, coordination, nutrition, cardiovascular endurance, vision and hearing, and cognitive performance, and (2) existence at a level that is close to or past the threshold for failure, with negligible tolerance of external stress.[2,3,4,5,6] Thus, a frail elderly person is someone with deficiencies in more than one functional domain with little or no reserve to cope with the routine stresses of day-to-day living. The result is that frail people are at high risk for inability to perform the instrumental activities of daily living (IADL) and the activities of daily living (ADL).

Frailty is not the same as disability.[1] Both frailty and disability are more common with advanced age, confer an increased risk of death, compromise function, and are associated with dependency. However, they differ in three respects. First, disability can arise from dysfunction of a single system or from many systems, whereas frailty always means multisystem dysfunction. Second, disability may be stable, whereas frailty is always unstable. In the context of frailty, "instability" means that small changes (e.g., minor illness or injury, low-grade physical or emotional stress) lead to disproportionately large effects (immobility, dependency, death). Third, frailty is present in a significant percentage of older people who are not disabled. These latter two points underlie the description of frailty as "subclinical" or "preclinical" disability, i.e., frail people may not be disabled, but they are at high risk for future disability.

Prevalence

Fried et al.[8] determined the prevalence of frailty in 5,317 community-dwelling people aged 65 years and older. Frailty was defined by the presence of three or more of the following criteria: unintentional weight loss, self-reported exhaustion, weakness, slow walking speed, and low physical activity. Frailty increased with age and was higher for women than men (Table 1).

Table 1. Prevalence of frailty (%) in community-dwelling subjects

Age	Subjects	Female	Male
65-70	2308	3.0	1.6
71-74	1271	6.7	2.9
75-79	1057	11.5	5.5
80-84	490	16.3	14.2
85-89	152	31.3	15.5
90+	39	12.5	36.8

Pathophysiology

Walston and Fried[1] proposed a triad of age-related changes that underlie the syndrome of frailty: sarcopenia (age-related decline in muscle mass); impaired regulation of the hypothalamus, pituitary gland, and adrenal glands; and dysfunction of the immune system. These abnormalities provide much of the explanation for why women are approximately twice as likely to develop frailty.

Predictors of frailty

Factors associated with morbidity and mortality

Table 2 lists many of the factors that have been associated with morbidity and mortality in the elderly. These factors are also common in frail elderly people.[5] Advanced age, functional decline, and comorbidity (multiple physical and/or cognitive disorders) are established predictors of deterioration. The geriatric syndromes are relatively recent additions to the list. This diverse group of disorders is strongly associated with decreased functional reserve and reduced life expectancy. With the exception of dementia, these syndromes are almost never listed on death certificates as the primary cause of death even though they often contribute to (e.g., depression, failure to thrive, osteoporosis, neglect and abuse) or directly cause (e.g., falls) death, and/or are markers of frailty that identify high risk people (e.g., delirium, incontinence, polypharmacy).

(See Table 2 on the next page)

Table 2. Factors associated with morbidity and mortality in the elderly

Advanced age
Functional decline
Instrumental activities of daily living
Activities of daily living
Comorbidity
Number of comorbid conditions
Severity of comorbid conditions
Geriatric syndromes
Delirium*
Dementia
Depression
Osteoporosis
Failure to thrive†
Falls
Incontinence
Neglect and abuse
Polypharmacy‡

* Delirium is transient cognitive impairment due to a medical condition unrelated to the central nervous system.

† Failure to thrive is a poorly understood syndrome characterized by weight loss despite adequate food intake.

‡ Polypharmacy means five or more medications.

Modified physical performance test

Brown et al. [4] used a modified version of the physical performance test (PPT) to provide an objective assessment of frailty in the elderly. The modified PPT includes the following: book lift; put on and take off a coat; pick up penny; chair rise; turn 360 degrees; 50-foot (15 m) walk; climb one flight of stairs; climb four flights of stairs; and balance tests. They found that scores on the modified PPT were significantly associated with laboratory measures of strength, balance, gait speed, range of motion, speed of movement, and sensation. Balance was most strongly associated with PPT score, a finding which agrees with other reports indicating that balance is a major determinant of frailty. Fast gait was also associated with PPT score.

Frailty index

Fried et al. [5] suggested a definition of frailty based on long term follow-up of 5,317 community-dwelling people aged 65 years and older (Table 3). Subjects with three or more criteria were considered frail, those with one or two criteria were hypothesized to be in an intermediate, possibly pre-frail stage, and people who met none of the criteria were considered not frail.

Table 3. Frailty criteria*

Criteria	Measurement
Unintentional weight loss of 10 pounds (4.5 kg) in past year	Single question
Self-reported exhaustion	Single question
Weakness	Hand-grip strength
Slow walking speed	Time to walk 15 feet (4.6 m) at usual pace
Low physical activity	Short questionnaire

* Three or more criteria = frail, one or two criteria = possibly pre-frail, no criteria = not frail.

At three- and seven-year follow-up, subjects defined as frail were far more likely to die, have a new hospitalization or fall, or experience worsening of ADL disability or mobility disability (Table 4). Individuals in the intermediate category experienced event rates that were between the frail and nonfrail subjects.

Noteworthy was the observation that some of the frail subjects had none of the major chronic diseases that are typically associated with frailty. The authors suggested that there might be two different pathways to frailty: (1) physiologic changes of aging that are not disease-based (sarcopenia, neuroendocrine dysregulation, immune dysfunction), and (2) a final common pathway of severe disease or comorbidity.

Self-reported function

Alexander et al. assessed the relationship between various self-reported physical functions and actual performance measures of walking ability, balance, and chair rise in 221 subjects (mean age, 80 years) in the U.S. Self-reported walking ability was the best single predictor of overall functional mobility, leading the authors to suggest that "self-reported walking ability may be the best indicator of ADL and mobility performance in community-dwelling older adults." A possible explanation is that decreased walking ability could be a marker of difficulty with other common tasks. Difficulty with mobility predicts future disability in tasks essential to living independently in the community (e.g., IADLs such as shopping and meal preparation) and to self-care (e.g., ADLs such as bathing and dressing).

Laboratory tests

Reuben et al. reported that the relative risk for mortality in healthy, nondisabled older persons was 2.2 times higher in subjects with a serum albumin level below the lower limits of normal. A study of 637 elderly hospitalized patients in Italy identified low serum cholesterol as a risk factor for frailty.

Exercise as a way to limit frailty

Physical capacity peaks in young adulthood and then declines at a rate which varies from one individual to another. Part of the physical decline is due to aging and is not amenable to intervention. Even healthy aging is associated with a striking loss of muscle mass and strength, with about half the muscle mass lost by age 80 years. The practical importance of this is

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Table 4. Incidence of adverse outcomes associated with frailty at 3 and 7 years after study entry (%)

	Death		New hospitalization		New fall		Worsening ADL disability		Worsening mobility disability	
Baseline status*	3 yr	7 yr	3 yr	7 yr	3 yr	7 yr	3 yr	7 yr	3 yr	7 yr
Not frail	3	12	33	79	15	27	8	23	23	41
Possibly pre-frail	7	23	43	83	19	33	20	41	40	58
Frail	18	43	59	96	28	41	39	63	51	71

*** At baseline, 2469 subjects were not frail, 2480 were possibly pre-frail, and 368 were frail.**

that an older person is often precariously close to the threshold at which a small decline in physical capacity (e.g., after a minor illness) will make it impossible to perform basic everyday activities, such as walking or rising from a chair.

Some age-related changes are due to disuse and not aging, and the lost fitness can be regained with regular physical activity, even in extreme old age.[13] Most of the health benefits of exercise can be gained by performing moderate intensity physical activities—equivalent to brisk walking at three to four miles (4.8 to 6.4 km) per hour—outside of formal exercise programs.[13] Strength training does not halt the underlying loss of muscle fibers, but the improvement in strength may be equivalent to 10 to 20 years of "rejuvenation" and may prevent an individual from falling below functionally important thresholds. Many other health benefits are associated with regular physical activity in old age. Weight-bearing exercise may slow the rate of bone loss in older women, balance training and tai chi may make falls less likely, and regular exercise may help in major depression. The social benefits of group exercise activities in later life should not be underestimated in a population where social isolation and loneliness may be common.

There is evidence that morbidity is being compressed into the final years of life and that healthy aging may be achievable, particularly for well educated, affluent older people. A landmark study from the University of Pennsylvania (U.S.) followed graduates from their early 40s to their mid-70s. The study focused on three potentially modifiable risk factors: cigarette smoking, body mass index, and exercise patterns. Subjects who had these risk factors (smoking, unfavorable body mass index, no regular exercise) in their mid-60s had both an earlier onset of disability and a greater level of cumulative disability, as well as more disability in the final year of life. In contrast, age at onset of disability was postponed by more than five years in the low risk group. In this study, adopting low risk habits in later life was associated with not only an increase in life span but also an increase in healthspan.[14]

Robert J. Pokorski, MD, is Vice President, Worldwide Medical Research & Development at GeneralCologne Re in Stamford, CT. He can be reached at **pokorski@gcr.com.**

Footnotes

1) Walston J, Fried LP. Frailty and the older man. Med Clin North America 1999;83:1173-94.

2) Hamerman D. Toward an understanding of frailty. Ann Intern Med 1999;130:945-50.

3) Brown M, Sinacore DR, Ehsani AA, et al. Low-intensity exercise as a modifier of physical frailty in older adults. Arch Phys Med Rehabil 2000;81:960-5.

4) Brown M, Sinacore DR, Binder, EF, et al. Physical and performance measures for the identification of mild to moderate frailty. J Gerontol A Biol Sci Med Sci 2000;55:M350-5.

5) Balducci L, Stanta G. Cancer in the frail patient: A coming epidemic. Hematol Oncol Clin North Am 2000;14:235-50.

6) Campbell AJ, Buchner DM. Unstable disability and fluctuations of frailty. Age and Ageing 1997;26:315-8.

7) Fried LP, Guralnik JM. Disability in older adults: Evidence regarding significance, etiology, and risk. J Am Geriatr Soc 1997;45:92-100.

8) Fried LP, Tangen CM, Walston J, et al. Frailty in older adults: Evidence for a phenotype. J Gerontol 2001;56A:M146-M156.

9) Alexander NB, Guire KE, Thelen DG, et al. Self-reported walking ability predicts functional mobility performance in frail older adults. J Am Geriatr Soc 2000;48:1408-13.

10) Yukawa M, McCormick WC. Cultural specific implications for decline in ADL and IADL. J AM Geriatr Soc 2000;48:1527-8.

11) Reuben DB, Ferrucci L, Wallace R, et al. The prognostic value of serum albumin in healthy older persons with low and high serum interleukin-6 (IL-6) levels. J Am Geriatr Soc 2000;48:1404-07.

12) Ranieri P, Rozzini R, Franzoni S, et al. Serum cholesterol levels as a measure of frailty in elderly patients. Exper Aging Res 1998;24:169-79.

13) McMurdo MET. A healthy old age: Realistic or futile goal? BMJ 2000;321:1149-51.

14) Vita AJ, Terry RB, Hubert HB, et al.Aging, health risks, and cumulative disability. N Engl J Med 1998; 338:1035-41.

Community service—a counterpoint

by Jay Novik

I reluctantly and respectfully disagree with Brad Smith's proposal published as a guest editorial in the April issue of *The Actuary*. I am reluctant to disagree because I fully support his goal of increasing voluntary commitments to community service in general, and to The Actuarial Foundation in particular. While I have not yet volunteered for active duty to the Foundation, I have contributed financially. In fact, I may be one of The Foundation's larger contributors.

Brad's proposal, however, is a case where the ends do not justify the means. Why are we selecting a small group of SOA members to bear a disproportionate burden? Why are we adding a new commitment to those who finally see daylight after years of exams? We are doing this because it is easier. It is easier than requiring all FSAs to commit the time. It is easier than increasing volunteerism and financial support by better selling the message of The Foundation to SOA members. Let's not take the easy way of shifting the burden to an already oppressed minority—new Fellows. While we believe that it will be good for them, we should be aware that 100 hours of community service could also be a sentence handed down for a misdemeanor. I think that we should increase the level of participation from younger members by example from the more senior members, not by sentencing them. In that spirit, I hereby sentence myself to 100 hours of community service. Who is with me?

Editor's note: Brad and Jay have an interesting point/counterpoint. Let's hear from the membership, especially recent Fellows.

Social Security and retirement options

In the March issue, Beverly Orth provides a brief summary of retirement option decisions for married couples in the U.S. Unfortunately, her description of current Social Security law is too brief —and inaccurate.

Ms. Orth correctly observes that the reduction factors applicable to Social Security retirement benefits first claimed between age 62 and normal retirement age (NRA) are roughly actuarial. (See the paper that Robert J. Myers and I coauthored on this topic in TSA, 1990.) Thus, Ms. Orth concludes that workers should claim their benefits at age 62 unless they plan to have "any significant earnings." This is bad advice, especially for one-earner couples, because the claiming of an early-retirement benefit limits future widow(er)'s benefits that can be paid on that worker's account.

Consider the case of a terminally ill married man who is age 62 and no longer working. Conventional wisdom might suggest that this worker should claim Social Security retirement benefits immediately, because he is unlikely to survive to his NRA and would not receive any benefits if he waits. This superficial analysis ignores the fact that his widow's benefit will be limited essentially to his benefit if he claims it but can be as large as his unreduced benefit if he dies without filing a claim. Thus, he might receive a few months of early-retirement benefits at the cost of reducing his widow's benefit for the rest of her life. Unless she earned more than he did in covered employment or also dies prematurely, this cost would be substantial.

Ms. Orth also recommends that workers who expect to have significant earnings between age 62 and NRA should postpone claiming benefits because of the retirement earnings test that still applies at those ages. This ignores the fact that automatic recomputations restore benefit reductions due to the earnings test when the worker reaches NRA. Thus, the decision is roughly neutral. I agree that workers should wait, but the penalty for not waiting is smaller than some might believe it to be.

Bruce D. Schobel

Beverly J. Orth responds:

I agree with Mr. Schobel that most couples should not begin their Social Security benefits before their Social Security retirement age (SSRA), despite the slight subsidy in the reduction factors. When I edited my paper to fit within the space limitations of "The Actuary," I left out two paragraphs where I presented a comparison of early versus late commencement of Social Security benefits. The complete paper, which appears on the SOA Web site, has the additional discussion, focusing particularly on the issue of increasing longevity as an important reason to avoid early commencement.

As my paper focused on married couples with two workers, I was primarily addressing the situation where both individuals have earned their own Social Security benefits and neither would be relying on the survivor's benefit. I agree with Mr. Schobel's analysis of the one-worker situation, where early commencement could severely impact the size of the survivor's benefit.

Finally, as Mr. Schobel points out, Social Security benefits are adjusted to restore benefit reductions due to the earnings test when the worker reaches SSRA. Accordingly, the benefit reductions alone may not be a sufficient reason for workers to postpone their Social Security benefits if they expect significant earnings before their SSRA. However, an additional factor to consider when making this decision is whether the earnings will cause the Social Security benefits to be taxed. The potential taxation of Social Security benefits could make postponement of benefit commencement more advantageous.

The quest for the theory of human longevity

by Leonid A. Gavrilov and Natalia S. Gavrilova

Actuaries and gerontologists (scientists who study aging) have some interests in common—members of both professions are interested in whether there are fundamental quantitative laws, which explain human survival up to extreme old ages. This common interest allowed them to share a foundation when they first met at the International Symposium "Living to 100 and Beyond: Survival at Advanced Ages" (January 17–18, 2002, Lake Buena Vista, Florida). This article summarizes our reflections on this interesting meeting and our presentations made there.

Attempts to develop a fundamental quantitative theory of aging, mortality, and life span have deep historical roots. In 1825, the British actuary Benjamin Gompertz discovered a law of mortality, known today as the Gompertz law. Specifically, he found that the force of mortality (known in modern science as mortality rate, hazard rate, or failure rate) increases in geometrical progression with the age of adult humans. According to the Gompertz law, human mortality rates double over about every eight years of adult age. Gompertz also proposed the first mathematical model to explain the exponential increase in mortality rate with age.

The Gompertz law of exponential increase in mortality rates with age is observed in many biological species, including humans, rats, mice, fruit flies, flour beetles, and human lice (see Gavrilov, L.A. & Gavrilova, N.S., *The Biology of Life Span: A Quantitative Approach*, NY: Harwood Academic Publisher, 1991), and, therefore, some general theoretical explanation for this phenomenon is required. Many attempts to provide such theoretical underpinnings for the Gompertz law have been made, and the problem now is to find out which of these theories is correct.

The current situation with applicability of the Gompertz law to extreme old ages is a paradoxical one. On the one hand, it has been well known for a long time that the Gompertz law is not applicable to mortality rates at advanced ages—the observed mortality rates are always lower than predicted by the Gompertz model, and, not surprisingly, the actual number of survivors to extreme ages is always higher than predicted by the Gompertz law. Figure 1 illustrates the mortality deceleration observed at advanced ages contrary to the predictions of the Gompertz law.

It is interesting to note that Gompertz (1825) himself found that at advanced ages mortality rates increase less rapidly than an exponential function, thus forestalling two centuries ago the recent fuss over "late-life mortality deceleration," "mortality leveling off," and "late-life mortality plateaus" (see review in Gavrilov L.A. and Gavrilova N.S., "The reliability theory of aging and longevity." *Journal of Theoretical Biology*, 213: 527-545).

Paradoxically, the Gompertz law and the Gompertz-Makeham law are nevertheless often applied to estimate the oldest-old mortality rates by extrapolation in order to "close" the life tables. When confronted with the question of why these "wrong" formulas are used, the demographers/actuaries usually reply that this is not an important issue, because life expectancy at birth is not very sensitive to how exactly

Figure 1

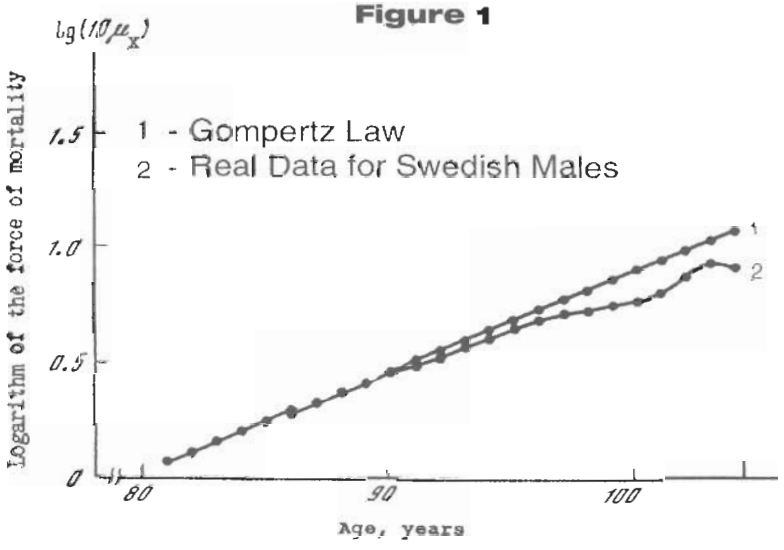

Figure 1. Mortality kinetics at advanced ages for Swedish males.

1 - theoretical dependence corresponding to exponential growth in the force of mortality with age (the Gompertz law with parameters $R = 3.46 \cdot 10^{-4}$ year^{-1} and $\alpha = 0.101$ year^{-1})

2 - real dependence for Swedish males. The statistical data for ages 81-85 are taken from the official Swedish 1956-60 life table, and data for ages 85-100 correspond to mortality in 1945-1967 estimated by the method of extinct generations (Depoid, 1973).

Adapted from: Gavrilov, L.A. & Gavrilova, N.S., *The Biology of Life Span: A Quantitative Approach*, NY: Harwood Academic Publisher, 1991.

the life tables are closed. The same "wrong" formulas and related assumptions are often used for graduation (smoothing procedures) of the mortality trajectories at advanced ages. It is extremely important, therefore, to know exactly how a particular life table was closed and/or graduated, before using it for testing of any statistical models. If the Gompertz or the Gompertz-Makeham laws were already introduced into the data by the method of life table construction/graduation, these data would not be useful for statistical hypothesis testing. Thus, the Gompertz law is known to be not applicable to the oldest-old mortality, unless the data are spoiled by artificial introduction of this law during extrapolation/graduation procedures.

The history of mortality studies at extreme ages is very rich in ideas and findings. In this article we would like to bring your attention to one seminal paper, which was published more than 60 years ago: Greenwood M., Irwin J.O. "Biostatistics of Senility," *Human Biology*, 1939, 11: 1-23. Interestingly, this article was considered to be so important that it was featured on the front page of the journal *Human Biology*.

This study, accomplished by the famous British statistician and epidemiologist, Major Greenwood, may be interesting to discuss again now for two reasons: (1) First, it is devoted to the studies of mortality at extreme ages. The authors of this paper admitted that the topic of their paper had "little actuarial importance" (in 1939), but may be of interest to biologists. However, now, 60 years later, this topic has great actuarial importance, as is evident from the topic of "Living to 100 and Beyond: Survival at Advanced Ages." (2) Second, this 1939 article correctly describes and forestalls the main specific regularities of mortality at advanced ages.

The first important finding was formulated by Greenwood and Irwin in the following way: "**...the increase of mortality rate with age advances at a slackening rate, that nearly all, perhaps all, methods of graduation of the type of Gompertz's formula overstate senile mortality**" (Greenwood, Irwin, 1939, p. 14). This observation is confirmed now and is known as the "late-life mortality deceleration."

The authors also suggested "**the possibility that with advancing age the rate of mortality asymptotes to a finite value**" (Greenwood, Irwin, 1939, p. 14). Their conclusion that mortality at exceptionally high ages follows a first order kinetics (also known as the law of radioactive decay) was confirmed later by other researchers, including A.C. Economos ("Kinetics of metazoan mortality," *J. Social Biol. Struct.* 1980, 3: 317-329), who demonstrated the correctness of this law for humans and laboratory animals. This observation is known now as the "mortality leveling-off" at advanced ages, and as the "late-life mortality plateau." Moreover, Greenwood and Irwin made the first estimates for the asymptotic value of human mortality (one-year probability of death, qx) at extreme ages using data from the life insurance company. According to their estimates, "**... the limiting values of qx are 0.439 for women and 0.544 for men**" (Greenwood and Irwin, 1939, p. 21). It is interesting that these first estimates are very close to estimates obtained later using more numerous and accurate human data including recent data on supercentenarians (those who survive to age 110).

Thus, the force of mortality practically ceases to increase at extreme old ages. The result is that the mortality kinetics of long-lived people is similar to the kinetics of radioactive decay, with a "half-life" corresponding to approximately one year (Gavrilov and Gavrilova, 1991). It is known from mathematics and physics that in this event there can be no absolute life span limit. This simple exponential law of survival at extreme ages allows us to estimate the chances of a centenarian (100 years) to become a supercentenarian (110 years), which is about $(0.5)^{10} = 0.001$. Thus, those countries that have more than 1,000 centenarians in their population, may expect the emergence of supercentenarians. If the numbers of centenarians are higher, persons with ages over 110 years could also be expected.

Greenwood and Irwin also proposed a possible explanation of very slow growth of mortality with age among centenarians. They suggested that very old people were less subjected to external stresses and shocks because they restricted their activities and rarely appeared in public (Greenwood and Irwin, 1939, p.14). Although this explanation could be challenged now, it still deserves some attention as a possible contributing factor to mortality deceleration at advanced ages. It is interesting that the authors also tried to analyze animal mortality at advanced ages and found the same regularities as in humans (Greenwood and Irwin, 1939, p. 21).

Further studies of mortality at advanced ages confirmed the major findings of Greenwood and Irwin (1939). The method of extinct generations proposed by P. Vincent ("La mortalité des vieillards," *Population* 1951, 6:181-204) and developed further by F. Depoid ("La mortalité des grands vieillards," *Population* 1973, 28: 755-792) and V. Kannisto ("On the survival of centenarians and the span of life." Population Studies 1988, 42: 389-406) opened new opportunities for more accurate mortality estimation at extreme ages. The dedicated research work by Väinö Kannisto (1916–2002), who collected a large body of data on mortality at advanced ages and finally created (together with Roger Thatcher), the Kannisto-Thatcher Oldest-Old Database, is of particular importance. More accurate data on human mortality at ages over 100 years allowed researchers to confirm earlier observations that human mortality at advanced ages is growing more slowly than is predicted by the Gompertz law.

The next important question is: why is the Gompertz law not applicable to the oldest-old mortality? As it was already noted, Greenwood and Irwin explained this phenomenon by taking into account

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the more protected environment of very old people. This explanation, however, is not applicable to laboratory animals demonstrating similar deceleration of mortality at advanced ages. A more general explanation of mortality deceleration phenomenon is that it may be the result of population heterogeneity at advanced ages. There is, however, one problem with testing this hypothesis. It may not be difficult to generate the mortality trajectories that will be close to the observed trajectories by assuming that population is a mixture of subgroups of people with different Gompertz parameters. The real problem here is whether there is any sense in such computational exercises. Is there any direct evidence indicating increased population heterogeneity at advanced ages?

The answer to this question is provided in Figure 2. The graph illustrates the dependence of the daughter's life span (expressed as additional years of life gained/lost compared to the reference life span level in the same birth cohort) as a function of maternal life span. For more methodological details of this study, see the original publication (Gavrilova N.S. and Gavrilov L.A. "When does human longevity start?: Demarcation of the boundaries for human longevity". *Journal of Anti-Aging Medicine*, 2001, 4: 115-124). Interestingly, the dependence of the daughter's life span on the maternal life span appears to consist of two lines—one for shorter-lived mothers (deceased prior to age 85) with a very weak dependence of the daughter's life span on the maternal life span, and another, for longer-lived mothers (deceased after age 85), with extremely strong and steep dependence.

Figure 2

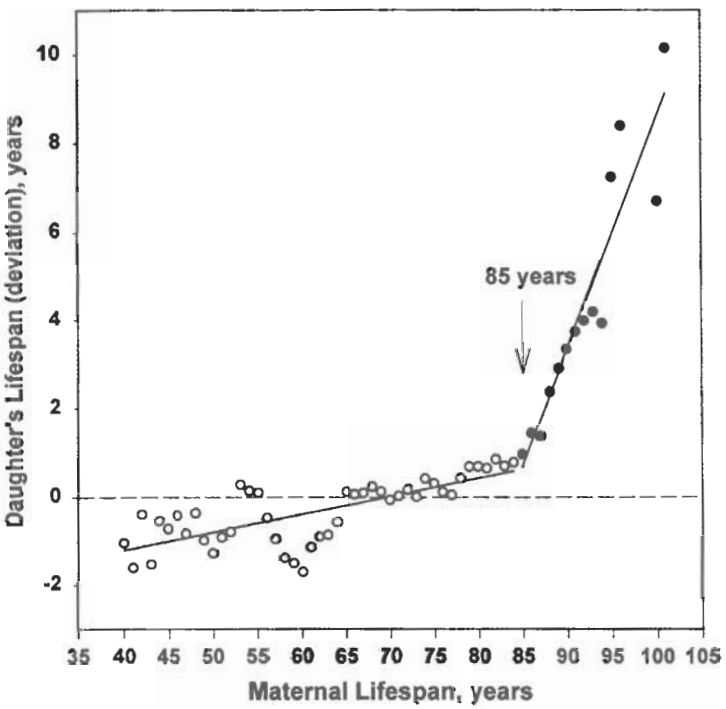

Figure 2. Daughter's life span (deviation from the cohort mean) as a function of *maternal* life span. Based on the data for 5,779 daughters from European aristocratic families born in 1800-1880 and survived by age 30. Data are smoothed by 5-year moving average.

Adapted from: Gavrilova N.S. and Gavrilov L.A. "When does human longevity start?: Demarcation of the boundaries for human longevity". *Journal of Anti-Aging Medicine*, 2001, 4: 115-124.

Daughters born to long-lived mothers may live 10 years longer, on average, if their mother reached age 100. This indicates that long-lived people are fundamentally different from other people in the sense that their children also live significantly longer lives. The breaking point at about age 85 for mothers indicates the age when death becomes much more selective in its timing and when population heterogeneity becomes an important issue. Thus, there is direct evidence for increased population heterogeneity at advanced ages, which may contribute to mortality deceleration in later life. This finding also has an actuarial significance, indicating that maternal life span is predictive for individual life span, if the mother lives beyond age 85.

Reliability theory of aging and mortality

The explanation of late-life mortality deceleration based on population heterogeneity is, however, completely untenable in the case of genetically uniform populations of laboratory animals, as well as technical devices, in which the same regularities are observed to hold as for human beings. Thus, we need to look for even more general explanations of the mortality kinetics at advanced ages. These explanations can be found in terms of reliability theory (see Gavrilov and Gavrilova, 1991; 2001).

Reliability theory is a general theory about systems failure. It allows researchers to predict the age-related failure kinetics for a system of given architecture (reliability structure) and given reliability of its components.

Reliability theory predicts that even those systems that are entirely composed of

non-aging elements (with a constant failure rate) will nevertheless deteriorate (fail more often) with age, if these systems are *redundant* in irreplaceable elements. Aging, therefore, is a direct consequence of systems redundancy. The "actuarial aging rate" (the relative rate of age-related mortality acceleration corresponding to parameter a in the Gompertz law) increases, according to reliability theory, with higher redundancy levels.

Reliability theory also predicts the late-life mortality deceleration with subsequent leveling-off, as well as the late-life mortality plateaus, as an inevitable consequence of *redundancy exhaustion* at extreme old ages. This is a very general prediction of reliability theory: it holds true for systems built of elements connected in parallel, for hierarchical systems of serial blocks with parallel elements, for highly interconnected networks of elements, and for systems with avalanche-like random failures (Gavrilov & Gavrilova, 1991). The reliability theory also predicts that the late-life mortality plateaus will be observed at any level of initial damage: for initially ideal systems, for highly redundant systems replete with defects, and for partially damaged redundant systems with an arbitrary number of initial defects. Furthermore, reliability theory predicts possible *paradoxical mortality decline in late life* (before eventual leveling-off to mortality plateau) if the system is redundant for *non-identical components* with different failure rates. Thus, in those cases when "apparent rejuvenation" is observed (mortality decline among the oldest-old) there is no need to blame data quality or to postulate initial population heterogeneity and "second breath" in centenarians. The late-life mortality decline is an inevitable consequence of *age-induced population heterogeneity* expected even among initially identical individuals, redundant in non-identical system components (Gavrilov & Gavrilova, 2001). Late-life mortality decline was observed in many studies and stimulated interesting debates because of the lack of reasonable explanation. Reliability theory predicts that the late-life mortality decline is an expected scenario of systems failure (Gavrilov & Gavrilova, 2001).

The reliability theory explains why mortality rates increase *exponentially* with age in many adult species (Gompertz law) by taking into account the *initial flaws (defects)* in newly formed systems. It also explains why organisms "prefer" to die according to the Gompertz law, while technical devices usually fail according to the Weibull (power) law. Moreover, the theory provides a sound strategy for handling those cases when the Gompertzian mortality law is not applicable. In this case, the second best choice would be the Weibull law, which is also fundamentally grounded in reliability theory. Theoretical conditions are specified when organisms die according to the Weibull law: organisms should be relatively free of initial flaws and defects. In those cases when none of these two mortality laws is appropriate, reliability theory offers more general failure law applicable to adult and extreme old ages. The Gompertz and the Weibull laws are just special cases of this unifying more general law (Gavrilov, Gavrilova, 2001).

The theory explains why relative differences in mortality rates of compared populations (within a given species) vanish with age, and mortality convergence is observed (known as the compensation law of mortality) due to the exhaustion of initial differences in redundancy levels.

The phenomena of mortality increase with age and the subsequent mortality leveling-off are theoretically predicted to be an inevitable feature of all reliability models that consider aging as a progressive accumulation of random damage (Gavrilov and Gavrilova, 1991; 2001). In short, if the destruction of an organism occurs, not in one, but in two or more, sequential random stages, this is sufficient for the phenomenon of aging (mortality increase) to appear and then to vanish at older ages. Each stage of destruction corresponds to one of the organism's vitally important structures being damaged. In the simplest organisms with unique, critical structures, this damage usually leads to their deaths. Therefore, defects in such organisms do not accumulate, and the organisms themselves do not age, they just die when damaged. In more complex organisms with many vital structures and significant redundancy, every occurrence of damage does not lead to death because of this redundancy. Defects do accumulate, therefore, giving rise to the phenomenon of aging (mortality increase). Thus, aging is a direct consequence (trade-off) of systems redundancy that ensures increased reliability and life span of organisms. As defects accumulate, the redundancy in the number of elements finally disappears. As a result of this redundancy exhaustion, the organism degenerates into a system with no redundancy, that is, a system with elements connected in series, with the result being that any new defect leads to death. In such a state, no further accumulation of damage can be achieved, and the mortality rate levels off.

Overall, reliability theory has an amazing predictive and explanatory power and requires only a few general and realistic assumptions. It offers a promising approach for developing a comprehensive theory of aging and longevity that integrates mathematical methods with biological knowledge, including evolutionary theory and systems repair principles. Reliability theory can be also useful in actuarial practice for predicting human survival up to extreme old ages.

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***Leonid Gavrilov, Ph.D. and Natalia Gavrilova, Ph.D. are Research Associates at the Center on Aging, NORC at the University of Chicago. They can be reached at* lagavril@midway.uchicago.edu.**

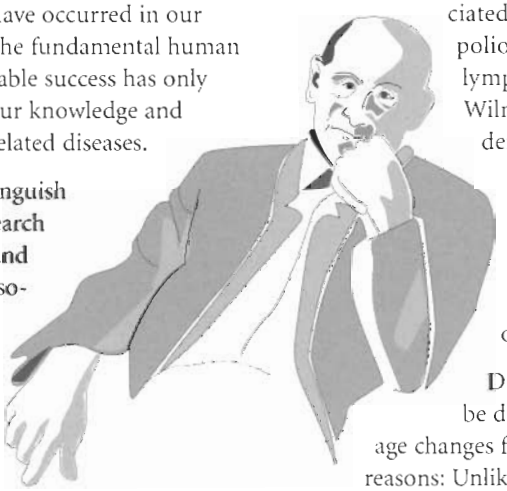
For more information, visit their scientific Web site:
"Unraveling the Secrets of Human Longevity"
http://www.src.uchicago.edu/~gavr1/.

Aging, longevity determination and disease

by Leonard Hayflick, Ph.D.

Introduction

Research on aging entered the mainstream of biological inquiry about 30 years ago but few notable advances have occurred in our understanding of the fundamental human aging process. Notable success has only been achieved in our knowledge and treatment of age-related diseases.



The failure to distinguish between aging research (biogerontology) and research on age-associated diseases (geriatric medicine) has been, and still is, the source of many misunderstandings that have led to questionable scientific, political, economic and societal decisions whose consequences have yet to be fully appreciated. There is little evidence that these misunderstandings, with their serious consequences, will soon be rectified. Thus, the present imbalance will continue in which resources available for research on the diseases of old age far exceed those available to increase our understanding of the more fundamental question: Why are old cells more vulnerable to disease than are young cells?

Disease and aging

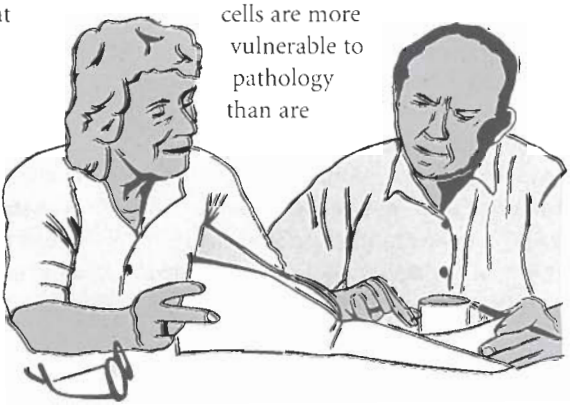
Because aging is not a disease, the resolution of the leading causes of death in old age—cardiovascular disease, stroke, and cancer—will tell us little about the fundamental biology of age changes. The resolution of all three causes will result only in an increase of about fifteen years in human life expectation (Anderson, 1999). Then, aging, or the inexorable loss in physiological capacity that underlies the cause of these pathologies, will be revealed as the leading cause of death. Resolution of age-associated diseases will advance our knowledge of aging processes to the same extent that the resolution of pediatric-associated diseases, such as poliomyelitis, acute lymphocytic leukemia, Wilms' tumors and iron deficiency anemia, advanced our knowledge of childhood development. That is, no advancement occurred at all.

Disease processes can be distinguished from age changes for at least five reasons: Unlike any disease, age changes occur in every animal that reaches a fixed size in adulthood. Unlike any disease, age changes cross virtually all species barriers. Unlike any disease, age changes occur in all members of a species only after the age of reproductive success. Unlike any disease, aging occurs in animals removed from the wild and protected by humans even when that species has not experienced aging for thousands or even millions of years. Finally, unlike any disease, aging occurs in both animate and inanimate objects.

Today, the study of age-associated diseases and manipulating biological development in lower life forms dominates what many in the scientific community consider to be the field of aging research. It is not. One example is that more than one-half of the budget of the National Institute on Aging in the United States is spent on Alzheimer's disease research, yet motor vehicle accidents cause twice as many deaths (Adelman, 1998; Anderson, 1999a) and from age 65 on, it is not even one of the five leading causes of death (Hobbs and Damon, 1996). The likelihood of dying from Alzheimer's disease is 0.7% (Anderson, 1999) and the complete resolution of this disease will add about 19 days onto average life expectation (Anderson, 1999). Nor will that enormous accomplishment advance our knowledge of the fundamental biology of aging.

In the minds of the public, policy makers and many biomedical scientists, no one suffers or dies from aging. We suffer and die from the diseases associated with the aging process. Yet, the aging process is the underlying cause of the increase in vulnerability to everything that is written on the death certificates of the elderly.

A good case can be made for the notion that no one over the age of, say 75, has, or will die from what is written on his or her death certificate. Death results from the inevitable increase in systemic molecular disorder that living long enough incurs. That fundamental or underlying disorder simply increases vulnerability to whatever was, or will be, written on death certificates. There is an almost universal belief that the greatest risk factor for the three leading causes of death is the aging process, yet that risk factor receives only a microscopic portion of the biomedical research budget. If we are to make any progress in understanding why old cells are more vulnerable to pathology than are



young cells, then our priorities must be reordered.

The hallmark of extreme old age is the presence of multiple pathologies making the determination of the cause of death difficult. Because autopsies of old people have become increasingly rare, the cause of most deaths in old age is still hidden in the proverbial black box. The numbers of autopsies that have been done on the elderly have continued to decrease in the last few decades. In those rare instances where autopsies have been done on a large number of elderly people, the findings have shown that from 40 – 50% of the causes of death cited on the death certificates have been inaccurate (Kohn, 1982; Paterson et al., 1992; Gee, 1993). In the most recent study of 93 postmortem examinations done in an Israeli hospital over a 20-year period, 42% of the causes of death written on the death certificates were incorrect (Leibovitz, et al, 2001). Over this 20-year period the rate of autopsies dropped from 2.8% to 0.25%. These findings should raise considerable concern for the many political, economic, actuarial and scientific decisions that, without benefit of autopsy, may have a 40-percent error rate.

The hallmark of extreme old age is the presence of multiple pathologies making the determination of the cause of death difficult.

Aging and longevity determination

If aging research is to advance, it will not only be necessary to distinguish biogerontology from geriatric medicine, but it will also be necessary to distinguish aging from longevity determination. Failure to do so often results in research interpreted to bear on aging when, in fact, the results impact on our knowledge of longevity determination.

Aging is a stochastic process that occurs after reproductive maturation and results from increasing systemic molecular disorder. This disorder has multiple etiologies, including damage by reactive oxygen species, but generally from the diminishing reduction in energy states necessary to maintain molecular fidelity.

Longevity determination, on the other hand, is not a random process. It is governed by the excess physiological capacity reached at the time of sexual maturation that, through natural selection, was achieved to better guarantee survival. Thus, longevity is only indirectly determined by the genome.

Species survival depends on a sufficient number of members living long enough to reproduce and, if necessary, to raise progeny to independence. Natural selection favors animals that have greater survival skills and, especially, redundant physiological reserve in vital organs beyond the minimum needed to survive the damage that might be exacted by predators, disease, accidents or environmental extremes.

Physiological capacity, beyond the minimum required for survival, increases the chances for animals to live long enough to achieve reproductive success, just as redundant capacity in complex machines better insures that they will achieve their goals. The amount of excess physiological capacity, like the amount of redundancy engineered into space vehicles, provides the potential for continued function beyond the primary goal (Hayflick, 1996; 1998).

For the survival of any species, energy is better spent on guarantying reproductive success than it is for increasing individual longevity. Because living long beyond reproductive success has diminishing value for the survival of a species, weakened members will be culled by natural selection. The molecular order achieved from conception to sexual maturation becomes increasingly disordered after reproductive success. Systemic molecular disorder, or aging, increases in spite of the presence of repair processes because these too incur disorder. In this way the acceleration of molecular disorder, or aging, increases vulnerability to predation, accidents and disease.

The developmental events that lead to the survival of animals to reproductive success are determined genetically, but the survival of animals beyond sexual maturation is determined only indirectly by the genome. It is for this, and other reasons, that biogerontologists may be asking the wrong question: "Why do we age?" The right question could be: "Why do we live as long as we do?"

Genes do not govern aging

Aging is not a programmed process governed directly by genes. Studies in lower animals that have led to the view that genes are involved in aging have not shown a reversal or arrest of the inexorable expression of molecular disorder that is the hallmark of aging. Those studies are more accurately interpreted to have impact on longevity determination because the observed effects occur before the aging process begins and alter physiological capacity.

Another argument against the direct role of genes in programming the aging

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process is that animals do not age at the same rate nor are the patterns of age changes identical. This results in the variations found in age of death. When the random events characteristic of aging are compared with the orderly, virtually lockstep, changes that occur during genetically driven embryogenesis and development, the orderliness and precision stands out in stark contrast to the quantitative and qualitative disorder of age changes. The variability in the manifestations of aging differs greatly from animal to animal, but the variability in developmental changes differs trivially. Humans from conception to adulthood are virtually identical in respect to the stages and timing of biological development but from about 30 years of age on, age changes make humans more heterogeneous.

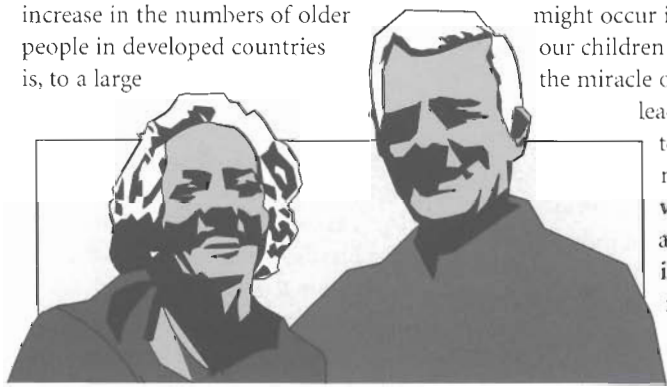
Just as a blueprint is vital to manufacture a complex machine and contains no information to cause the aging of that machine, the genome is necessary for biological development but unnecessary to cause the animal's aging. The animal and the machine fail as a result of increasingly irreparable loss of molecular fidelity, which in living systems increases vulnerability to predation, accidents or disease and in inanimate objects increases vulnerability to analogous failures in some vital component.

Longevity determination in higher animals has been a profoundly neglected area of research. One class of animals that may provide some answers to the determination of longevity are those animals that do not reach a fixed size in adulthood and age slowly or not at all. If these animals do age, the process is either negligible or it occurs below the limits of detection. Animals of this class include some tortoises, many sport and cold-water deep-sea fish, some amphibians and the American lobster. Even telomerase expression, the hallmark of immortal cells, has been found at extraordinary high levels in the cells of negligibly aging animals like the American lobster and the rainbow trout. The question of whether these animals age at all, and the reasons for this, has been almost entirely neglected. They are not immortal, because, like animals that do age, there is a constant threat of disease, predation and accidents (Hayflick, 2000a).

The aging of living things is not unlike the aging of everything in the universe, including the universe itself. Although biological aging occurs in an open system, the Second Law of Thermodynamics applies in that entropy increases despite the constant availability of energy in the form of food. Entropy increases in biological systems because natural selection has not favored systems that can maintain molecular fidelity indefinitely. Energy is better spent on strategies that insure reproductive success in order to perpetuate the species rather than spending it on post-reproductive longevity that has little species survival value. The verity of this statement can be found in the observation that, for feral animals that reach a fixed size in adulthood, death occurs from predation, accident or disease shortly after the period of reproductive success necessary for species survival. Then, as physiological capacity begins to wane, natural selection culls the weakened animals.

Aging as an artifact of civilization

Aging is a phenomenon unique to the human species because it is a consequence of our advancing knowledge of hygiene and biomedicine. The resulting increase in the numbers of older people in developed countries is, to a large extent, an unintended consequence of these advances and an artifact of human civilization (Hayflick, 1996, 1998, 2000b).



Humans, and the animals we chose to protect, are the only species in which large numbers experience aging. Furthermore, old humans, or old animals, are not essential for the survival of any species. The evidence for this is that humans have had a life expectation at birth of 30 years or less for more than 99.9% of the time that we have inhabited this planet. Prehistoric human remains have never revealed individuals older than about 50 years of age. There appears to be no selective advantage favoring the survival of old animals or old humans.

Members of exotic feral animal species, who for millions of years have not experienced aging, reveal those changes when protected by humans as pets or in zoos. It would be difficult to explain how evolution could have selected for a process like aging that could be made to appear in all members of a species after, perhaps, millions of years of suppression.

Because modern humans, unlike feral animals, have learned how to escape death long after reproductive success, we have revealed a process that, teleologically, was never intended for us to experience. Again, one might properly conclude that aging is an artifact of civilization.

Future possibilities

There is no evidence to support the many outrageous claims of extraordinary increase in human life expectation that might occur in our lifetime or that of our children or their children. Even if the miracle of eliminating the three leading causes of death were to occur tomorrow, a maximum of 15 years would be gained in average life expectation. Any increase beyond that number would depend on slowing or stopping the fundamental processes

that produce molecular disorder. The likelihood that that can be done is remote, if not impossible. It is remote because, like inanimate objects such as automobiles, the aging process is inevitable and it is impossible to reverse or stop, because even if all parts could be replaced, the result would not be the same automobile or the same human.

One must eliminate almost all mortality risks from 1995 levels before age 85 to achieve a life expectation greater than 100 (Olshansky, et al., 2001). The 1995 death rates would have to decline by more than 50% at every age in order for life expectancy to reach 85 years in the United States (Olshansky, et al., 2001). Even among Japanese women who are the longest lived sub-group in the world, total mortality at every age would have to drop 20% in order to raise life expectancy by two years from its current 83 years. The mortality reductions at every age required to achieve a one-year increase in life expectancy at birth today are more than twice those needed to achieve the same gain early in the 20th century (Olshansky, et al., 2001). It is not possible to reach life expectations of 100 or more unless the method will not only completely eliminate all causes of death currently appearing on death certificates, but will include an intervention to slow or stop the fundamental aging process.

Thus, the approximate 25-year increase in life expectancy that occurred in the United States from 1900 to 2000 will be impossible to achieve in the 21st century and well beyond.

However, it is likely that a natural increase in the human life span is presently occurring, but so slowly that our ability to detect it will only be made after millennia of careful record keeping. This belief is based on persuasive evidence in the fossil record that suggests that the life spans of most animals increase as evolution proceeds (Hayflick, 1996).

As some civilizations have, our society must learn that aging and youth should be valued equally if for no other reason than that the youth in developed countries have an excellent chance of experiencing the phenomenon that they may now hold in low esteem. Then, the misplaced passion for cosmetic surgery, anti-aging nostrums and similar snake oil remedies touted to arrest aging will be recognized for what they truly are—at best, a cover-up for an irreversible and inexorable process and, at worst, a delusion and waste of money by the uninformed.

If the main goal of our biomedical research enterprises is to resolve causes of death, then every old person becomes a testimony to those successes.

Biogerontologists have an obligation to emphasize that the goal of research on aging is not to increase human longevity regardless of the consequences but to increase active longevity free from functional disability and dependence.

REFERENCES

1. Adelman, R., The Alzheimerization of Aging: A Brief Update, Exp. Gerontol. 33, 155-157, 1998.

2. Anderson, R.N. U.S. decennial life tables for 1989-91, Vol. 1, No. 4, Pages 6-8, United States life tables eliminating certain causes of death. National Center for Health Statistics, Hyattsville, Maryland, (1999a).

3. Gee, W.M., Causes of death in a hospitalized geriatric population: an autopsy study of 3000. Virchows Arch. 423, 343-349, 1993.

4. Hayflick L: How and Why We Age, (Ballantine Books, New York City, N.Y., 1996).

5. Hayflick L. How and Why We Age. Exp. Gerontol. 33:639-653 (1998).

6. Hayflick, L. The Illusion of Cell Immortality. Brit. J. Cancer, 83, 841-846 (2000a).

7. Hobbs, F.B. & Damon, B.L. 65+ in the United States. U.S. Bureau of the Census, Current Population Reports, Special Studies, P23-190, Page 3-9, U.S. Government Printing Office, Washington, D.C. (1996).

8. Kohn, R.R., Cause of death in very old people, JAMA, 247: 2793-2797,1982.

9.Leibovitz, A., Blumenfeld, O., Baumoehl, Y., Segal, R. Habot, B., Aging Clin. Exp. Res. 13, 406-409, 2001.

10. Olshansky, S.J., Carnes, B.A., Désesquelles, A. 2001. Prospects for Human Longevity. Science 291 (5508):1491-1492.

11. Patterson, D.A., Dorovitch, M.I., Farquhar, D.L., Cameron, H.M., Currie,C.T.,Smith, R.G., MacLennan, W.J., Prospective study of necropsy audit of geriatric inpatient deaths. J. Clin. Pathol., 45, 575-578, 1992.

***Leonard Hayflick, Ph.D, is a Professor in the Department of Anatomy at the University of California, San Francisco, School of Medicine. He can be reached at* len@gene.com.**

ELECTION REMINDER!!

The Board of Governors' second ballot elections and the section council elections will be done electronically. You will receive your password to vote by having a valid e-mail address on the SOA database. To update your membership information, use the *Directory of Actuarial Memberships* on the SOA Web site (*http://www.soa.org*). If you would prefer to receive paper ballots, you must contact Lois Chinnock at the SOA office (847/706-3524 or *vote@soa.org*) **no later than Friday, May 24.** Please indicate whether you are a Fellow or an Associate of the Society of Actuaries.

Joint research, education efforts benefit actuarial profession and other organizations

by Anna M. Rappaport

The issues actuaries deal with are complex. We are often trying to help make financial security systems work better. We are dealing with changing demographics, a fluid economy, and changes in health care systems.

Given the complexity of these changes and the challenges that surround them, the best solutions can often be found by working in multi-disciplinary teams. Most of us participate in such teams in our daily work. Likewise, the SOA often participates in joint activities with other professional organizations. This article focuses on some of the SOA relationships and activities that are undertaken jointly with others. It should be noted that while the SOA has important relationships all over the world, this article focuses on SOA relationships in North America. For more than five years, the SOA has worked hard to build these relationships and has proactively sought out areas where we could do a better job of collaborating with others. These efforts aptly mirror the theme of *Actuaries Making a Difference.*

The SOA's cooperative ventures are built around research and education, designed to serve our membership and the public. Let's start with two examples from major annual conferences. The Annuity Conference is sponsored jointly with LIMRA and LOMA, and brings together actuaries, marketers, administrative professionals and others to discuss new developments and trends in the sale and management of annuity products. This area is particularly important as employee benefit plans are shifting more risk to individuals. Outliving assets is a key risk. Each spring the Long Term Care Insurance Section, together with several outside organizations, sponsors an annual Long Term Care Insurance Conference. This conference brings together actuaries with underwriters, claim specialists, and marketers for mutual problem solving around this important and growing product line. As with the Annuity Conference, it is sponsored by multiple organizations. Both of these product lines are important in addressing issues surrounding post-retirement risk, and in achieving results that make a difference to all Americans.

Actuaries regularly stay in contact with other professionals in different groups. SOA members in the National Academy of Social Insurance (NASI) have worked together to increase the participation of actuaries in NASI. Not only was the SOA able to sponsor a a table at the NASI annual meeting, but we were particularly pleased to give a preliminary presentation of the post-retirement risk research discussed below at that meeting (see picture at left). NASI is a leader in

Seated: *Joe Applebaum, GAO, Anna Rappaport, Laurel Beedon, AARP (and member of the Planning Committee for RIDFC), Leslie Kramerich, formerly head of the Labor Department's PWBA*

Standing: *Stephanie Poe, William M. Mercer, Incorporated, Cindy Hounsell, Executive Director of WISER, Matt Greenwald, Mathew Greenwald & Associates, Mavis Walters, FCAS and Chair of the Actuarial Foundation Board, Dick Schreitmueller, Chair of the Task Force which wrote the background paper for the WISER booklet, and Steve Kellison, formerly a public trustee of Social Security*

addressing social insurance issues using fact-based research and in a forum where diverse viewpoints and disciplines are represented. NASI offers the opportunity to do research and discuss social insurance policy matters in a multi-disciplinary forum where care is taken to include all points of view. NASI has also cosponsored events with the SOA.

Another important multi-disciplinary research group is the Pension Research Council (PRC), which is connected to the Wharton School at the University of Pennsylvania. The PRC has also been a cosponsor of SOA events and several actuaries participate actively in the work of the Council.

The SOA also has collaborated with groups whose primary focus is research and/or education for the employer market, and the benefit plan sponsor community. WorldatWork worked with us on a session for the 2001 Annual Meeting, and they sponsored a prize for the Retirement Implications of Demographic and Family Change Call for Papers. The International Foundation of Employee Benefit Plans has been a joint sponsor of multiple events. The Employee Benefit Research Institute (EBRI) has also joined with the SOA in sponsoring some events, as well as assisting in the recent Retirement Risk Research.

In the last few years, the SOA's Retirement Systems Practice Area has sponsored several calls for papers on important topics. These have been followed by multi-disciplinary symposia with authors from both inside and outside of the actuarial profession. This series started with the recognition that there was inadequate focus on post-retirement risk in our retirement systems; most of the focus had been on building up assets, not on how they were used. The recognition that post-retirement risk was not adequately explored led to the Retirement Needs Framework Symposium, and then to Retirement 2000. The first set of papers focused on data and modeling, while the next dealt with policy. The Social Insurance Committee expressed concern that while policy focus is on overall system structure, there are some issues that are not being addressed. For example, women do not fare as well in retirement, and while people are leaving the labor force in steps, the formal retirement systems have not adjusted. These concerns led to the next call for papers and symposium, which will be on Retirement Implications of Demographic and Family Change. There are 16 cooperating organizations working with the SOA on this symposium. The organizations were listed and information about the symposium was included in the March issue of *The Actuary*. Previously, a symposium, Retirement 2000, was held on retirement policy. By working together with other organizations, we garner a wider range of paper authors and attendees, which results in a greater variety of points of view. This makes for a better discussion, and helps actuaries to understand other points of view. It also encourages other professionals to include actuaries in their teams, and to think about actuarial points of view.

Actuaries also work jointly with outside groups to provide public service. Most of these activities are channeled through The Actuarial Foundation, which sponsors programs that help young Americans better learn math, as well as providing adults with education related to financial security. The consumer education programs are viable due to the research and learning that results from the work of the Retirement Systems Practice Area research. The Consumer Education Committee has worked on a project with WISER, the Women's Institute for a Secure Retirement, and it is now working with the Profit Sharing Council of America on another project. In both cases, the information is designed to be available to individuals, either through the work site or on a stand-alone basis.

Survey research offers another opportunity for joint projects. During 2001, the SOA engaged Mathew Greenwald & Associates with support from EBRI to do a research project on public attitudes to post-retirement risk. This research provides another important link in understanding the gaps in post-retirement coverage and working to solve them. By working cooperatively with the American Academy of Actuaries, we were able to present the research to a large group of Congressional staffers on February 25, 2002. The importance of this research was evidenced by its coverage on the cable network C-Span. This research will also probably influence some of our public education and service.

These are some examples of projects in which the actuarial profession is working jointly with others. The American Academy of Actuaries also has many relationships, with a major focus on policy. The SOA and AAA are working together to leverage our effectiveness in partnerships. I see joint projects as vitally important for the future of the profession and hope to see more such projects in the future.

Anna M. Rappaport can be reached at **anna.rappaport@us.wmmercer.com.**

Practice Area update

This update is meant to inform our members of the important projects going on within the various SOA Practice Area committees. This work is often done in conjunction and coordination with our various Sections. We hope to continue to improve communication regarding the efforts SOA is making to support the practitioner in our various practices areas in the future.

A primary effort being made is a review of SOA committee and Section structure. There are currently four different Practice Areas that operate within SOA: Retirement Systems, Health Benefit Systems, Life Insurance, and Finance. Volunteers and a full-time SOA staff actuary manage each Practice Area. With oversight by a Practice Advancement committee, each Practice Area exists to ensure that the relevant research and educational needs (both preliminary and continuing education) of the practitioners in the particular area of practice are met. While much of this work is done by different Sections of SOA, the Practice Areas exist to make sure that all the work needed by our members is getting accomplished.

As part of the strategic plan adopted last year, the Board of Governors started to examine the work that was being done within the Practice Area committees, and specifically asked for a recommendation for how the Practice Area committees and the various SOA Sections could work better and more effectively together. They put together a task force to examine this (the Task Force on Sections and Practice Areas, as mentioned on the Web site http://www.soa.org/committees/spa.html), and recommend improvements so as to better utilize the volunteer and staff resources available to advance the initiatives of our membership at large.

This Task Force has now been working for nine months and is getting closer to a recommendation for Board consideration. Further updates on this Task Force's work will be published in future editions of *The Actuary*.

Practice Area Update: A brief update from each area is presented here.

Retirement Systems Practice Area (http://www.soa.org/committees/retire.html): Much of the work of the Retirement Systems Practice Area is coordinated with the Pension Section. Please check out their combined web site at http://www.soa.org/sections/pension.html . There are several research initiatives beginning in the Retirement Systems Practice Area, including a study of the factors affecting retirement mortality and a new turnover study. The turnover study is expected to produce a series of tables reflecting different levels of turnover. In addition, coordination with the American Academy of Actuaries (AAA) continues for addressing a long-range solution for the issues related to 30-year Treasury rates. Several continuing education initiatives are being explored, particularly web-based and on-line education programs. And, finally, the Retirement Systems Practice Area is devoting considerable resources to assist the new working groups that have been put together to review our preliminary and actuarial education process. The practice area advisory groups will be working to ensure that the content of our examination syllabus has the relevant content material necessary for practitioners in the Retirement Systems field.

Health Benefit Systems Practice Area (http://www.soa.org/committees/health.html): The Health Benefit Systems Practice Area initiated a project late last year to provide for some of the current needs in terms of modeling, assumption development, or perspectives on the current health care reform debate in the United States. The project is currently in an initial phase of a review of current literature and research on several subtopics related to the troubled U.S. healthcare system. The Health Benefit Systems Practice Area and the Health Insurance Association of America are also cooperating on the development of a consumer education piece on disability risk and the public and private sources of coverage available to mitigate those risks. This consumer education project, through the development of a disability income "Chartbook," has been undertaken to help address the lack of public awareness of the risks associated with disability. Finally, the Health Benefit Systems Practice Area is also actively engaged in providing input into discussions regarding potential future changes to the SOA's Education and Examination System, so that the needs of health actuaries and their current and future employers can be appropriately considered. The strategic planning process and related projects of the Health Benefit Systems Practice Area were also described in more detail in the October 2001 edition of *The Actuary*.

Life Insurance Practice Area (http://www.soa.org/committees/life.html): The many subcommittees on Research, Education and Professionalism do the work accomplished within the Life Insurance Practice Area. The new CSO table work was completed within the framework of the research committees of the Life Insurance Practice Area. Those committees continue, working with the AAA, to identify items that need to be addressed as a result of this new table. A life insurance practitioner's working group is being put together to help review our exam and education syllabus for appropriateness of content for life insurance practitioners. And, finally, the Practice Advancement Committee is just now being revitalized to start to identify the important practice needs of our actuaries that will direct the SOA Life Insurance Practice Area's activities for the upcoming year.

Finance Practice Area (http://www.soa.org/committees/fin.html): The Finance Practice Area consists of numerous committees and task forces and is

headed by the Finance Practice Area Advancement Committee. The goals of the Practice Area are to further the actuarial profession through identification and development of finance-and-investment-related tools for the practitioners in the industry, contribute to the future of the profession by investing in the cutting-edge research initiatives, and find new and exciting applications for the actuarial involvement in the non-traditional arena of the broader financial industry. The Committee on Finance Research is currently putting together a survey of CFT software models with the goal of producing a report that will serve as a reference for comparing the available software packages. In addition, it is hoped that production of such a report will, in turn, lead to better software and modeling techniques. Another major ongoing initiative in the Finance Practice Area is the Risk Management Task Force. This task force, and the numerous sub-task forces, were discussed in the April 2002 edition of *The Actuary.*

research corner

SOA PROJECT & EXPERIENCE STUDIES UPDATE

Life Insurance

• "Why Men Die Younger: Causes of Mortality Differences by Sex" by Barbara Blatt Kalben is now available in monograph form (M-LI01-1). This well-documented paper attempts to synthesize the evidence supporting and refuting the hypotheses for the sex mortality differential. To obtain an order form, go to the SOA Web site (*http://www.soa.org/research/why_orderform.html*), or place a telephone order by calling Beverly Haynes (847/706-3526) in the Books Department.

• A subcommittee of the Society of Actuaries' Committee on Life Insurance Mortality and Underwriting Surveys has completed its Regulation XXX Survey Report and it is now available on the SOA Web site (*www.soa.org*) under Research. This survey examined the early responses, including pricing methodology changes, to the Regulation XXX term reserving requirements.

The Society of Actuaries' Individual Life Insurance Valuation Mortality Task Force has prepared a final report on the 2001 CSO Basic Tables and it is on the SOA Web site (*www.soa.org*) under Research.

Retirement Systems

• The Society of Actuaries' Group Annuity Experience Committee has completed their 1997–98 Report, which presents the 1997 and 1998 calendar year experience of retired individuals in the United States who are covered under group pension contracts. This report can be found on the SOA Web site (*www.soa.org*) under Research.

AERF ACTIVITY

Outstanding papers at ICA

AERF announced James C. H. Anderson Memorial Awards for outstanding papers submitted for presentation at the 27th International Congress of Actuaries meeting held in Cancun, Mexico from March 18–22, 2002.

A First Prize of $10,000 (U.S.) was awarded to Victor Manuel Jimenez Escobar (Mexico) for his paper "Testing the Stability of the Components Explaining Changes of the Yield Curve in Mexico: A Principal Component Analysis Approach."

Two Second Prizes of $5,000 (U.S.) each were awarded. One was presented to Andrew Wise, Andrew Barnes and Andrew Reid (United Kingdom) for their paper "Risk Sharing in Employer Pension Provision," and the other was awarded to Javier Guitierrez Garcia and Jesus Alan Elizondo Flores (Mexico) for their paper "Credit Risk: The Actuarial Value."

The papers are posted on the ICA Web site at *www.ica2002.com.*

Anderson Memorial Fellowship

The Actuarial Education and Research Fund awarded a second Anderson Memorial Fellowship to Edoh Afambo, a Ph.D. candidate in the Department of Risk Management and Insurance at Georgia State University. The $12,000 fellowship is in recognition of Afambo's past achievements and future ambitions.

Mr. Afambo, a West African actuary, is focusing his research at Georgia State on building financial and insurance models, taking into account the economic and social context of Africa. Following the completion of his Ph.D., Mr. Afambo plans to return to Africa to resume his consulting practice and address the problems facing the financial services industry in West African countries.

Wooddy Scholarship reminder

The AERF will award up to four $2,000 Wooddy Scholarships to undergraduate students who will have senior standing during the 2002–2003 academic year. The deadline for applications is Friday, June 28. Applications are available on the AERF Web site at *www.aerf.org/grants&competitions.html* or from Judy Yore, AERF Business Manager (phone: 847-706-3573, e-mail: *jyore@soa.org*).

New Associates March 2002

It is a pleasure to announce that the following 103 candidates have completed the educational requirements for Associateship in the Society of Actuaries as a result of attendance at the Associateship Professionalism Courses held in March 2002.

Allard, Gilbert
Amar, Madhu Gupta
Atwater, Eric J.
Audi, Nabila
Barker, David Alfred
Bellanca, Joseph Paul
Betzag, Todd Jason
Bi, Hai
Blichar, Roman George
Burkhardt, Caroline Emmanuel
Butterfield, Robert K. Jr.
Campbell, Sekayi Ayanna
Cao, Yue
Castagnoli, Guy Eugene
Chatterjee, Sharmila
Chen, Weihua
Cheng, Chen-Te
Chng, Tze Ping
Christopher, George E.
Daniels, Ryan
Davis, Alan Frederick
Dube, Etienne
Embers, Dale G.
Eom, Seong-Min
Erhart, Xavier J.
Falikson, Aleksandr
Fei, Huafeng
Fischer, Jason Robert
Freedman, Barry
Gary, Eric Halladay
Gengenbach, Mark Bruce
Giese, Amy Rose
Gould, Ryan Edward
Guo, Yan
Hall, Elizabeth Skiba
He, Sasa
Hilton, Michael James
Hirabayashi, Kohji
Holloman, James Jared
Hoxmeier, Steven Paul
Huixiu, Ma
Itkin, Vladimir Yury
Jacques, Frederic
Jaloway, Alexander Jose
Kimmick, Anne Marie
Kozak, Tatiana
Kropf, Frank Joseph
Lai, Anthony
Lam, Margaret Wai-Kwok
Lauziere, Isabelle
Lawry, Donna Christine
Levin, Samantha Casanov
Levine, Adam M
Levine, Lauren Feldman
Liang, Yonghua
Lin, Rui
Lin, Sheng Lun
Liu, Wanyin
Liu, Wenjing
Lo, Ka Wai
Lynch, Douglas
Mensana, Tina Christian
Moje, Marissa Ann
Noble, Christopher F.
Patel, Deep M.
Pedersen, Craig Franklin
Petruso, Cristina Liana
Phan, Chan Huu
Phelan, Alice Elizabeth
Puffer, Kyle Andrew
Remes, Heather Marie
Rennison, Adam Joseph
Scattone, Anthony Charles
Schlafly, Aaron Hutchin
Schneevoigt, Frank
Seow, Fan Chong
She, Xiaotie
Silvers, Louis Harold
Stovall, Brenda Gail
Tao, Yuan
Tobia, Anthony Joseph
Tucek, Christopher Ryan
Tun, Moe M.
Vaughn, Lynnette Dawn
Wade, Daniel Richard
Watkins, David William
Wendling, Adam K
Wickenheiser, Wesley Jay
Wieck, Matthew Robert
Winkels, Travis John
Wong, Yuk Lun
Wu, Haichuan
Xiang, Yu
Xu, Yulong
Yang, Xuelian
Yiu, Man Hay
Zagortz, Kevin M.
Zeillmann, Peter Raymond
Zelazoski, Paul Timothy
Zhang, Ge
Zhang, Min
Zhang, Zhenyong
Zheng, James Jie

New Fellows March 2002

It is a pleasure to announce the following 121 additions to the Fellows of the Society of Actuaries as a result of successfully completing the Fellowship Admission Course in March 2002.

Abate, Glenn D.
Anderson, Gregg W.
Ashford, Debbie L.
Balevich, Igor
Begley, Alison J.
Bergeron, Eric
Binioris, Steve P.
Bolduc, Lisa
Braza, David J.
Byers, Nancy Ellen
Camba, Francisco
Carlson, Marjorie S.
Carlson, Thomas P.
Chee, Shui-Chu Jennifer
Clingerman, Curtis M.
Cohen, Eliezer
Collie, Henry N.
Crawford, Ronald N. H.
Curley, Bryan J.
DeSoto, Kathleen Ann
Desrosiers, Patrick
Dionne, Jerome
Dossett, Kristen S.
Draper, Chris L.
Duchesne, Julie
Elbert, Melissa Brisson
Fienman, Matthew D.
Fischer, Larry A
Floman, Keith E
Forcier, Marie-Josee
French, Timothy C.
Gaynor, Timothy P.
Gibson, Sabrina H.
Girard, Jean-Francois
Glenn, Brian G.
Gray, Corrine M.
Guerin, Monique V.
Gupta-Lavey, Anju
Gurreri, Josephine A.
Hanzlik, David L.
Hardiman, Nathan W.
Hawksworth, Michael J.
Hayes, Michael C.
Hebert, Michel
Keppler, Matthew P.
Kiang, Grace C.
Kinnison, Chris L.
Kirk, Ketra N.
Kjellsen, Jennifer S.
Kline, Martin G.
Kong, Wun Joanna
Kordovi, Joseph
Lambright, Marc Alan
Laudato, Anthony C.
Lee, Myra Mo Chi
Lee, Theresa H.
Levy, Kelly A.
Lewis, Jennifer L.
Liang, Hung-Hsun
Lin, Terence Chun-Hung
Linneman, David K.
Lipkin, Glen M.
Lockhead, Brigitte
Lucia, Jeffrey E.
Lui, Fung-Chu
Lyon, Brenda Kay
Mangum, Nancy E.
Mastroberto, J. C.
Matte, Emmanuel D.
Matthews, Angela M.
McGuire, Mark E.
McNamara, Stephen J.
McPherson, Peter D.
Mermelstein, Joshua C.
Monstvil, Kimberly L.
Muhlhausen, Christopher
Na, William
Nault, Louis
Nimmer, Timothy N.
Parson, Trevis G.
Pauls, David J.
Pauwels, Rebecca
Pedersen, Kirsten M.
Pellerin, Francois
Pepin, Pascal
Ploc, Kathryn S.
Porcelli, Julie L.
Procaccitto, Lisa
Radunz, Wendy M.
Reichert, Enid M.
Rine, Jay S.
Roy, Marie Andree
Ruest, Gaetan P.
Santerre, Stephane
Sasveld, Karen J.
Schott, Theresa A.
Scott, Rebecca Irene
Seiler, Brent W.
Sidikman, Kenneth L.
Simanek, Bradley H.
Tabor, Todd D.
Tang, Connie W.
Terry, Keith A.
Thompson Jr., Stafford L.
Thompson, Darren C.
Turner, Keith D.
Ulm, Eric Robert
Van Schie, Alyssa M.
Velasquez, Dawn L.
Weltzin, Michael Allen
Westphal, Amanda M.
Whittaker, Jeffrey Bruce
Williams, Charles R.
Williams, Gayle L.
Winkelman, Ross A.
Witt, Julie M.
Wofford, Richard K.
Wolownik, Teresa Ellen
Zain, Jefferey
Zavist, Thomas More
Zhu, Julie Y.

CE corner

On CE partnerships with private companies

by John Riley, SOA Managing Director of Continuing Education

By the end of this year, the SOA's Continuing Education (CE) department will have conducted at least three seminars jointly sponsored with private companies. In the CE lexicon, "joint sponsorship" means that SOA and a company agree to an equal share of the proceeds (or losses) from seminar registration fees after costs. In dividing the labor for these endeavors, SOA handles publicity, registration and meeting logistics, while the private company handles program development and speaker recruitment. These early courses are "trial balloons" to determine the feasibility of the joint sponsorship idea. Of course, the question: "does it work?" is entirely different than the question: "should the SOA do it?" which deserves debate even if the experiment yields positive results. What follows here is a defense of the nonprofit/private company collaboration and an invitation for you to participate or at least to let us know your position.

In August of 2001, the Continuing Education Coordinating Committee (CECC) approved a trial of the CE seminar partnership and also recommended that we inform Presidents, CEOs, CFOs, and other company leaders. The SOA President and other officers on the Board were supportive of the idea because it meshes with the organization's strategic objectives. So on September 10, an e-mail letter was sent to nearly 1000 people in the SOA database. The tragic events of the next day certainly diminished the discussion, but most respondents were favorably disposed to the idea. Opponents to nonprofit/private joint sponsorship offered two counterarguments: the arrangement compromises SOA's neutrality since alternative points of view are not represented; and that such courses are unfair to competitor companies that were not selected or aware of the opportunity.

To state these views briefly is not to discredit them. In fact, no argument completely refutes these concerns. Any time that SOA provides a forum, it can be said to tacitly endorse presenters and their companies, even though we make no representations regarding content. Additionally, whenever we produce seminars, qualified presenters may get overlooked and could understandably feel slighted. So the question becomes: does the nonprofit/private company arrangement elevate these concerns to such a level that these ventures should not be pursued? We believe that safeguards and practices are in place or can be developed to address these concerns. Staff actuaries review course content for seeming commercialism and antitrust considerations. And, there is also a constraint of commonsense, since no self-respecting company representative is naïve enough to believe that selling in a seminar will win over customers as knowledgeable and innately skeptical as actuaries.

Questions of which companies get to go first or which companies get the "hot topics" are more difficult to address equitably. One solution might be for SOA to issue requests for proposals and set up some adjudication process. CECC could also establish guidelines on faculty representation, topic selection, etc. Although steps will be taken in this area, we must be careful that an effort to manage the process does not end up discouraging participation and encouraging bureaucracy. Many good partner candidates will not go through a lengthy qualifying process and the SOA's CE department will face an additional burden administering one. Finally, even with dozens of regulations in place, charges of favoritism, neglect and abuse of process are probably unavoidable. In the end, one simple but important principle should govern: SOA must give serious consideration to any joint sponsorship proposal from any company that identifies a legitimate educational need.

The SOA/private company CE model has much to commend it. Nonprofits and private companies make excellent training partners because nonprofits can reach the right audience and private companies have "real world" course content. SOA will always produce collaborative programs with help from the Sections and Practice Areas, but few people would maintain that all topics are best addressed in this manner. With a financial incentive and industry recognition, private companies are motivated to provide the best speakers and programs possible. They have real clout with high profile presenters who wouldn't deign to come to SOA events otherwise. Freed from these responsibilities, SOA can concentrate on publicity and support of all its programs. These alliances are clearly the best way for CE to increase the overall quality and number of seminars it produces.

The SOA strategic plan establishes a goal of becoming "the first choice provider of direct and indirect education," and it states that "buying instead of building" should be utilized in pursuit of that goal. A lot of our membership work in companies that offer world-class consulting and training services and a lot of our membership can benefit from this fact. SOA can bring these groups together. While issues of fairness and objectivity should be considered, providing continuing education of the highest quality argues strongly for the joint sponsorship proposition. Again, I welcome your proposals and comments.

John Riley is the Managing Director of Continuing Education. He can be reached at jriley@soa.org.