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Successful aging—an emerging paradigm of gerontology

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The social, medical, and economic problems of aging populations in the developed nations have evolved over the first 70 years of the past century. Over an historically brief time, an age in which infectious diseases dominated the morbidity and mortality patterns gave way to an era in which chronic diseases, such as cancer, heart disease, and stroke on the mortality side and arthritis and Alzheimer's disease on the morbidity side, came to dominate the illness burden of developed nations. In the late 1960s, another epoch began; one in which declines were noted in the incidence of some of the most important chronic illnesses, particularly cardiovascular disease and stroke [1].

Increasingly, therefore, the illness burden began to be determined by the frailty of seniors and the frailty adding to and complicating the problems of chronic illness. The acute diseases of premature death with little morbidity first had given way to chronic problems of much longer duration and much greater cumulative disability. The scientific advances of antibiotics, insulin, and many others enabled people to live longer and thus to experience greater cumulative disability. This paradoxic problem was termed by some the ''failure of success'' [2], because rapid increases in life expectancy from birth (from 47 years at the turn of the twentieth century to 76 years later in the century) were accompanied by an increase in the absolute amount of morbidity in the typical life and an increased fraction of that life spent in other than perfect health. Demographic projections suggested a very gloomy health future for developed nations, with predictions of astronomic nursing home needs and a decline in the national quality of life.

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Emergence of the compression of morbidity paradigm

The hypothesis of the Compression of Morbidity was presented initially in 1980 [3], and the author has structured this article as a largely personal view of the evolution of the data and implications over the past two decades. In 1980, the bulk of the national illness burden was noted to occur toward the end of life, perhaps between the ages of approximately 55, where disability levels began to be above zero for the typical person, and the age of 76 when the median person died. Over the preceding approximately 20 years or more, there had been, on average, steadily increasing morbidity,

The Compression of Morbidity hypothesis suggested that the national illness burden could be substantially reduced if the point in age at which chronic illness or frailty began could be moved later in life, approaching more closely the average age at death, and compressing morbidity between two points now closer together. Fig. 1 diagrams morbidity compression and contrasts it with the

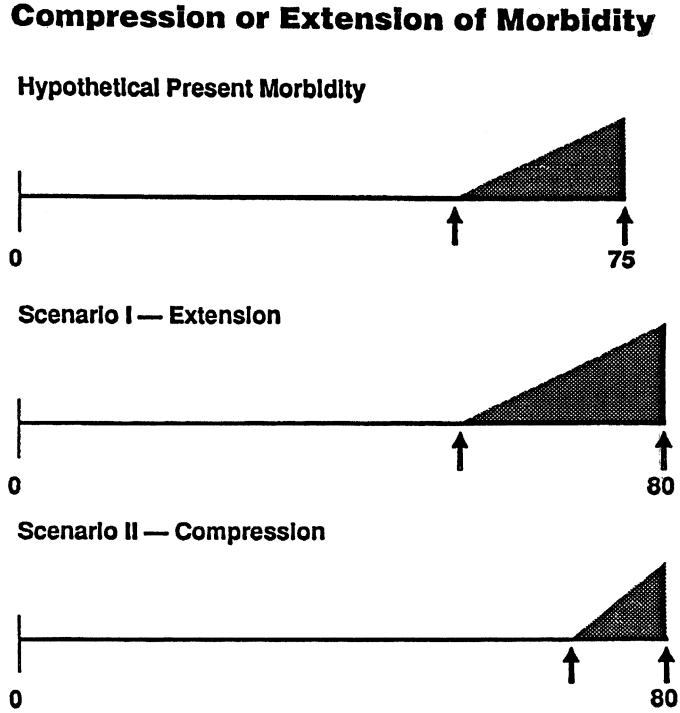

Fig. 1. Schematic representation of present and future health. Cumulative disability is represented by the shaded regions. The average life is represented from birth until death. Area-under-the-curve disability is influenced by the relative movement of the two arrows: one representing the age of onset of first chronic infirmity and the second representing the average age at death. (From Fries J, Vita A, Terry R, et al. Aging, health risk, and cumulative disability. The New England Journal of Medicine 1998;338:1035–41, with permission.)

increasing misery predicted by the "failure of success" notion. A proper metric to assess progress toward this goal would be computation of cumulative lifetime morbidity (or cumulative lifetime disability), represented schematically on the figure by the shaded areas. If lifetime morbidity (disability) could be decreased, then positive effects might be expected for the health of seniors, for age-specific disability levels, and for the costs of medical care [4].

The compression hypothesis seemed an ideal life pattern to many, presenting a vision of a long and vigorous lifetime ending after a relatively short terminal decline, and the vision of a society of healthier seniors contributing positively seemed a similarly happy one. A worthy goal, to be sure, but was it realistic? The Compression hypothesis did not fit the dominant medical paradigm, which implicitly considered the relationship of age and morbidity to be a fixed one, while death could be indefinitely postponed, or even "cured." After all, although life expectancy from birth was rising rapidly and was well documented, no comparable data existed on age-specific morbidity rates. Moreover, postponing the onset of morbidity clearly implied primary prevention and a role for behavioral and physical interventions, contrary to the curative expectations of rapidly advancing medical science and the technologic imperative. To suggest that progress might occur if people took better care of themselves was to sound dangerously naïve or, worse, antiscience.

Two assumptions underlay the Compression hypothesis [1,3,4]. The first is that the life span is ultimately fixed and finite, with an average age at which natural death occurs even in the absence of disease or trauma beyond which age life expectancy may not be further prolonged and against which morbidity might be compressed. This assumption is useful but not necessary. The second assumption is that the age at first chronic disability or initial chronic morbidity may be postponed. This is a necessary assumption. The important concept is the dynamic relationship over time between the age of illness onset and the age at death. Many measures that might postpone morbidity also tend to extend life expectancy (as with heart disease and cancer but not with arthritis or Alzheimer's disease). The issue is whether it is easier to postpone the onset of morbidity or the end of life [4].

The Compression hypothesis was initially met with controversy [5–7]. Some demographers had presented projections for longevity without any upper limits. Some social scientists feared that any measure of optimism about some of the problems of aging might deflect planning priorities for the age boom, as people already born grew older. Some medical scientists worried that resources might be diverted to behavioral and preventive activities, which instead should be devoted to finding cures for diseases of the aging (eg, Alzheimer's disease). Several critics had belief structures that required the possibility of immortality.

The concept of natural death, absent disease or trauma, can be at once familiar and foreboding. The gerontology literature generally embraces the idea of different life spans for different species and of generally linear declines in organ reserve capacity (beginning at age 30 in humans), which must at some point eventuate in an organism that cannot sustain life [1,3]. A common human experience is

that of pets that clearly grow old and feeble and will soon die and of the universality of death in man and beast. Yet, the computation of an expected maximum life span at about 85 years of age or so initially was received with skepticism, partly because the only reasonably certain conclusion about optimal life expectancy is that it must be somewhat above current life expectancy.

The mortality experience

Since 1980 there has been an increase in life expectancy from all ages; however, the increases have been slower from more advanced ages [8]. The rate of increase per year in life expectancy from birth is 0.150 years per year; from age 65 the rate is 0.066 (less than half) and from age 85 the rate is 0.017 (approximately 2% of a year per year). In the original presentation of the Compression of Morbidity hypothesis [3], the author argued that such differential increases in life expectancy from different ages mandated that when trends with different slopes were projected into the future a point of paradox would be reached when the lines crossed. After this point the life expectancy age from birth would be greater than life expectancy age from, for example, age 65. Because this is not possible, projected curves must flatten asymptotically so that the point of paradox is not reached.

Life expectancy is thus increasing much more rapidly from birth than from age 65, where it in turn is increasing much more rapidly than from older ages [8]. These rates are shown in Table 1. If these linear increases continue, they will reach an intersection beyond which the expected age of death from birth exceeds the expected age of death for those already 65 or older. This point was originally calculated at approximately 85 to 86 years and would be reached in the mid-twenty-first century. The trends could not extend beyond that point, at which there

Table 1
Projecting the human life span—US Data 1980–1998

Both sexes combined			
	From birth	From age 65	From age 85
Current life expectancy (1998)	76.6	82.7	91.3
Rate of increase/yr	0.150	0.066	0.017
Projected life expectancy (2048)	84.1	86.0	92.15
Point (yr) of paradox		87.8 (2076)	
Males			
Current life expectancy (1998)	73.7	81.0	90.6
Rate of increase/yr	0.20	0.10	0.04
Projected life expectancy (2048)	83.7	86.0	92.55
Point (yr) of paradox		88.3 (2071)	
Females			
Current life expectancy (1998)	79.3	84.1	91.6
Rate of increase/yr	0.094	0.028	0.011
Projected life expectancy (2048)	84.0	85.5	92.15
Point (yr) of paradox		87.1 (2104)	

would be no deaths at all before age 65. The point of paradox cannot be passed, and computation of the point of paradox gives an upper bound to the optimal life span given that current trends continue or there is a future decrease in rates.

Although projections using the most recent data and the same technique to project age at death from birth and from other ages yield similar results, the point of intersection is closer to 2100 and is a little higher. The lines intersect in the year 2076 at 87.8 years of age. At this point, there are projected to be no deaths at all below the age of 65. Because this is not a realistic expectation, ultimate longevity values must necessarily remain below these projections.

As with projections made by the Census Bureau or other analysts, projections here are essentially linear in nature. A scientific advance in genetic manipulation and changes in telomerase activity or in another area could result in abrupt deviation from trends that have been remarkably consistent for many years.

Current data [8] and projections from other sources suggest that current life expectancy for individuals at age 65 will increase by at most 3.3 years over the next 50 years. As a percentage of years remaining after age 65, life expectancy will go up perhaps 15% in 50 years. After age 85, life expectancy will increase by only 0.85 years in the next 50 years. As shown in Table 1, the point of paradox currently projects to be lower and reached later in time in females than males. This unlikely prediction suggests the substantial magnitude of error inherent in 50-year projections.

These projections of slow growth in life expectancy from advanced ages do not materially affect the usual computations of the actual number of seniors alive in, say, 2050. Those projected numbers are driven mostly by increases in the size of successive birth cohorts and in the proportion of each birth cohort which reaches age 65 (or 85) [9]. Only about 3% of projected increases in senior populations come from increases in life expectancy after age 65 (or 85).

The morbidity experience

Since 1980 there has been no national experiment that could fully test the hypothesis of Compression of Morbidity; the possibility of more successful aging. Some improvement has been noted in age-specific functional capacity [10]. On the other hand, although smoking prevalence has declined, an increase has been noted in obesity and little change in exercise levels. Hence, there has been little overall change in the major risk factors for disease and frailty on a population-wide basis. Over these years there have been increases in longevity for all age groups. The longevity increases have been smaller, however, particularly from advanced ages, than in the prior two decades.

In the 1980s, studies suggested little Compression of Morbidity on a national basis. In the early 1990s, data began to indicate a decrease in age-specific disability [10,11] and a decrease in age-specific mortality rates [8]; however, the relative amounts of these changes could not be compared in a definitive way. There was confounding by the association among fewer health care risk factors and education, income, occupation, and access to medical care.

Later in the 1990s several studies began to ask the question of the marginal benefit of primary prevention [12–14]. This is at once a more difficult but more relevant question: given that education, affluence, and access to excellent medical care advantages are already in place, how much additional benefit can be accrued by reducing health risks? To approach this question one needs to study populations that already have access to good medical care and already enjoy several societal advantages.

Longitudinal study of such populations comparing higher and lower health risk strata offers the opportunity to observe if lower lifestyle risk factors can be associated with additional postponement of the onset of morbidity or disability. Importantly, these studies can estimate the magnitude of such postponement. If the magnitude of such postponement was only a year or so, little might be expected from interventions to reduce health risks. If the magnitude of postponement was substantially larger, the potential for general health improvement could be very large.

Postponement of disability—how far?

Several studies now provide data that disability may occur substantially later, even in already healthy populations, in individuals with few behavioral health risk factors [12–14]. For the past 14 to 16 years, the authors have been performing two such large longitudinal studies to determine the magnitude of the effect of healthy behaviors on the development of disability. These studies were designed to directly test the compression of morbidity hypothesis, and our results have been replicated by other investigators.

In the University of Pennsylvania study [14], the authors studied 1741 University alumni who were surveyed first during their college years in 1939 and 1940 and again in 1962 (at an average age of 43 years) and then annually by our group starting in 1986. Our major dependent variable was cumulative lifetime disability. Three strata of high, moderate, and low health risk subjects were defined prospectively on the basis of smoking, body mass index, and lack of exercise risk factors. Cumulative disability was determined by summing disability scores of the Health Assessment Questionnaire (HAQ), administered each 6 months and scored on a scale of zero to three. Cumulative disability from 1986 to 1994 (average age in 1994: 75 years) or to death was the measure of lifetime disability.

Persons with higher health risks assessed either in 1962 or in 1986 had twice the cumulative disability of those with low health risks (disability index, 1.02 versus 0.49; $P < 0.001$). The results were consistent among survivors, subjects who died, men, women, and for both the last year and the last two years of observation. The onset of disability was postponed by an estimated 7.75 years in the low-risk group as compared with the high-risk group. The cumulative disability index in the last 1 or 2 years of observation for the low-risk subjects who died was approximately half that for the high-risk subjects who died, indicating that those with fewer health risks also had less disability at the end of their lives.

Fig. 2 graphically shows the disability index according to age at the time of the last survey by health risk category in 1986. Although average disability increased with age in all three risk groups, progression to a given level of minimal disability was postponed by 7 to 9 years in the low-risk group as compared with the high-risk group. The horizontal line on the figure indicates a disability index of 0.1, which corresponds to very minimal disability as measured by functional abilities at daily activities.

Fig. 3 shows cumulative disability according to health risk groups formed from 1986 data. Disability was assessed at an average age of 67 years. The data represent approximately 8 years of follow-up for subjects who were alive at the end of the study and an average of 5 years of follow-up for subjects who died. Cumulative disability in the high-risk group was approximately twice that in the low-risk group for all subjects, those with no disability in 1986, those who survived, and those who died. For reference, a score of 1.0 would correspond to moderate disability in performing two of eight activities of daily living (eg, walking or rising from a chair) for 2 years or to mild disability in performing all eight activity categories for 1 year.

Thus, fewer health risks, prospectively defined, assessed in subjects with economic and access advantages, are associated with delay in disability of nearly

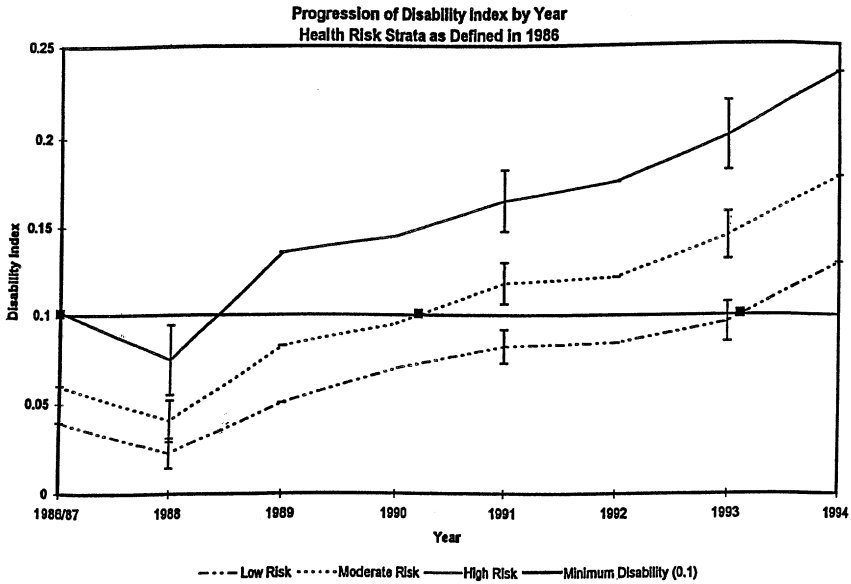

Fig. 2. The University of Pennsylvania study. Subjects in three strata of health risk, based on smoking behavior, exercise, and obesity, are followed over time for development of disability. The computed delay between high and low risk groups in reaching a level of minimal disability is 7.75 years. (From Fries J, Vita A,Terry R, et al. Aging, health risk, and cumulative disability. The New England Journal of Medicine 1998;338:1035–41, with permission.)

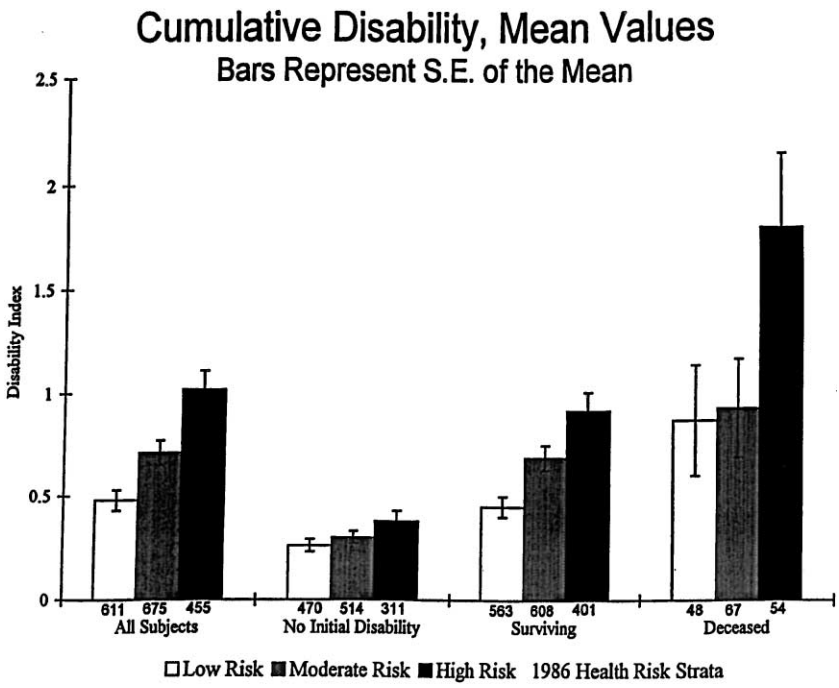

Fig. 3. The University of Pennsylvania study. Subjects in the same three strata of health risk are plotted by disability against year of age. Development of disability is successively delayed in the moderate and then the low-risk group. (From Fries J, Vita A, Terry R, et al. Aging health risk, and cumulative disability. The New England Journal of Medicine 1998;338:1035–41, with permission.) Copyright © 1996 Massachusetts Medical Society. All rights reserved.

8 years. This finding is a large effect and contrasts with a smaller delay of 2 to 3 years with these same health risk groups. These studies are ongoing.

Our second longitudinal population study is a prospective cohort study of senior runners club members from across the United States and controls from a Stanford University community study with annual follow-up from 1984 through 1997 [15,16]. Our goal was similar to that of the University of Pennsylvania study: to understand the magnitude and duration of benefit or harm from running and other aerobic exercise on subsequent disability and mortality. The authors hoped to quantify the benefits of long distance running and other aerobic exercise in seniors and to test the hypothesis that morbidity can be postponed and compressed into the later years of life by healthy lifestyle habits including particularly regular vigorous exercise. Our participants were a cohort of 370 runners club members and 249 community controls initially 50 to 72 years old (mean, 59 years) also classified as ever- runners (n = 464) and never-runners (n = 155). Main outcome measures were the cumulative HAQ disability score, duration of postponement of disability, and time to and cause of death.

The study design paid special attention to elimination of self-selection bias, using large differences in the independent variable between groups, longitudinal study, intention-to-treat analyses, stratification for age and gender, and adjustment for variables such as injury, obesity, congenital abnormality, baseline disability status, family history of arthritis, and others.

The authors found that initial lower disability levels in runners club members over controls ($P < 0.0001$) and ever-runners versus never-runners ($P < 0.0001$) were sustained for at least 13 years and that the differences increased over time ($P < 0.0001$). A disability level of 0.075 occurred 8.7 years later in runners club members than in community controls, and a disability level of 0.1 was estimated to be reached over 12 years later in runners club members than in community controls (Fig. 4) Females appeared to derive the greatest disability benefits from exercise. Running club membership and participation in aerobic exercise other than running also protected against premature mortality. The rate ratios for mortality were 0.36 and 0.88, respectively. Male gender and smoking were detrimental; rate ratios for mortality were 2.4 and 2.2, respectively. Control subjects had higher death rates in every disease category, including cardiovascular disease and most cancers. Through an average age of 72 years, disability remained almost nonexistent in vigorously exercising individuals, even after controlling for

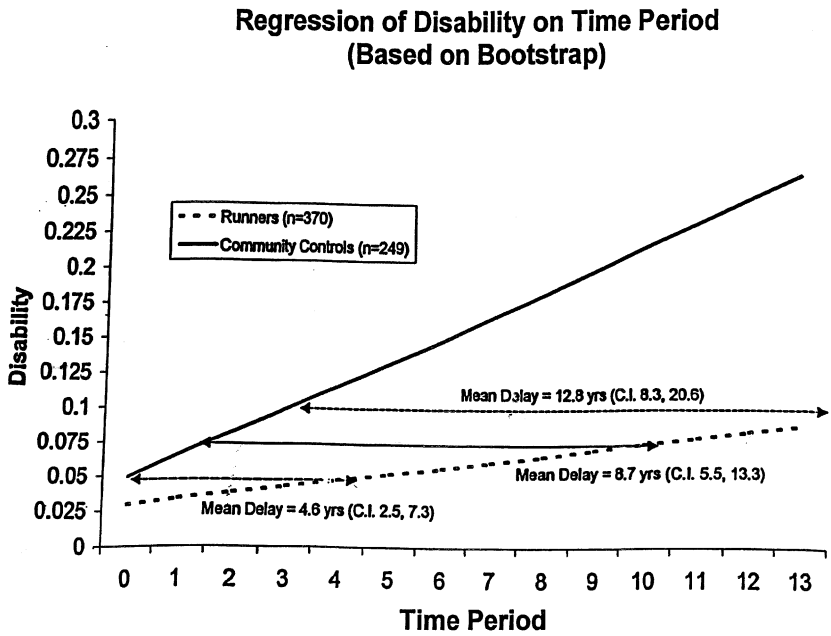

Fig. 4. The Precursors of Arthritis study. Very slow development of disability over time in vigorously exercising subjects compared with more sedentary controls is evident. The delay in reaching a minimal disability level increases with time to over 12 years.

eight separate forms of selection bias. Mortality differences were also seen but were less striking.

Additional studies have confirmed these findings and have extended the associations to include reduced medical care costs, which are strongly correlated with disability levels. Stearns et al [17] used Medicare data to link health habits and practices to expenditures. Reed et al [18] studied 8000 Japanese males older than 28 years of age, with the results closely paralleling those above. Cigarette smoking, obesity, hypertension, and lack of exercise in midlife and beyond accounted for much of the disability of the elderly. Daviglus et al [19] found that cardiovascular risk factors in midlife were strongly associated with Medicare costs. Blair and Wei [20] recently reviewed the literature and found more than 30 studies linking sedentary habits to subsequent decline in health and function in older adults. A striking characteristic of this literature is the quality of the methodology, the quality of the journals, the number of investigative groups with colnfirmatory findings, and the absolute absence of contrary studies.

Health risk reduction in senior populations

Because of the now proved association between health risks and subsequent health, the burden of proof now shifts to questions concerning interventions. Can risks be reduced in seniors? How? Are there health gains? How large? Are there associated reductions in medical costs? What is their magnitude? This literature is rapidly enlarging and is now supported by multiple randomized clinical trials and several large controlled but nonrandomized studies. This literature is not entirely positive. Some interventions do not appear to work, and some approaches, such as community-based interventions, show promise but little proof. Successful programs have included self-management techniques and approaches to increasing personal self-efficacy and reduction in specific health risks and have been delivered in small group settings or through telephone and mail delivery.

Our group has participated in four randomized controlled trials in seniors, using a mail-delivered, computer-driven, tailored print program, which is serially reinforcing and extremely personalized to individual needs. All have been very positive studies, reducing risks, improving health, and reducing costs. The Bank of America retiree study [21] included 4712 participants studied over a year and randomized into program, questionnaire only, and claims-only controls**.** Health risks were reduced 12%, costs were reduced 20%, and self-reported health status improved approximately 12%. Return on investment was six to one. The California Public Employees Retirement System Study [22] had a similar design and included 57,268 subjects. Health risks were down 10% and claims costs were down about the same. Return on investment was five to one. Smaller trials of arthritis-specific [23] and Parkinson's disease-specific [24] programs, with similar designs, had similar results. Table 2 summarizes results of both randomized and controlled but not randomized studies with the same type of intervention [25,26].

Fries et al [27,28] have reviewed this area and have summarized the program attributes that are associated with the best results. Health policy implications are

Table 2
Need and demand reduction

	n	Time	Health risk score	Cost per person	Savings per person	ROI
Randomized trials						
Bank of America	4712	12 mon	– 12%	$29	$179	6.1
CALPers	57,268	12 mon	– 10%	$59	$300	5.1
Arthritis	809	6 mon	– 7%	$50	$260	5.2
Parkinson's	290	6 mon	– 10%	$100	$570	5.7
Take Care of Yourself	2833	12 mon	– 17%	$6	$20	3.5
Controlled but not randomized studies						
Citibank (Medstat)	22,933	32 mon	– 18%	$158	$1060	6.7
High-Risk	2586	6 mon	– 11%	$69	$484	7.0
General Program	103,937	30 mon	– 19%	$30	$70	2.3
Take Care of Yourself	5200	12 mon		$6	$42	7.0

Abbreviation: ROI, return on investments.

clear [29,30]. Cost-saving health risk reduction programs can be effective in senior populations and can improve health. Provision of such programs as a Medicare benefit could substantially reduce Medicare expenditures and could substantially improve the health of seniors.

Summary

In the largely successful preventive approach to reduction in cardiovascular disease prevalence, three classic stages of investigation were used. First, an hypothesis was raised that diet and cholesterol levels were a cause of heart disease. Second, multiple longitudinal observational studies, led by the Framingham group, documented a strong association between these health risks and heart disease mortality. Finally, randomized controlled trials of cholesterol-lowering drugs established proof of causality. Our understanding of the Successful Aging phenomenon has followed the same sequence. The Compression of Morbidity hypothesis sets forth a new and promising paradigm. Multiple longitudinal and cross-sectional observational studies show strong associations consistent with the hypothesis. Finally, randomized controlled trials of healthy aging interventions prove our ability to successfully intervene in this most important of all contemporary health problems: the health of seniors [28].

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