

## **Causes of Improving Health and Longevity at Older Ages: A Review of the Explanations**

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## **Abstract**

This essay documents trends in elderly health and longevity and reviews various environmental explanations for the decline in elderly mortality and morbidity, including improvements in medical technology, reduced infectious disease rates, reduced occupational stress, improved nutritional intake, life style changes, rising incomes, and rising education.

Questo saggio documenta la tendenza verificata nella salute e nella longevità degli anziani ed esamina varie spiegazioni ambientali del declino della mortalità medica, della ridotta morbosità, incluse quelle del miglioramento della tecnologia medica, della ridotta proporzione di malattie infettive, del ridotto stress professionale, del miglioramento della nutrizione individual, del cambiamento del modo di vivere, degli aumenti salariali, e del miglioramento della istruzione.

Cet essai documente des tendances dans la santé et longévité des vieux et réexamine de diverses écologiques explications pour le déclin dans la mortalité et la morbidité, y compris les améliorations dans la technologie médicale, la réduite des taux des maladies contagieuses, la tension de métier réduit, la prise alimentaire améliorée, des changements de style de vie, élevant des revenus, et education montant.

### **1. Trends in Elderly Health and Longevity**

Life expectancy at older ages rose very slowly at the beginning of the twentieth century and then accelerated sharply toward the end of the century. Between 1970 and the end of the twentieth century life expectancy at age 65 increased by 4 years in Sweden and France and by 3 years in England and Wales and in the United States. In contrast, between 1900 and 1960 life expectancy at age 65 rose by only 2 years in Sweden and France and by one and a half years in England and Wales. National data are unavailable for the United States for this period but at most the increase in life expectancy was two and a half years and was probably considerably less. The life expectancy pattern at age 85 is even more striking. Life expectancy at age 85 rose by almost 2 years in all four countries between 1960 and the end of the twentieth century after having risen by only about 1 year between 1900 and 1960.<sup>1</sup>

On the whole population aging has been accompanied by declines in disability at older ages (see the review by Freedman, Martin, and Schoeni, 2002). Manton, Corder, and Stallard (1997) find that between 1982 and 1989 disability among those older than 64 declined by 1.1 percent per year and between 1989 and 1994 by 1.5 percent per year. In exactly comparable data for 1994 and 1999 disability declined by 2.1 percent per year (Manton and Gu, 2001), suggesting that improvements in elderly health have been accelerating. Using different data sources and different measures of disability, Cutler and Richardson (1997) find declines of 0.5 to 1.0 percent per year between 1980 and 1990; Freedman and Martin (1998) observe declines of 0.9 to 2.3 percent per year between 1984 and 1993; Schoeni, Freedman, and Wallace (2001) find declines of 1.0 percent per year; Crimmins, Saito, and Reynolds (1997) observe a disability decline of 0.9 percent

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<sup>1</sup> See the Human Mortality Database and the Berkeley Mortality Database, <http://www.demog.berkeley.edu>. All life expectancies are period life expectancies.

per year between 1982 and 1993; and Liao *et al.* (2001) found declines of 1.53 percent per year between 1984 and 1995. Looking at the 1990s alone, Waidmann and Liu (2000) found disability declines of 1.5 percent per year between 1992 and 1996. Comparing the offspring of individuals in the Framingham study with the original cohort shows that among those age 55-70 disability has fallen substantially between 1977 and 1994 (Allaire *et al.*, 1999). Declines in functional limitation appear to follow a similar trend to declines in disability with declines of 1.5 percent per year between 1992 and 1996 (Waidmann and Liu 2000) and of 1.3 to 1.6 percent per year between 1984 and 1993 or 1995 (Freedman and Martin 2000; 1999; 1998). Looking at trends over a century, Costa (2002) finds that among men aged 60 to 74 functional limitations declined by 0.6 percent per year between 1910 and the 1990s, suggesting that there has been an acceleration in improvements in disability and in functioning.

Trends in the prevalence of chronic conditions are less clear, but clinician reports document continuous improvements in chronic conditions since the 1970s (Waidmann, Bound, and Schoebaum, 1995). In contrast, self-reports show increases in chronic conditions during the 1980s and during the 1970s (Freedman and Martin, 2000: Chirikos, 1986: Colvez and Blanchet, 1981: Crimmins, 1990: Verbrugge, 1984), suggesting that awareness of health problems has increased. However, if self-reports are accurate then they imply that although chronic disease rates have risen disability has fallen since the 1980s because of declines in the debilitating effects of chronic disease. Clinician reports covering the span of a century suggest that average decline in chronic respiratory problems, valvular heart disease, arteriosclerosis, and joint and back problems was about 0.7 percent per year from the 1900s to the 1970s and 1980s (Costa, 2000). Over a

century 24 percent of the decline in functional limitations is attributable to declines in the debilitating effects of chronic disease, 37 percent is due to reduced chronic disease rates, and the remainder is unknown (Costa, 2002).

Fogel and Costa (1997) argue that the recent increases in health and in longevity are too rapid to have been caused by genetic or evolutionary change and that explanations should focus on changes in the physical environment. Of course, life span and health are to some extent inherited. The siblings of centenarians also have low mortality rates (Perls, Wilmoth, *et al.*, 2002); the life spans of adopted children are positively correlated with those of their parents for early deaths (Nielsen *et al.*, 1992); twin studies show that susceptibility to disease is inherited (Yashin and Iachine, 1995) and estimate the heritability of longevity to be roughly 25 percent (Herskind *et al.*, 1996). Genetic and environmental factors may be linked. Oxidative stress from environmental agents may damage DNA (Bohr *et al.*, 1998) while clusters of protective genes may increase resistance to particular chronic diseases (Takata *et al.*, 1987) and genetic variations may promote environmental influences by altering metabolic pathways (Finch and Tanzi, 1997). The genetic component of variation in both cognitive and physical ability increases with age, but so does the environmental component of variation (McClearn *et al.*, 1997; Christensen *et al.*, 2003). Nonetheless, genetic evolution takes time: studies of aging and mutations in fruit flies use populations that have been maintained for more than 100 generations (Rose *et al.*, 2002).

This essay reviews various environmental explanations for the decline in elderly mortality and morbidity, including improvements in medical technology, reduced infectious disease rates, reduced occupational stress, improved nutritional intake, life

style changes, rising incomes, and rising education. Improvements in medical technology can have an immediate effect on older age mortality whereas the other explanations are likely to have lagged effects.

## **1. Explanations**

### **2.1 Medical Care**

The availability of appropriate medical care has an immediate effect on older age mortality, as seen in such rare cases as the unification of Germany, where a failing health care system was immediately replaced by a highly developed medical care system (e.g. Gjonça, Brockmann, and Maier, 2000) and as inferred from mortality comparisons between Western and Eastern Europe in conditions amenable to medical intervention (Velkova, Volleswinkel-Van den Bosch, and Mackenbach, 1997). Such discontinuities in mortality are rarely observable in time series data. Diffusion of new procedures and knowledge of how to implement them is not immediate. In the United States programs such as Medicare which began to provide health care coverage for the elderly beginning in 1965 probably had a lagged, indirect effect by encouraging treatment innovations for the elderly through teaching hospitals and clinical research, leading to improved mortality for both simple and complex procedures, and improved mortality for years after surgery (Manton, Stallard, *et al.*, 1997). Cutler, McClellan, and Newhouse (1999) conclude from their review of the clinical studies that changes in medical treatment used in the managements of acute myocardial infarction account for approximately 55 percent of the

reduction in mortality that has occurred in acute myocardial infarction cases between 1975 and 1995, a time period when heart attack mortality fell by almost one-third.

The impact of medical care on disability rates is less clear cut. Even with no decline in chronic disease rates disability may have declined because medical technology allows for short-term symptom relief and for long-term control of chronic conditions. Since the late 1960s there has been a remarkable increase in therapies targeted at the elderly population. Non-steroid anti-inflammatory drugs, gold compounds, hydroxychloroquine, sulfasalazine, corticosteroids, and cytotoxic and immuno-suppressive drugs all alleviate rheumatoid arthritis. Fries et al. (1996) find up to a 30 percent reduction in long-term disability in patients with rheumatoid arthritis with consistent use of disease-modifying antirheumatic drug-based treatment strategies. Cataracts was previously one of the main causes of blindness at older ages (Costa, 1998: 64), but today cataracts extraction and implantation of a plastic or silicone lens is a common outpatient procedure. Kahn (1999) emphasizes that new medications have increased diabetic quality of life. Such surgeries as cardiac catheterization, bypass, angioplasty, and installation of pacemakers and such drugs as beta-blockers, Ca blockers, vasodilators, ACE inhibitors, and thrombolytics control cardiac conditions. Chronic obstructive airway disorders such as asthma and chronic obstructive pulmonary disease are controlled by bronchodilator drugs (e.g. beta-agonists, anticholinergic drugs, theophylline, and corticosteroids) and by antibiotics when exacerbated by viral infections.

## 2.2 Reduced Infectious Disease Rates

The medical and epidemiological literature provide many examples of the possible linkages between early life infectious disease and chronic disease and cognitive disorders in later life. The link between infectious disease and rheumatic heart disease, a form of heart disease common in most developing countries and in the United States at the beginning of this century, is particularly well-established. Acute rheumatic fever involves the joints and the heart and subcutaneous tissue and results in damage to the heart valves. Today acute rheumatic fever most frequently occurs in children 6-15 years of age following group A streptococcal upper respiratory tract infection whereas in the nineteenth century it occurred more frequently among older persons, with 25 percent of first attacks occurring between ages 20-25 and 49 percent between ages 30-39 (Kiple, 1993: 971). Individuals who have had acute rheumatic fever become particularly likely to experience recurrences of attacks following the streptococcal infection. Crowding associated with poor living conditions is the major predisposing condition for increased risk of streptococcal infection and acute rheumatic fever.

Other infectious diseases that can affect cardiac functioning include late stage syphilis, measles, typhoid fever, and malaria. In a sample of typhoid patients, electrocardiogram assessments showed that 12 percent had cardiac involvement (Khosla, 1981). Electrocardiogram assessments of Nigerian children with measles showed that 35 percent had some electrocardiogram abnormality (Olowu and Taiwo, 1990). Malaria can result in either changes in the myocardium or myocardiopathy arising from malarial chronic anemia (Charles and Bernard, 1982).

There is also increasing evidence of a link between infectious disease and atherosclerosis. Several studies have detected chlamydia pneumoniae, a bacterium that causes acute upper and lower respiratory infections, in atherosclerotic lesions from coronary and carotid arteries, abdominal aortic aneurysms, and in sclerotic and aortic valves. Infections are known to influence lipid metabolism and may play some role in the atherosclerotic process itself by inducing damage and inflammation in the vascular endothelium in the presence of hypercholesterolemia (see reviews by Lindholt *et al.*, 1999; Valtonen, 1991; Wong, Gallagher, and Ward, 1999). In older work Buck and Simpson (1982) found a high correlation in the United States between atherosclerotic heart disease at older ages and diarrheal deaths from birth to age 20 and speculated that infection facilitates the production of autoimmune complexes that promote the later development of atherosclerotic lesions. The fatty streaks that are the precursors of these lesions may already be present in children by age 10.

Researchers have hypothesized that lower respiratory tract infections in childhood may aid in the development of chronic obstructive lung disease in later life, particularly during early infancy when children's lungs are undergoing developmental change. In a longitudinal study of British men, Barker (1992; 1994) found that bronchitis, pneumonia, and whooping cough before age 5 are associated with reduced mean forced expiratory volume in one second (FEV1) at ages 59 to 70. Follow-ups of disadvantaged children in South Africa revealed that lung function abnormalities persisted years after the contraction of pneumonia (Wesley, 1991). In this group of children, and in most developing countries, measles is a major cause of acute lower respiratory infection (Markowitz and Nieburg, 1991). Autopsies and radiographs show that measles

bronchopneumonia results in bronchiolar obstruction, airways distension, and a thickening of the peri-bronchial walls (Jean *et al.*, 1981). Previous tuberculosis disease may also lead to respiratory abnormalities. One third of typhoid patients also suffer from cough, suggesting that typhoid fever may also be a likely candidate for respiratory distress problems in later life. Other viral infections, such as pulmonary syphilis, might also play a role.

Musculoskeletal symptoms are common with many viral infections, as well as syphilis and malaria. Although in most cases these symptoms fade away after a short period, permanent joint damage can occur for a series of diseases such as tuberculosis, vaccinia, and gonorrhea, among others (von Hartmann, 1974). In rare instances a residual progressive arthropathy may develop after rheumatic fever (Katz, 1977).

Cognitive disorders have been related to specific infections, including bacterial meningitis (Bohr *et al.*, 1983), Lyme disease (Logigian *et al.*, 1990), and HIV infection. The most common causes of memory loss, however, include Alzheimer's disease, head trauma or injury, seizures, alcoholism, and stroke. Atherosclerosis and valvular heart disease all raise the risk of stroke and, as discussed above, these have been related to infectious disease.

Maternal infectious disease can also affect children's health in utero. Rubella and subsequent deafness is a well known example. Almond (2003) finds that cohorts who were in utero during the 1918 Influenza Pandemic experienced worse health and economic outcomes.

### **2.3 Reduced Occupational Stress**

Reduced occupational stress includes both the shift from manual to white-collar work and the reduction in occupational hazards within manual and factory work. In the United States circa 1940 men's risk of death on wage-paying jobs fell from roughly 0.17 per million hours worked to less than 0.07 by 1970, with most of the decline due to decreased risk of death within jobs (Costa and Kahn, 2004). Case and Deaton (2003) find that even with comprehensive controls for education and for income, those in manual occupations have worse self-reported health and more rapidly declining health with age.

Health could be directly affected through on the job exposure. Lung diseases and respiratory symptoms resulting from occupational exposure to dust, fumes, or gases include asthma, chronic bronchitis, chronic airflow limitation, and tuberculosis. Workers in mining, steel foundries, tool grinding, glass making, metal casting, and stone polishing are particularly prone to occupational lung disease. Farmers are also affected because of inhalation of organic dust from moldy plant materials and animal waste, hair, and feathers.

Occupational stress is also an important determinant of musculoskeletal capacity. Joint and back problems are substantial burdens in developing countries (Muirden, 1995) and were common afflictions in the United States at the beginning of the twentieth century (Costa, 2000). Even in modern, developed country populations musculoskeletal capacity is not only lower among men in physical rather than mental or mixed work, but the rate of deterioration in musculoskeletal capacity among men in physical work is greater (Nygard, Luopajarvi, Ilmarinen, 1988).

Direct physical injuries represent another form of on-the job stress. When in the form of head trauma these could lead to memory loss or other cognitive impairments through the accumulation of cerebrospinal fluid in the ventricles of the brain or direct cortical damage.

An indirect pathway might be through stress. Manual workers have less control over their work schedules, face repetitive chores, and hold jobs where prestige is low. Marmot *et al.* (1999) point out this may contribute to higher rates of cardiovascular disease, musculoskeletal problems, and psychological disorders.

## **2.4 Improved Nutritional Intake**

Explanations for disability declines focusing on improved nutritional intake encompass both the effects of early nutrition (including prenatal) and of mother's nutritional status on health. Barker (1992; 1994) finds in longitudinal data that undernutrition in utero and in the first year of life, as proxied by anthropometric measures of the child, increase risk of coronary heart disease and stroke. Other degenerative conditions of old age, including non-insulin-dependent diabetes and autoimmune thyroiditis have been linked to nutritional status as well (Barker 1992; 1994). These associations have been replicated in several European countries and in India (e.g. Koupilova *et al.*, 1993; Forsen *et al.*, 1997; Leon *et al.*, 1998; Stein *et al.*, 1996; 1997). Eriksson *et al.* (2003) found that faltering growth in length in the first few months after birth predicted type 2 diabetes. Studies of the Dutch famine of 1944-45 have found that the timing of malnutrition is critical: those exposed to famine in late or mid-gestation had

reduced glucose tolerance; those exposed to famine in early gestation had higher risk factors for cardiovascular disease and were more likely to report their health as poor; those whose mothers ate little protein in relation to carbohydrates during the third trimester had higher blood pressure at adult ages; and those exposed to famine in mid gestation had an increased prevalence of obstructive airways disease (Roseboom *et al.*, 2001). Maternal nutrition may explain the higher old age mortality of individuals born in the second relative to the fourth quarter (Dobblehammer and Vaupel, 2001). Dietary protein restriction during gestation in rats produces vascular dysfunction in pregnant rats and hypertension in their offspring (Koumentaki *et al.*, 2002; Brawley *et al.*, 2003).

The exact mechanisms through which undernutrition in utero programs adult disease are still unclear. Adaptation of the fetus to undernutrition is associated with changes in the concentrations of fetal placental hormones and these could be the link. In the case of non-insulin-dependent diabetes, the interaction between a poor prenatal “diet” and a rich adult diet may be important. Martyn *et al.* (1996) found that stroke risks were related to poor maternal nutrition that resulted in hip and pelvic structure defects and that heart risks were related to fetal nutritional deficiencies that may have affected the development of the liver and the pancreas. Subtle nutritional differences might affect the physiochemical development of organs and their function and lead to chronic disease risk differences at older ages as the homeostatic capacity of organs is challenged by age related pathologies accumulated in the course of normal life experiences (Kirkwood, 1998).

Nutritional differences at older ages may be important as well. One theory of circulatory disease (hyperhomocysteinemia) suggests that high levels of homocysteine (an

amino acid used in cellular metabolism and in the manufacture of proteins) chemically damage the arterial endothelium (artery wall) and initiate a pathological process (fibrotic change) in the arterial wall that results, in the presence of other co-factors, in the initiation and growth of atheromas in human arteries (McCully, 1983). The elevation of this metabolite may be related to folic acid and vitamin B6 deficiencies. High levels of homocysteine have been linked to poor performance on memory tests and to Alzheimer's disease, while high blood levels of folate improved memory recall (Morris *et al.*, 2001). In addition, improved nutrition may affect memory recall by reducing risk for such chronic conditions as stroke.

## **2.5 Life Style Changes**

Life-style changes such as the cessation of smoking or the change to a low-fat diet may account for some of the recent improvements in elderly health. Vita *et al.* (1998) find that such modifiable health risks as smoking, BMI, and exercise suggest that not only do persons with better health habits survive longer, but that disability is postponed and compressed into fewer years at the end of life. Ewbank and Preston (1990) and Mokyry and Stern (1996) emphasize increased knowledge of the germ theory of disease in reducing infectious disease mortality in the first half of the twentieth century. Those with chronic diseases (and therefore those at greater risk) may in particular be making better choices. Kahn (1999) shows that diabetics are making better choices relative to earlier cohorts and non-diabetics in terms of smoking and the consumption of foods rich in cholesterol, of alcohol, and of sweets. Progress, however, need not necessarily be

continuous. The increase in smoking from the late 1900s through the first half of the twentieth century probably contributed to increased morbidity and mortality rates.

Migration and technology have also affected life-styles. The move in the United States toward warmer winter climates may have increased exercise among the elderly and reduced their chances of falls. Assistive devices, such as grip bars and walkers, can reduce disability even if chronic disease rates remain unchanged.

## **2.6 Rising Incomes**

Rising incomes have enabled individuals to consume better food, housing, sanitation, and medical care. For example, crowding was associated with rheumatic fever and therefore valvular heart disease. Tuberculosis and other respiratory diseases spread in more crowded conditions. Barker and Lackland (2003) emphasize that areas of England and Wales with high stroke mortality were characterized in the past by poor living conditions, including high infant and maternal mortality and short stature of the adult population. Case (2001) finds that in South Africa the extension of generous pensions to elderly blacks led to improved self-reported health status and decreased depression for all household members because richer households were able to obtain on-site water and flush toilets and were less likely to skip meals.

The relation between health and income is a dual one. Poor health leads to lower earnings and greater health expenditures (Smith 1999). Case, Fertig, and Paxson (2003) and Case, Lubotsky, and Paxson (2003) emphasize that economic status in childhood determines health in adulthood – those born into poorer families experience poorer

childhood health, poorer health in early adulthood, and lower educational attainment, all of which are associated with lower earnings in middle age and the child rearing years.

## **2.7 Rising Education**

Many studies find evidence of a strong positive relationship between education and adult health, regardless of whether the evidence is mortality differentials (Kitagawa and Hauser, 1973; Elo and Preston, 1995; 1996), broader measures of health (Ross and Wu, 1995; 1996; Leigh and Fries 1994), or cognitive functioning at older ages. There is a strong relationship between health and education for the oldest age groups, although the association diminishes with age in some (Elo and Preston, 1996; Kitagawa and Hauser, 1973) and increases in others (Ross and Wu, 1996). Kahn (1998a) finds that education has an impact on diabetic health investment. Education may be related to health either as an indicator of income, of more positive early life experiences (including early preventive care), of patience or ability to delay gratification (and thereby avoid high risk behaviors such as smoking and excessive drinking), of greater knowledge and better ability to process information (and therefore avoid high risk habits), and of reduced stress and environmental exposure. Freedman and Martin (1999) find that education was the most important demographic and socioeconomic determinant of disability declines in the 1980s. Although Freedman and Martin (1999) do not find that the relationship between education and disability has changed, over the long-run education has probably become more important. Ewbank and Preston (1990) argue that infant mortality differentials by social class increased beginning in the 1920s because knowledge of the germ theory of

disease first spread to the professional classes. In recent data, education, not income is a better predictor of health. For example, Rosenzweig and Wolpin (1991) argue that even if parental incomes were equalized, most inequality in birth weights would remain. Meara (2001) finds that a limited set of maternal health habits during pregnancy, particularly smoking habits, can explain about half of the correlation between socioeconomic variables and birth weight. She argues that most of the differences between health habits and socioeconomic factors are due to such “third” variables as time preference, but that knowledge differences and differential responsiveness to common knowledge play a role as well.

### **3. Evaluating the Explanations**

Evaluating which environmental factors have contributed the most to the observed declines in morbidity and mortality remains an active research agenda. Increases in education and in income, changes in life styles, improvements in nutritional intake, reductions in occupational stress, declines in infectious disease rates, and improvements in medical care are all related to each other, have lagged effects, and all changed dramatically within a very short period of time. Those who were 70 in 1980 were born in 1910 when infectious disease rates were still high and when incomes were low and spent their prime years in relatively dangerous jobs. In contrast, those who were 70 in the year 2000 were born in 1930 when infectious disease rates, though still high by today’s standards, had fallen. They enjoyed higher incomes, improvements in food distribution that allowed them to eat a more balanced diet, more education, less dangerous jobs, and

improved medical care. Perhaps the only way to disentangle the effects of the various factors would be through the use of longitudinal datasets that allow us both to relate health factors to older age health and longevity and that allow us to determine how the relationship between health factors and older age health and longevity has changed.

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