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CHANGES IN THE DISPARITIES IN CHRONIC DISEASE DURING THE COURSE OF THE TWENTIETH CENTURY

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Changes in the Disparities in Chronic Disease during the Course of the Twentieth Century
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ABSTRACT

Longitudinal studies support the proposition that the extent and severity of chronic conditions in middle and late ages are to a large extent the outcome of environmental insults at early ages, including in utero. Data from the Early Indicators program project undertaken at the Center for Population Economics suggest that the range of differences in exposure to disease has narrowed greatly over the course of the twentieth century, that age-specific prevalence rates of chronic diseases were much lower at the end of the twentieth century than they were at the beginning of the last century or during the last half of the nineteenth century, and that there has been a significant delay in the onset of chronic diseases over the course of the twentieth century. These trends appear to be related to changes in levels of environmental hazards and in body size. These findings have led investigators to posit a synergism between technological and physiological improvements. This synergism has contributed to reductions in inequality in real income, body size, and life expectancy during the twentieth century.

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The main proposition of my talk today is that the extent and severity of chronic conditions in middle and late ages are to a large extent the outcome of environmental insults at early ages, including *in utero*. Chronic diseases at middle and late ages are due to a large extent to the toll taken by infectious diseases at young ages. Evidence to support this proposition comes partly from the study being conducted here at Chicago and elsewhere on the aging of the Union Army Veterans, and I'll refer to that study as the Early Indicators Project (EI). But it is also supported by other longitudinal studies in the U.S. and abroad.

Some recent studies indicate two-generation effects among humans. Perhaps the most widely case involves women who were born or were in gestation during the Dutch famine, which lasted about eight or nine months toward the end World War II and was precipitated by sharp reductions in rations. Although this famine resulted in an elevated perinatal death rate, it was for a long time thought that women who survived and were subsequently well-fed were just as healthy as women who were born before the famine or afterwards. However when these famine women began giving birth, their children had elevated perinatal death rates. Of course we will still have to wait to see what the effects of the famine will be on the rate of increase in chronic conditions and on longevity at older ages. However, there is evidence that children of mothers who were in the first trimester of gestation when the famine struck and who survived infancy have elevated prevalence of coronary heart disease now that they are in their mid-fifties (Roseboom et al. 2000).

Environmental conditions were far more severe in 1900 than in recent decades. There are many ways this can be measured. One of them is the infant death rate. Figure !

presents some results of an ongoing study of changes in disparities in infant mortality during the twentieth century by the neighborhoods of the 24 largest U.S. cities in 1900. At the present time we are using wards averaging about 30,000 people as the unit of observation. But such large units tend to mask disparities since they tend to combine both rich and poor neighborhoods. We hope eventually to conduct the analyses at the level of census districts, which average about 8,000 individuals, and are more homogenous with respect to socioeconomic status and health conditions than wards.

However, even when the analysis is conducted at the ward level, the results are striking, as is shown by Figure 1. In this figure, the y-axis is the infant death rate, and the x-axis is year. There are three sets of observations: The maximum infant death rate across about 120 wards of six large cities are clustered around the top line; the minimum infant death rates are clustered around the bottom line; and the line in the middle represents the average. The main point of this figure is how wide the range in infant death rates were at the beginning of the period, 226 per thousand between the best and the worst wards in 1900. But by 1950 the differential between the best and the worst wards had declined to about 38 per thousand. During the first half of the twentieth century the spread between the best and the worst wards diminished by 83 percent.

Hence, the first point to be noted about changes and disparities in the twentieth century is that the range of differences in exposure to disease has narrowed greatly over the course of the century. The disparities we observe today are much smaller than they used to be. From the perspective of 1900 we are currently engaged in fine-tuning, not in gross corrections of disparities.

Many examples of how much wider disparities were among ethnic groups can be presented. One example is from a paper by Todd Bridges (2002). The infant death rate from diarrheal diseases during the summer months in Chicago in 1910 for foreign born mothers was 9 times as high as the rate for native born mothers (Bridges 2003).

I began this talk by focusing on infectious diseases, although my assignment is the explanation for the decline in chronic diseases, because one of the principal findings of the Early Indicators Project is that insults from infectious diseases at early ages has a large impact on the prevalence rates of chronic diseases and disabilities in middle and late ages.

Another major finding of the early indicators project is that age-specific prelevance rates of chronic diseases were much lower at the end of the twentieth century than they were at the beginning of that century or during the last half of the nineteenth century. The Union Army data reveal the ubiquity of chronic health conditions during the century before World War II. Not only was the overall prevalence rate of these diseases much higher among the elderly than today, but they afflicted the teens, young adults and middle ages to a much greater extent than today. This fact is brought out by Table 1, which shows that more than 80 percent of all males aged 16–19 in 1861 and more than 70 percent of men ages 20–24 were examined for the Union Army. These examinees were overwhelmingly volunteers (less than 4 percent were drafted), who presumably thought they were fit enough to serve. Yet disability rates were higher than today. Even among teenagers more than one of six was disabled, and among men aged 35–39, more than half were disabled. Despite their relatively young ages, cardiovascular diseases (mainly rheumatic) accounted for 11 percent of the rejections; hernias another 12 percent; eye, ear, and nose diseases 7 percent; tuberculosis and other respiratory diseases 7 percent; tooth and gum diseases 8 percent. Most of the other rejections were due to orthopedic conditions and general debility (Lee 2001).

These findings about the early onset of chronic diseases cast new light on the debate about the effect of increased longevity on the prevalence rates of chronic diseases. Those who argued that the effect of increased longevity was to increase the average duration of chronic disease assumed no delay in the average age of onset of these diseases. They were also influenced by cross-sectional evidence that showed some increases in disability rates during the 1970s and 1980s, despite the continuing decline in mortality rates (Riley 1990, 1991; Wolfe and Haveman 1990). It seemed plausible that various health interventions and environmental changes served to reduce the severity of diseases and thus delayed death without providing cures, as has been the case with AIDS.

However, as Table 2 shows, there has been a significant delay in the onset of chronic diseases during the course of the twentieth century. Men age 50–54 were 24 percent more likely to be free of chronic conditions in 1994 than a century earlier. At age 60–64, white males today are two-and-a-half times more likely to be free of chronic diseases than their counterparts a century ago. Further light is shed on the issue by considering specific diseases (see Table 3). Arthritis began 11 years later among men who turned 65 between 1983 and 1992 than those turned age 65 between 1895 and 1910. The delay in the onset of a chronic condition was about 9 years for heart diseases, about 11 years for respiratory diseases (despite much higher rates of cigarette smoking), and nearly 8 years for neoplasms.[1]

Union Army veterans who endured poor health did not typically die quickly. Veterans who lived to be at least age 50, and who entered the pension system before age 51, lived an average of 24 years past age 50. Moreover, at their last examination on or before age 51, their average degree of disability was 58 percent, where 100 percent indicates complete incapacity for manual labor. Between age 50 and 60 disability ratings (controlled for age at death) continued to rise sharply, and then increased at a decreasing rate. It is worth noting that of the veterans who lived to be age 50, about 29 percent lived to be age 80 or more. For these "Old Old," the level of disability for manual labor averaged between 85 and 100 percent for a decade or more. Indeed, some survived with such high levels of disability for as much as a quarter of a century (Helmchen 2003). As Table 4 shows, survivors usually acquired more and more comorbidities as they aged.[2] Those who lived past age 85 had twice as many comorbidities as those who died by age 55.

Consideration of the sweep of the twentieth century puts the debate over the relationship between the increase in life expectancy and the change in the burden of chronic disease among the elderly in a new perspective. It now appears that the decline in morbidity rates paralleled the decline in mortality rates. Indeed, the delay in the onset of chronic disabilities between 1900 and the 1990s for those who lived to age 50 was greater than the increase in life expectancy at age 50 over the same periods. The average delay in the onset of chronic conditions over the century was more than 10 years (Helmchen 2003).[3] However, the average increase in male life expectancy was about 6.6 years (Bell, Wade, and Goss 1992).

Public health policies before 1940 had a large impact on the decline in chronic disability rates decades later. Dora Costa (2000) has estimated the impact of public health and socioeconomic status factors at late developmental and young adult ages on risks of incurring chronic conditions at middle and late ages. Significant predictors included mortality rates in counties of enlistments, infectious diseases experienced during the Civil War, and being a prisoner of war. She focused on a set of chronic conditions for which clinical diagnoses were essentially the same in the early 1900s as today (such as lower back pain, joint problems, decreased breath or adventitious sounds, irregular pulse, and valvular heart disease). This procedure permitted her to estimate how much of the observed decline in the prevalence rates of comparable conditions was due to the reduction in specific risk factors. Prevalence rates for 1971–1980 were computed from the National Health and Nutrition Examination Survey (NHANES).

She found that elimination of exposure to specific infectious diseases during developmental and young adult ages explained between 10 and 25 percent of the declines in the specified chronic diseases of middle and late ages between 1900–1910 and 1971–1980. Occupational shifts were also important, accounting for 15 percent of the decline in joint problems, 75 percent of the decline in back pains, and 25 percent of the decline in respiratory diseases.

Costa (2002) pushed this line of analysis further by documenting the decline in functional limitations among U.S. men between ages 50 and 74 over the course of the twentieth century. A central issue is the factoring of the decline in functional limitations among three processes: the decline in the prevalence rates of specific chronic diseases, the reduction in the debilitating sequelae of these diseases, and the influence of new medical technologies that relieve and control the sequelae. Her analysis turned on five functional limitations: difficulty walking, difficulty bending, paralysis, blindness in at least one eye, and deafness in at least one ear. Prevalence rates of these limitations among men aged 50–74 were computed for the Union Army, NHANES (1988–1994) and NHIS (1988–1994).

On average these five functional limitations declined by about 40 percent during the course of the twentieth century. Using probit regressions, Costa attributed 24 percent of the decline to reduction in the debilitating effect of chronic conditions and 37 percent to the reduced rates of chronic conditions.

The Significance of Changes in Body Size

The contribution of improvements in body size as measured by stature, BMI, and other dimensions have run through the research of the Early Indicators project like a red line. The discovery of correlations in time series going back to the colonial period between changes in stature and changes in life expectancy for the U.S. were reported first in 1986, although it was known as early as 1978.[4] Pursuit of a variety of issues called attention to the significance of changes in body size to the long-term decline in chronic conditions and mortality. For example, Diane Lauderdale and Paul Rathouz (1999) sought to investigate the impact of unhealthy environments on the genetic component of height. They hypothesized that an unhealthy environment might attenuate the effects of genotype. To test that hypothesis they constructed a sample of brothers who served in the Union Army. Their analysis showed that brothers from unhealthy counties had both higher variances in height and lower covariance in the heights of siblings than was expected from standard equations for measuring genetic influences in the heights of siblings. Study of the likelihood of developing specific diseases while in the service also pointed to the importance of stature. For example, short recruits were more likely to develop tuberculosis while in service than taller ones (Birchenall 2003. Cf. Lee 1997).

In 1995 Dora Costa discovered a sample of 23,000 Union Army recruits who were, for scientific reasons, more intensively examined than the typical recruit (Costa, forthcoming). Benjamin A. Gould, a leading astronomer and one of the founders of the National Academy of Sciences, who was in charge of the project, collected information on waist and hip circumference, lifting strength, vital capacity of lungs, height, weight, shoulder breadth, and chest circumference. The sample covered whites, blacks, and Native Americans. Costa linked a subsample of 521 white recruits who survived to 1900 to their pension records. She also compared the Union Army soldiers with soldiers measured in 1946–47, 1950, and 1988.

Over a span of one hundred years men in the military became taller and heavier. Their height increased by 5 cm and the BMIs of men aged 31–35 increased from 23 to 26. Controlling for BMI and age, the waist-hip and chest-shoulder ratios (both measures of abdominal fat) were significantly greater in the Gould sample than in the 1950 and 1988 samples.

Using an independent competing risk hazard model to estimate the effect of changes in body shape on the risk of death from cerebrovascular and ischemic heart disease at older ages, she found that a low waist to hip ratio increased mortality by 4.4 times relative to the mean and controlling for BMI, while a high waist-hip ratio increased mortality risk by 2.9 times. Substituting the characteristics of soldiers in 1950, who reached age 65 or over during the late 1980s, into her regression model produced a 15 percent decline in all cause mortality above age 64, implying that changes in frame size explain about 47 percent of the total decline in all cause mortality at older ages between the beginning and the end of the twentieth century.

The Theory of Technophysio Evolution

Recognition of environmentally induced changes in human physiology during the twentieth century that had a profound impact on the process of aging did not become apparent to EI investigators until mid-1993. The key finding was that prevalence rates in the main chronic diseases among Union Army veterans age 65 and older were much higher in 1910 than among veterans of World War II of the same ages during the mid-to-late 1980s. That finding was first set forth in a 1993 working paper and was elaborated and subsequently characterized as a “theory of technophysio evolution.” The theory of technophysio evolution arose out of intense discussion among the senior investigators, consultants, and research assistants during 1993–1994, with the physicians providing much of the intellectual leadership. This theory points to the existence of a synergism between technological and physiological improvements that has produced a form of human evolution that is biological but not genetic, rapid, culturally transmitted, and not necessarily stable. The process is still ongoing in both rich and developing countries.

Unlike the genetic theory of evolution through natural selection, which applies to the whole history of life on earth, technophysio evolution applies only to the last 300 years of human history, and particularly to the last century.[5] The theory of technophysio evolution rests on the proposition that during the last 300 years, particularly during the last century, human beings have gained an unprecedented degree of control over their environment—a degree of control so great that is sets them apart not only from all other species, but also from all previous generations of *Homo sapiens*. This new degree of control has enabled *Homo sapiens* to increase its average body size by over 50 percent, to increase its average longevity by more than 100 percent, and to greatly improve the robustness and capacity of vital organ systems.[6]

Figure 2 helps to point up how dramatic the change in the control of the environment after 1700 has been. During its first 200,000 or so years, *Homo sapiens* increased at an exceedingly slow rate. The discovery of agriculture about 11,000 years ago broke the tight constraint on the food supply imposed by a hunting and gathering technology, making it possible to release between 10 and 20 percent of the labor force from the direct production of food and also giving rise to the first cities. The new technology of food production was so superior to the old one that it was possible to support a much higher rate of population increase than had existed prior to c. 9000 B.C. Yet, as Figure 2 shows, the advances in the technology of food production after the *second* Agricultural Revolution (which began about A.D. 1700) were far more dramatic than the earlier breakthrough, since they permitted the population to increase at so high a rate that the line of population appears to explode, rising almost vertically. The new technological breakthroughs in manufacturing, transportation, trade, communications, energy production, leisure-time services, and medical services were in many respects even more striking than those in agriculture. Figure 2 emphasizes the huge acceleration in both population and technological change during the twentieth century. The increase in the world's population between 1900 and 1990 was four times as great as the increase during the whole previous history of humankind.

The Contribution of Technophysio Evolution to the Remarkable Reduction in Inequality during the Twentieth Century

The twentieth century contrasts sharply with the record of the two preceding centuries. In every measure that we have bearing on the standard of living, such as real income, homelessness, life expectancy, and height, the gains of the lower classes have been far greater than those experienced by the population as a whole, whose overall standard of living has also improved.

The "Gini ratio," which is also called the "concentration ratio," is the measure of the inequality of the income distribution most widely used by economists.[7] This measure varies between 0 (perfect equality) and 1 (maximum inequality). In the case of England, for example, which has the longest series of income distributions, the Gini ratio stood at about 0.65 near the beginning of the eighteenth century, at about 0.55 near the beginning of the twentieth century, and at 0.32 in 1973**,** when it bottomed out, not only in Britain, but also in the United States and other rich nations.[8] This measure indicates that over two-thirds of the reduction in the inequality of income distributions between 1700 and 1973 took place during the twentieth century. The large decrease in such inequality, coupled with the rapid increase in the average real income of the English population, means that the per capita income of the lower classes was rising much more rapidly than those of the middle or upper classes.[9]

A similar conclusion is implied by the data on life expectancies. For the cohort born about 1875, there was a gap of 17 years between the average length of life of the

British elite and of the population as a whole. There is still a social gap in life expectancies among the British, but today the advantage of the richest classes over the rest of the population is only about 4 years. Thus about three-quarters of the social gap in longevity has disappeared. As a consequence, the life expectancy of the lower classes increased from 41 years at birth in 1875 to about 74 years today, while the life expectancy of the elite increased from 58 years at birth to about 78 years. If anything sets the twentieth century apart from the past, it is this huge increase in the longevity of the lower classes.[10]

Data on stature also indicate the high degree of inequality during the nineteenth century. At the close of the Napoleonic wars, a typical British male worker at maturity was about 5 inches shorter than a mature male of upper class birth. There is still a gap in stature between the workers and the elite of Britain, but now the gap is only on the order of an inch. Height differentials by social class have virtually disappeared in Sweden and Norway but not yet in the United States. Statistical analysis across a wide array of rich and poor countries today shows a strong correlation between stature and the Gini ratio (Steckel 1995; Rona, Swan, and Altman 1978).

Weight is another important measure of inequality. Despite the great emphasis in recent years on weight reduction, the world still suffers more from undernutrition and underweight than from overweight, as the World Health Organization has repeatedly pointed out. Although one should not minimize the afflictions caused by overnutrition, it is important to recognize that even in rich countries such as the United States, undernutrition remains a significant problem, especially among impoverished pregnant women, children, and among the aged.

The secular increase in body builds is due primarily to the great improvement in socioeconomic conditions over the past several centuries, rather than to genetic factors, as can be seen by considering Holland. The average height of young adult males was only 64 inches in that country during the middle of the nineteenth century. The corresponding figure today is about 72 inches. An increase of 8 inches in just four generations cannot be due to natural selection or genetic drift because such processes require much longer time spans. Nor can it be attributed to heterosis (hybrid vigor) because Holland's population has remained relatively homogeneous and because the effects of heterosis in human populations have been shown both empirically and theoretically to have been quite small. It is hard to come up with credible explanations for the rapid increase in heights that do not turn on environmental factors, especially improvements in nutrition and health. These environmental factors appear to be still at work. Stature is still increasing, although at a somewhat slower rate, and nations have not yet reached a mean height that represents the biological limit of humankind under current biomedical technology (Fogel 1992; cf. van Wieringen 1986; Drukker 1994; Drukker and Tassenaar 1997; Schmidt, Jorgenson, and Michaelsen 1995).

Homelessness is another indicator of the dramatic reduction in inequality during the twentieth century. Down to the middle of the nineteenth century, between 10 and 20 percent of the population in Britain and on the Continent were homeless persons whom officials classified as vagrants and paupers. Estimates of vagrancy and pauper rates in the United States during the nineteenth century are less certain, but these rates appear to have reached European levels in the major cities during the middle decades of that century. When we speak of homelessness in the United States today, we are talking about rates below 0.4 percent of the population. Many of the homeless today are mentally ill individuals prematurely released from psychiatric institutions that are inadequately funded. Many others are chronically poor and inadequately trained for the current job market (Cipolla 1980; Laslett [1965] 1984; Himmelfarb 1983; Soltow 1968; Lindert and Williamson 1982; Fogel 1987, 1989, 1993; Colquhoun 1814; Hannon 1984a, 1984b, 1985; Jencks 1994).

The relatively generous poverty program developed in Britain during the second half of the eighteenth century, and the bitter attacks on that program by Malthus and others, have given the unwarranted impression that government transfers played a major role in the secular decline in beggary and homelessness. Despite the relative generosity of English poor relief between 1750 and 1834, beggary and homelessness fluctuated between 10 to 20 percent. Despite the substantial reduction in the proportion of national income transferred to the poor as a result of the poor laws of 1834 and later years, homelessness declined sharply during the late nineteenth and early twentieth centuries.

The fact is that government transfers were incapable of solving the problems of beggary and homelessness during the eighteenth and much of the nineteenth centuries, because the root cause of the problems was chronic malnutrition. Even during the most generous phases of the relief program, the bottom fifth of the English population was so severely malnourished that it lacked the energy for adequate levels of work (Fogel 1997; Fogel, Floud and Harris n.d.).

At the end of the eighteenth century British agriculture, even when supplemented by imports, was simply not productive enough to provide more than 80 percent of the potential labor force with enough calories to sustain regular manual labor. It was the huge increases in English productivity during the later part of the nineteenth and the early twentieth centuries that made it possible to feed even the poor at relatively high caloric levels. Begging and homelessness were reduced to exceedingly low levels, by nineteenth-century standards, only when the bottom fifth of the population acquired enough calories to permit regular work. The principal way in which government policy contributed to that achievement was through its public health programs. By reducing exposure to disease, more of the calories that the poor ingested were made available for work.

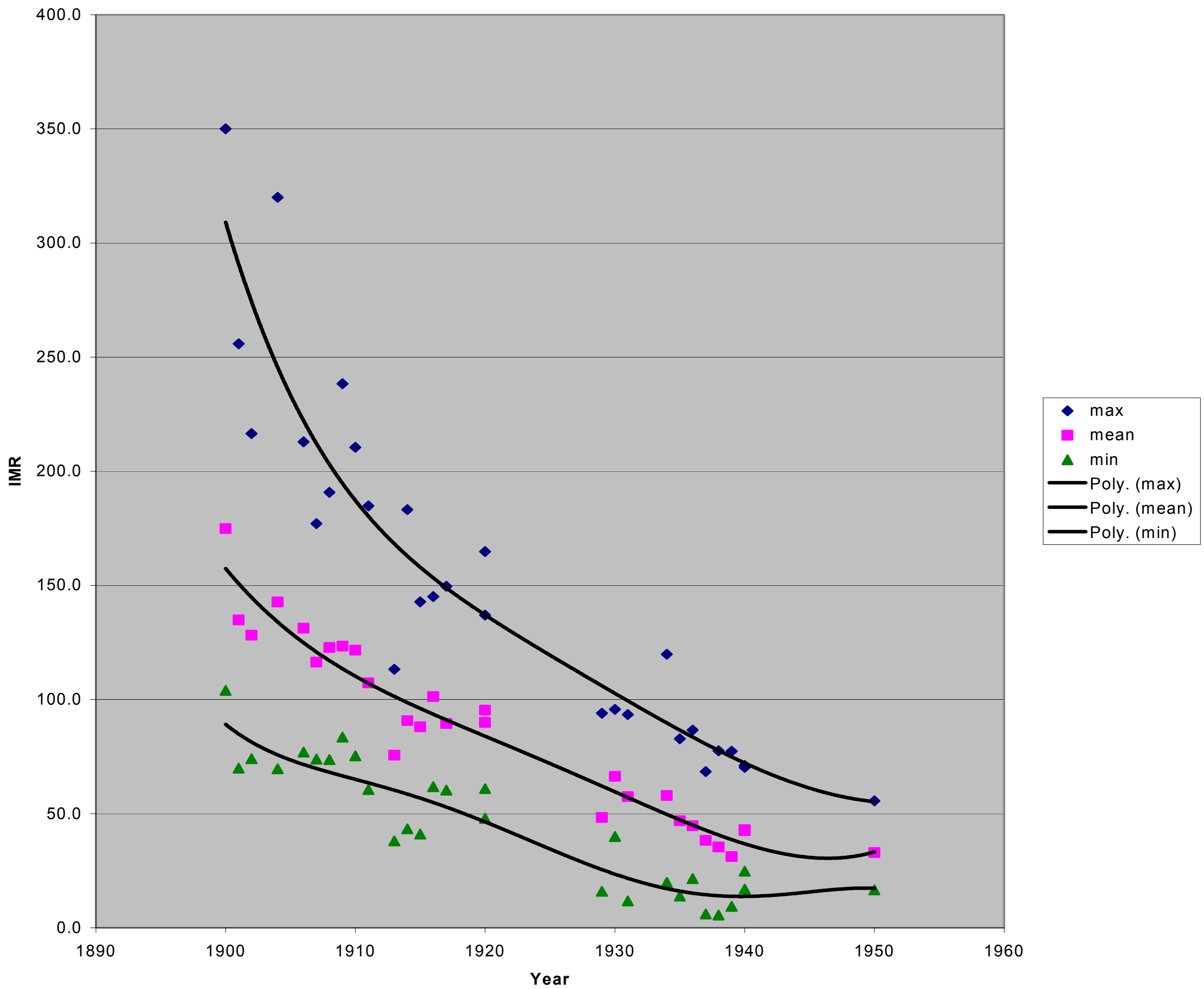
Concluding Comment

Technophysio evolution implies that some theoretical propositions that underlie some current economic models are misspecified because the initial endowments of health capital of successive cohorts increased over the course of the twentieth century. The common assumption that the endowment of human physiological capacity is fixed, so that medical intervention can only slow down the rate of deterioration in the original endowment, means that ways of forecasting future improvement in human physiology are sometimes neglected and possible paths of increase in health endowments play little role in forecasting future health care costs or longevity.[11]

The theory of technophysio evolution implies that health endowments in a given population change with the year of birth. It also points to complex interactions between date of birth and the outcome of exposures to given risk factors. Hence, not all improvements in the outcome of exposure to health risks between, say, 1970 and 1990 are due to health interventions during that period. Improvements in life expectancy may depend only partly on the more effective medical technologies of those years. It could also reflect the improved physiologies experienced by later birth cohorts that are due to improved technologies in food production, public health practices, personal hygiene, diets, and medical interventions put into place decades before 1970, and hence cannot be attributed exclusively, perhaps even primarily, to health inputs between 1970 and 1990.[12] Moreover, many health interventions which are effective at late ages today would not be feasible if the level of physiological capital at late ages was as low today as it was at the beginning of the twentieth century.

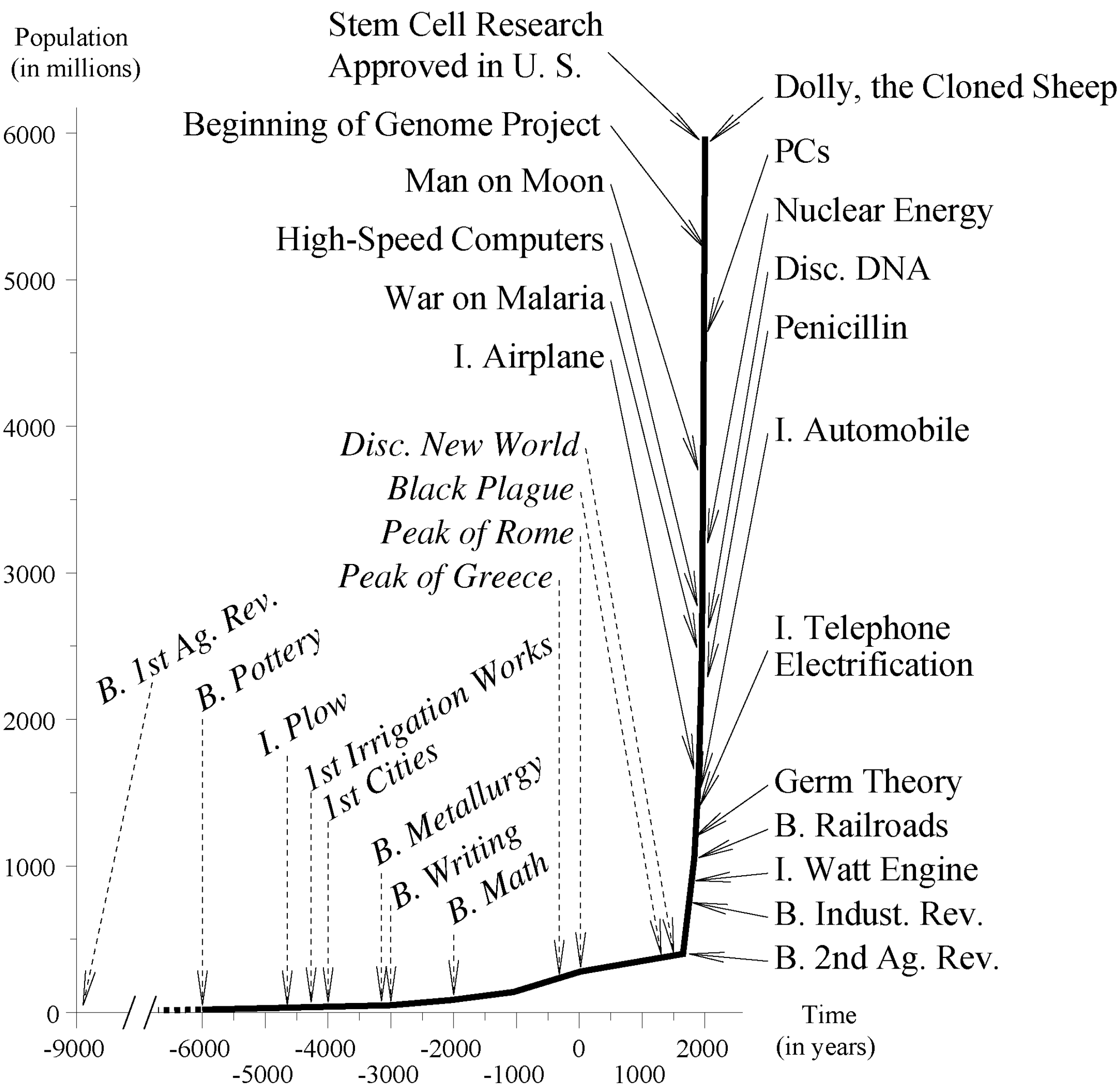
Figure 1

Preliminary Findings on Trends in Disparities in Infant Death Rates of the Wards of 6 Large U.S. Cities, 1900-1950



Source: Center for Population Economics website (www.uchicago.edu)

Figure 2
The Growth of World Population and Some Major Events in the History of Technology



Sources: Cipolla 1974; Clark 1961; Fagan 1977; McNeill 1971; Piggott 1965; Derry and Williams 1960; Trewartha 1969. See also Allen 1992, 1994; Slicher van Bath 1963; Wrigley 1987.

Note: There is usually a lag between the invention (I) of a process or a machine and its general application to production. “Beginning” (B) usually means the earliest stage of this diffusion process.

Table 1

The Share of Northern White Males of Military Age Unfit for Military Service in 1861

Age	Percentage of Cohort Examined	Percentage of Examinees Who Were Rejected
16–19	80.9	16.0
20–24	70.4	24.5
25–29	52.3	35.8
30–34	41.0	42.9
35–39	41.6	52.9

Table 2

The Increase in the Proportion of White Males without Chronic Conditions during the Twentieth Century

	Proportion without Chronic Conditions	
Age Interval	1890–1910	c. 1994
50–54	**0.33**	**0.41**
55–59	**0.21**	**0.29**
60–64	**0.10**	**0.25**
65–69	**0.03**	**0.14**

Table 3

Average Age of Onset of Some Chronic Conditions among American Males near the Beginning and near the End of the Twentieth Century

Condition	Men Born 1830–1845	Men Born 1918–1927
Heart disease	55.9	65.4
Arthritis	53.7	64.7
Neoplasm	59.0	66.6
Respiratory	53.8	65.0

Table 4

Average Number of Comorbidites among Veterans Who Lived To Be at Least Age 50

Average Age of Death	Percentage of Veterans Who Lived To at Least Age 50, Who Died in Interval	Average Number of Comorbidities at Last Examination before Death
50–54	3.9	4
55–59	6.4	5
60–64	9.8	6
65–69	14.0	6
70–74	18.3	7
75–79	19.1	7
80–84	15.5	7
85-89	9.0	8
90-94	3.4	8
95 or over	0.7	7

Notes

1. Since current diagnostic techniques make it possible to diagnose heart disease and neoplasms sooner in the development of these diseases than used to be the case c. 1910, the figures given in the text should be considered lower bounds on the delay in the onset of these conditions.
2. Comorbidity: the coexistence of two or more disease processes.
3. The delay in the average age of onset in chronic diseases can be decomposed into two parts: (1) the shift in the age-specific disease schedule; (2) the change in the distribution of ages due to the increase in life expectancy and the decline in the fertility rate. We have not yet completed this decomposition. However, preliminary estimates indicate that the contribution of the change in the age distribution was small.
4. For reviews of earlier work dealing with the use of height, BMI and other anthropometric measures as indexes of changes in health and the standard of living over time, see Steckel 1995; Komlos and Cuff 1998.
5. Costa and Fogel limit technophysio evolution to the last 300 years for two reasons. It was not until about 1700 that changes in technology permitted population growth far in excess of previous rates. Moreover, after 1700 body weight and stature increase to unprecedented levels. See Figure 1 in Fogel and Costa 1997.
6. Although a considerable body of empirical evidence has accumulated indicating that a "good" environment both speeds up biological development at young ages and delays the onset of chronic conditions at middle and late ages, there is as yet

no agreed upon theory about the cellular and molecular processes that explains these observations.

7. Stature and Gini ratios are significantly correlated, but as the following discussion of height and BMI indicates, the anthropometric measures reveal important aspects of welfare that are not as apparent in the movement of Gini ratios.

8. There has been a rise in the Gini ratio since 1973 in virtually all the rich nations for which such information is available. Cf. Fogel 2000.

9. On trends in the Gini ratio between ca. 1690 and 1973 and the debate over this trend, see Soltow 1968; Feinstein 1988; Williamson 1985; Lindert and Williamson 1982, 1984; Floud, Wachter, and Gregory 1990. Cf. Fogel 2000.

10. Data for males are presented in Case et al. 1962; Hollingsworth 1977; Hattersley 1999.

11. Among the exceptions are Rosenzweig and Schultz 1988 and Dasgupta 1993.

12. Much recent research indicates that waiting time to the onset of chronic diseases is a function of exposure to insults *in utero* and infancy. See Barker 1998; Scrimshaw 1997.

Works Cited

Allen, R. C. 1992. *Enclosure and the yeoman: The agricultural development of the South Midlands, 1450-1850*. Oxford, Oxford University Press.

Allen, R. C. 1994. "Agriculture during the industrial revolution," in *The economic history of Britain since 1700*, vol. 1, *1700–1860*, Floud, R. and McCloskey, D. (eds.), 2d ed., pp. 96–122, Cambridge: Cambridge University Press.

Barker, D. J. P. 1998. *Mothers, babies and disease in later life.* Edinburgh and New York, Churchill Livingstone.

Bell, Felicitie C., Alice H. Wade, and Stephen C. Goss. 1992. *Life Tables for the United States Social Security Area 1900–2080*. Actuarial Study No. 107. Baltimore: U.S. Department of Health and Human Services, Social Security Administration, Office of the Actuary.

Birchenall, Javier A. 2003. "Airborne diseases: Tuberculosis in the Union Army." Paper presented at the Joint Meeting of the *Early Indicators of Later Work Levels, Disease, & Death* Program Project and Cohort Studies groups, National Bureau of Economics, 25–26 April 2003, Cambridge, MA.

Bridges, T. A. 2002. "Preliminary Findings on the Linkage Between Infant Mortality and Education during Early Twentieth Century Chicago." Working Paper, Center for Population Economics (http://www.cpe.uchicago.edu/publication/publication.html).

Case, R. A. M., C. Coghill, et al. 1962. *Chester Beatty Research Institute abridged serial life tables, England and Wales 1841-1960, Part 1.* London, Chester Beatty Research Institute.

Cipolla, C. M. 1974. *The economic history of world population*. Harmondsworth, Middlesex, UK, Penguin.

Cipolla, C. M. 1980. *Before the industrial revolution: European society and economy, 1000-1700*. New York, W. W. Norton.

Clark, G. 1961. *World prehistory: An outline*. Cambridge, Cambridge University Press.

Colquhoun, P. 1814. *Treatise on the wealth, power, and resources of the Bristish Empire.* London:Joseph Mawmay.

Costa, Dora L. 2000. "Understanding the twentieth-century decline in chronic conditions among older men," *Demography* 37(1): 53–72.

Costa, Dora L. 2002. "Changing chronic disease rates and long-term declines in functional limitation among older men," *Demography* 39(1): 119–137.

Costa, Dora L. Forthcoming. "The measure of man and older age mortality: Evidence from the Gould Sample," *Journal of Economic History*.

Dasgupta, Partha. 1993. *An Inquiry into Well-Being and Destitution*. Oxford: Clarendon.

Derry, T. K. and T. I. Williams. 1960. *A short history of technology*. London: Oxford University Press.

Drukker, J. W. 1994. "The tradition of anthropometric history in the Netherlands." Paper presented at the National Bureau of Economic Research, Cambridge, Mass., 11-12 July.

Drukker, J. W. and Tassenaar, V. 1997. "Paradoxes of modernization and material well-being in the Netherlands during the 19th century. In *Health and welfare during industrialization,* Steckel R. H. and Floud, R. (eds.), pp. 331-77. Chicago: University of Chicago Press.

Fagan, B. M. 1977. *People of the earth*, 2nd ed. Boston: Little, Brown.

Feinstein, C. H. 1988. "The rise and fall of the Williamson Curve." *Journal of Economic History* 48: 699-729.

Floud, R., K. W. Wachter, et al. 1990. *Height, health, and history: Nutritional status in the United Kingdom, 1750-1980.* Cambridge, Cambridge University Press.

Fogel, R. W. 1987, Biomedical approaches to the estimation and interpretation of secular trends in equity, morbidity, mortality, and labor productivity in Europe, 1750–1980, Typescript, Center for Population Economics, University of Chicago.

Fogel, R. W. 1989, *Without consent or contract*, vol. 1, New York: W. W. Norton.

Fogel, R. W. 1997. "Economic and social structure for an ageing population." *Philosophical Transactions of the Royal Society, ser. B., Biological Sciences* 352 (1363): 1905-17.

Fogel, R. W., Floud, R. and Harris, B. (n.d.), *A treatise on technophysio evolution and consumption.*

Fogel, Robert William and Dora L. Costa. 1997. "A theory of technophysio evolution, with some implications for forecasting population, health care costs, and pension costs," *Demography* 34(1): 49–66.

Fogel, Robert William. 1992. "Introduction: Notes on the art of empirical research in the social sciences during an age of plunging costs in data processing," in *Without Consent or Contract*, vol. 2, *Evidence and Methods*, eds. Robert William Fogel, Ralph A. Galantine, and Richard L. Manning. New York: W. W. Norton, pp. 1–41.

Fogel, Robert William. 1993. "New sources and new techniques for the study of secular trends in nutritional status, health, mortality, and the process of aging," *Historical Methods* 26(1): 5–43.

Hannon, J. U. 1984a, "The generosity of antebellum poor relief," *Journal of Economic History* 44: 810–21.

Hannon, J. U. 1984b, "Poverty in the antebellum northeast: The view from New York State's poor relief rolls," *Journal of Economic History* 44: 1007–32.

Hannon, J. U. 1985, "Poor relief policy in antebellum New York State: The rise and decline of the Poorhouse," *Explorations in Economic History* 22: 233–56.

Hattersley, L. 1999, "Trends in life expectancy by social class—an update." *Health Statistics Quarterly* 2: 16–24.

Helmchen, L. 2003. "Changes in the Pattern of Chronic Disease Among Elderly Americans, 1870 – 2000." Working Paper, Center for Population Economics (www.cpe.uchicago.edu).

Himmelfarb, G. 1983, *The idea of poverty: England in the early industrial age*, New York: Random House.

Hollingsworth, T. H. 1977. "Mortality in the British peerage families since 1600." *Population* 32 (Numéro special): 323–52.

Jencks, C. 1994. *The homeless*, Cambridge: Harvard University Press.

Komlos, John and Timothy Cuff, eds. 1998. *Classics in Anthropometric History*. St. Katharinen, Germany: Scripta Mercaturae Verlag.

Laslett, P. [1965] 1984. *The world we have lost: England before the industrial age.* New York, Charles Scribner's Sons.

Lauderdale, Diane S. and Paul J. Rathouz. 1999. "Evidence of environmental suppression of familial resemblance: Height among U.S. Civil War brothers," *Annals of Human Biology* 26(5): 413–426.

Lee, Chulhee. 1997. "Socioeconomic background, disease, and mortality among Union Army recruits: Implications for economic and demographic history," *Explorations in Economic History* 34(1): 27–55.

Lee, Chulhee. 2001. "Exposure to disease during growing ages and service," in *Early Indicators of Later Work Levels, Disease, & Death*, program project proposal submitted to the N. I. A. Typescript, Center for Population Economics, University of Chicago.

Lindert, P. H. and Williamson, J. G. 1982, Revising England's social tables: 1688–1812, *Explorations in Economic History* 19: 385–408.

McNeill, W. 1971. *A world history*, 2nd ed. New York: Oxford University Press.

Piggott, S. 1965. *Ancient Europe from the beginnings of agriculture to classical antiquity*. Chicago: Aldine.

Riley, J. C. 1991. "The prevalence of chronic diseases during mortality increase: Hungary in the 1980s." *Population Studies* 45(3): 489-96.

Riley, James C. 1990a. "Long-term morbidity and mortality trends: Inverse health transitions," in *What We Know about Health Transition: The Cultural, Social and Behavioural Determinants of Health, The Proceedings of an International Workshop, Canberra, May 1989*. Canbera: Health Transition Centre, Australian National University.

Riley, James C. 1990b. "The risk of being sick: Morbidity trends in four countries," *Population and Development Review* 16(3): 403–432.

Rona, R. J., A. V. Swan, et al. 1978. "Social factors and height of primary schoolchildren in England and Wales." *Journal of Epidemiology and Community Health* 32: 147-54.

Roseboom, T. J., van der Meulen, J. H. P., et al. 2000. "Coronary heart disease after prenatal exposure to the Dutch famine, 1944-45." *Heart* 84(6):595-598.

Rosenzweig, Mark R. and T. Paul Schultz. 1988. "The stability of household production technology: A replication," *Journal of Human Resources* 23(4): 535–549.

Schmidt, I. M., Jorgensen, M. H. and Michaelsen, K. F. 1995. "Height of conscripts in Europe: Is postneonatal mortality a predictor?" *Annals of Human Biology* 22: 57–67.

Scrimshaw, Nevin S. 1997. "More evidence that foetal nutrition contributes to chronic disease in later life." *BMJ* 315(7112): 825–826.

Slicher van Bath, B. H. 1963. *The agrarian history of Western Europe A.D. 500–1850*, Ordish, O. (trans.), London: Edward Arnold.

Soltow, L. 1968. "Long-run changes in British income inequality." *Economic History Review* 21: 17–29.

Steckel, Richard H. 1995. "Stature and the standard of living," *Journal of Economic Literature* 33(4): 1904–1940.

Trewartha, G. T. 1969. *A geography of populations: World patterns.* New York: John Wiley & Sons.

van Wieringen, J. C. 1986. "Secular growth changes." In *Human growth: A comprehensive treatise*, vol. 3, *Methodology. Ecological, genetic, and nutritional effects on growth*, Falkner, F. and Tanner, J. M. (eds.), 2d ed, pp. 307–71, New York: Plenum Press.

Williamson, J. G. 1982. "Was the industrial revolution worth it? Disamenities and death in 19th century British towns." *Explorations in Economic History* 19: 221-45.

Williamson, J. G. 1984. "British mortality and the value of life, 1781-1931." *Population Studies* 38: 157-72.

Williamson, J. G.1985. *Did British capitalism breed inequality*. Boston, Allen & Unwin.

Wolfe, Barbara L. and Robert Haveman. 1990. "Trends in the prevalence of work disability from 1962 to 1984, and their correlates," *Milbank Memorial Fund Quarterly* 68(1): 53–80.

Wrigley, E. A. 1969. *Population and history*. London: Weidenfeldt and Nicolson.