A THEORY OF TECHNOPHYSIO EVOLUTION, WITH SOME IMPLICATIONS FOR FORECASTING POPULATION, HEALTH CARE COSTS, AND PENSION COSTS*

ROBERT W. FOGEL AND DORA L. COSTA

We argue that over the past 300 years human physiology has been undergoing profound environmentally induced changes made possible by numerous advances in technology. These changes, which we call technophysio evolution, *increased body size by over 50%, and greatly improved the robustness and capacity of vital organ systems. Because technophysio evolution is still ongoing, it is relevant to forecasts of longevity and morbidity and, therefore, to forecasts of the size of the elderly population and pension and health care costs.*

Research during the past two decades has produced significant advances in the description and explanation of the secular decline in mortality. Although many of these findings are still tentative, they suggest a new theory of evolution that we call *technophysio evolution*. Study of the causes of the reduction in mortality point 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. This process is ongoing in both rich and developing countries. In the course of elaborating this theory, we will define thermodynamic and physiological aspects of economic growth and discuss their impact on economic growth rates.

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, particularly to the last century.[1] Despite its limited scope, technophysio evolution appears to be relevant to forecasting, over the next century or so, likely trends in longevity, the age of onset of chronic diseases, body size, and the efficiency and durability of vital organ systems. It also has a bearing on pressing issues of public policy such as the growth in population, in pension costs, and in health care costs.

The theory of technophysio evolution rests on the proposition that during the last 300 years, particularly during the last century, humans have gained an unprecedented degree of control over their environment—a degree of control so great that it 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%, to increase its average longevity by more than 100%, and to improve greatly the robustness and capacity of vital organ systems.

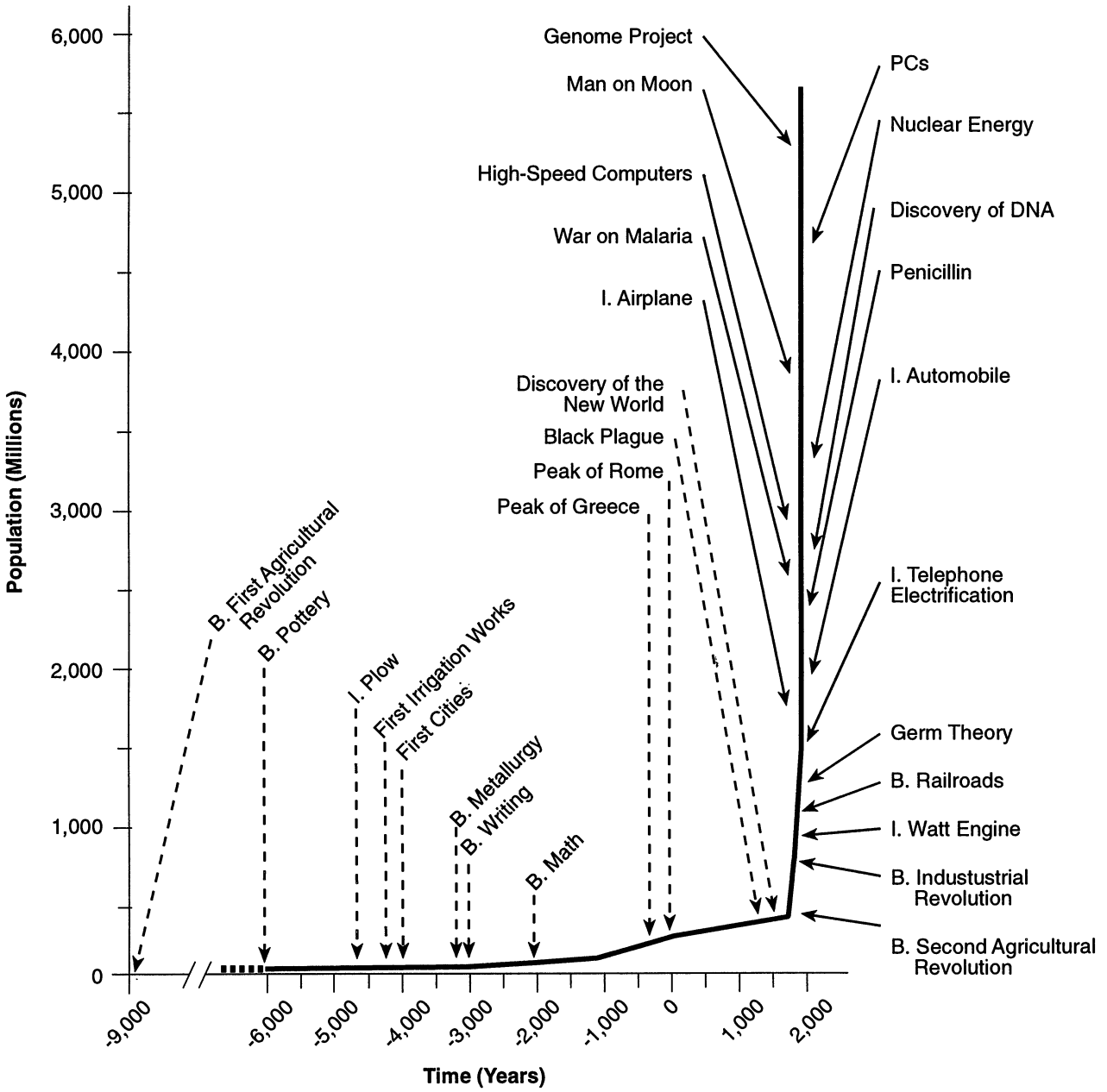
Figure 1 helps to point out how dramatic the change has been in the control of environment after 1700. During its first 100,000 or so years, *Homo sapiens* increased at an exceedingly slow rate. The discovery of agriculture about 11,000 years ago broke the tight constraint on the food supply imposed by a hunting and gathering technology, making it possible to release between 10% and 20% of the labor force from the direct production of food and 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 before about 9000 B.C. Yet, as Figure 1 shows, the advances in the technology of food production after the *second* Agricultural Revolution (about A.D. 1700) were far more dramatic than the earlier breakthrough, because 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 ser-

*Robert W. Fogel, University of Chicago, National Bureau of Economic Research, 1101 E. 58th Street, Chicago, IL 60637; e-mail: imok@tao.cpe.uchiago.edu. Dora L. Costa, Massachusetts Institute of Technology, National Bureau of Economic Research. Both authors gratefully acknowledge the support of NIH Grant AG10120-05 and NSF Grant SES-9114981. Dora Costa also has benefited from the support of a NIA Aging Fellowship at the National Bureau of Economic Research and of NIH Grant AG12658-01A1. We thank Samuel H. Preston and two anonymous referees for helpful comments.

1. In this paper we use *evolution* in two ways. The term *genetic evolution* refers to species change through natural selection among species with different genetic characteristics. We use the term *technophysio evolution* to refer to changes in human physiology brought about *primarily* by environmental factors. The environmental factors include those influencing chemical and pathogenic conditions of the womb in which the embryo and fetus develop. Such environmental factors may be concurrent with the development of the embryo and fetus or may have occurred before the conception of the embryo earlier in the life of the mother or higher up in the maternal pedigree. Experimental studies on animal models indicate that environmental insults in a first generation continue to have potency in retarding physiological performance over several generations despite the absence of subsequent insults; the potency of the initial insult, however, declines from one generation to another (Chandra 1975, 1992; Fraker et al. 1986; Meinhold et al. 1993).

We italicized *primarily* to indicate that the potency of particular environmental insults varies from one individual to another in a manner that might reflect complex interactions between environmental and genetic factors. We abstract from the uses of the term *evolution* in embryogenesis and in its use as a bridging function in the principle of recapitulation. On these meanings of the term *evolution* and its general history see Richards (1992) and Mayr (1982).

FIGURE 1. THE GROWTH OF THE WORLD POPULATION AND SOME MAJOR EVENTS IN THE HISTORY OF TECHNOLOGY



Sources: Cipolla (1974), Clark (1971), Fagan (1977), McNeill (1971), Piggott (1965), and Trewartha (1969). See also Allen (1992, 1994), Slicher van Bath (1963), and Wrigley (1987).

Notes: I = invention; B = beginning. There is usually a lag between the invention of a process or a machine and its general application to production. "B" is intended to identify the beginning, or the earliest stage, of this diffusion process.

vices, and medical services were in many respects even more striking than those in agriculture.

Although Figure 1 points to changes in technology that permitted a vast increase in population, it does not reveal a connection between technological changes and physiological benefits. Moreover, down to the beginning of World War I it was widely believed that the impact of industrialization on human physiology was negative. In England, for example, the large proportion of men rejected by recruiters for the Boer War set off an alarm among authorities that was exac-

erbated by data that seemed to show that recruits who reached maturity in about 1900 were shorter than those who had reached maturity at the time of the Crimean War in the mid-1850s (Floud and Wachter 1982). These findings seemed to be confirmed by studies reporting that 27% of the British population was living in such deep poverty that its consumption of food and other necessities was below the level needed to maintain physical efficiency (Bowley and Burnett-Hurst 1915; Rowntree 1901).

In the balance of this paper we briefly describe various new categories of evidence that bear on a theory of technophysio evolution. We begin with the new findings on the secular decline in mortality.

THE SECULAR DECLINE IN MORTALITY

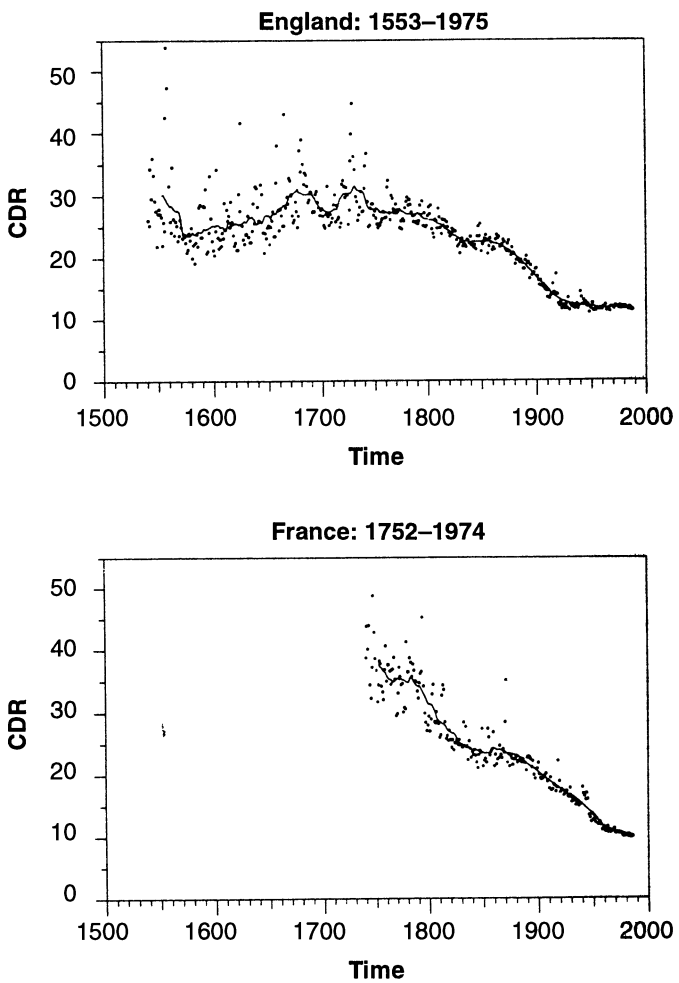
By the third decade of the twentieth century improved death registration made it obvious that the new declines in British mortality rates were not just a cyclical phenomenon. Between 1871 and 1901 life expectation in Britain increased by four years. During the next three decades there was an additional gain of 16 years. Similar declines in mortality were recorded in other European nations and in America (Case et al. 1962; Dublin and Lotka 1936; Dublin, Lotka, and Spiegel 1949; Gille 1949; Stolnitz 1955/1956, 1956/1957; United Nations 1953).

The plunge in mortality rates during the early decades of the twentieth century delivered a major blow to the Malthusian theory of population. Improvements in mortality were supposed to be short-lived because, under the conditions of population pressure against the food supply that Malthus specified, the elimination of deaths due to one disease would be replaced by those due to some other malady. Efforts to reconcile Malthusian doctrine with the observed mortality decline—to modify it or to replace it—produced a large new body of literature.

One aspect of the new research was a concerted effort to develop time series of death rates that extended as far back in time as possible to determine just when the decline in mortality began. Before the 1960s, such efforts were focused primarily on notable local communities and parishes. Developments in statistical techniques and the remarkable reductions in computational cost, however, made it possible to draw and process large nationally representative samples.

The results of these efforts are displayed in Figure 2. Analysis of the French and English series revealed that the secular decline in mortality took place in two waves. In the English case the first wave began during the second quarter of the eighteenth century and lasted through 1820, after which mortality rates stabilized for half a century. The decline resumed during the 1870s and continues through the present. The French case is similar except that the first wave of the decline in mortality began about half a century earlier in France and its rate of decline during the first wave was more rapid.[2]

FIGURE 2. THE SECULAR TRENDS IN MORTALITY RATES IN ENGLAND AND FRANCE



Note: Each diagram shows the scatter of annual death rates around a 25-year moving average.

Perhaps the most surprising aspect of Figure 2 is the implication that the elimination of crisis mortality, whether related to famines or not, accounted for less than 10% of the secular decline in mortality rates. By demonstrating that famines and famine mortality were a secondary issue in the escape from the high mortality rates of the early modern era, these studies shifted attention to the neglected issue of chronic malnutrition as the principal pathway through which malnutrition contributed to the high mortality rates of the past.

2. The time series for England and France and the data from which they have been computed are discussed in DuPâquier (1989), Fogel (1992b), Galloway (1986), Lee (1981), Richards (1984), Weir (1982), and Wrigley and Schofield (1981). For data on other European countries see Bengtsson and Ohlsson (1984) and Galloway (1987); cf. Eckstein, Schultz and Wolpin (1984), Fridlizius (1984), and Perrenoud (1984, 1991).

TABLE 1. ESTIMATED AVERAGE FINAL HEIGHTS (CM) OF MEN WHO REACHED MATURITY BETWEEN 1750 AND 1875 IN SIX EUROPEAN POPULATIONS, BY QUARTER CENTURIES

	(1) Date of Maturity, by Century and Quarter	(2) Great Britain	(3) Norway	(4) Sweden	(5) France	(6) Denmark	(7) Hungary
1.	18–III	165.9	163.9	168.1	—	—	168.7
2.	18–IV	167.9	—	166.7	163.0	165.7	165.8
3.	19–I	168.0	—	166.7	164.3	165.4	163.9
4.	19–II	171.6	—	168.0	165.2	166.8	164.2
5.	19–III	169.3	168.6	169.5	165.6	165.3	—
6.	20–III	175.0	178.3	177.6	172.0	176.0	170.9

Sources: For all countries except France see Fogel (1987: table 7). For France, rows 3–5 were computed from von Meerton (1989) as amended by Weir (1993), with 0.9 cm added to allow for additional growth between age 20 and maturity (Gould 1869: 104–105; cf. Friedman 1982:510, fn. 14). The entry to row 2 was derived from a linear extrapolation of von Meerton's data for 1815–1836 back to 1788, with 0.9 cm added for additional growth between age 20 and maturity. The entry in row 6 was taken from Fogel (1987: table 7).

THE SECULAR TREND IN FOOD SUPPLIES AND BODY SIZE

As a result of the work of agricultural historians we now have estimates of British agricultural production by half-century intervals dating back to 1700. These estimates provide the basis for national food balance sheets, which indicate that average daily caloric consumption in Britain around 1790 was about 2,060 kcal per capita or about 2,700 kcal per consuming unit (equivalent adult males ages 20–39). The corresponding figure for France was about 2,410 kcal per consuming unit.[3]

One implication of these estimates is that mature adults of the late eighteenth century must have been very small by current standards. Today the typical American male in his early thirties is about 177 cm (70 in.) tall and weighs about 78 kg (172 lb) (U.S. Department of Health and Human Services 1987). Such a male requires daily about 1,800 kcal for basal metabolism and a total of 2,300 kcal for baseline maintenance, which includes 500 kcal for digestion of food and for vital hygiene. If either the British or the French had been that large during the eighteenth century, virtually all of the energy produced by their food supplies would have been required for personal maintenance, and hardly any would have been available to sustain work. To have the energy necessary to produce the national products of these two countries around 1700, the typical adult male must have been quite short and very light.

This inference is supported by data on stature and weight that have been collected for European nations. Table 1 provides estimates of final heights of adult males who reached maturity between 1750 and 1875. It shows that during the eighteenth and nineteenth centuries Europeans were severely stunted by modern standards (cf. line 6 of Table 1). Estimates of weights for European nations before 1860 are much more patchy. Those that are available (mostly inferential) suggest that around 1790 the average weight of English males in their thirties was about 61 kg (134 lb), which is about 20% below current levels. The corresponding figure for French males around 1790 may have been only about 50 kg (110 lb), which is about a third below current standards.

Further insight into the extent of chronic malnutrition in Europe at the beginning of the nineteenth century is obtained by switching focus from the mean levels of caloric consumption to the size distributions of calories associated with these mean levels. Table 2 shows the exceedingly low level of work capacity permitted by the food supply in France and England around 1790, even after allowing for the reduced requirements for maintenance because of small stature and

3. We have not presented confidence intervals for the estimates of mean caloric intake in England and France around 1790 because the estimates are constructed from national aggregates, not samples. In the English and in other cases there are samples of household consumption, but the principal issue with these is not sampling error but systematic biases in reporting and in sample selection. Metabolic techniques of measuring daily caloric energy utilization, such as the doubly labeled water method, indicate that samples of food diaries today and other self reports understate actual food consumption by about 25% (Bingham 1994; Black et al. 1996; Schoeller 1990).

The means reported here are informed judgements based on the evaluation of numerous potential systematic errors in the data used to derive the estimates using techniques that are similar to those that have proven useful in national income accounting. The principal instrument in this work is sensitivity analysis, which focuses on the range of under- or overestimates of true means due to plausible errors in the data or in estimating procedures.

Another important technique is the establishment of a series of constraints that the constructed estimates must satisfy, such as the consistency between the estimates of agriculture output, of labor productivity in agriculture, and of the food energy required to produce that output. Fogel (1993) summarizes the procedures and presents examples of their application to the French and English cases. Fogel et al. (1996) discuss the procedures at greater length and report the results of these tests in the French, English, and American cases.

As indicated in these sources, the estimates presented here appear to be the best estimates based on the currently available data. Both the English and French estimates, however, may overstate the energy actually metabolized. Plausible upper and lower bounds of the reported figures for c. 1790 are about ±10%.

TABLE 2. A COMPARISON OF THE PROBABLE FRENCH AND ENGLISH DISTRIBUTION OF THE DAILY CONSUMPTION OF KCALS PER CONSUMING UNIT TOWARD THE END OF THE EIGHTEENTH CENTURY

	France around 1785		England around 1790	
	$\bar{X}$ = 2,290	($s/\bar{X}$ = 0.3)	$\bar{X}$ = 2,290	($s/\bar{X}$ = 0.3)
(1)	(2)	(3)	(4)	(5)
Decile	Daily kcal Consumption	Cumulative Percentage	Daily kcal Consumption	Cumulative Percentage
1. Highest	3,672	100	4,329	100
2. Ninth	2,981	84	3,514	84
3. Eighth	2,676	71	3,155	71
4. Seventh	2,457	59	2,897	59
5. Sixth	2,276	48	2,684	48
6. Fifth	2,114	38	2,492	38
7. Fourth	1,958	29	2,309	29
8. Third	1,798	21	2,120	21
9. Second	1,614	13	1,903	13
10. First	1,310	6	1,545	6

Source: Fogel (1987: tables 4 and 5 and note 6).

body mass. In France the bottom 10% of the labor force lacked the energy for regular work and the next 10% had enough energy for less than three hours of light work daily. The English situation was only slightly better.

Table 2 also points out the problem with the assumption that for ancien regime populations, a caloric intake that averaged 2,600 calories per consuming unit daily was adequate. In populations experiencing such low levels of average consumption, the bottom 20% subsisted on such poor diets that they were excluded effectively from the labor force; many of them lacked the energy even for a few hours of slow walking. That appears to be the principal factor explaining why beggars constituted as much as a fifth of the populations of ancien regimes (Cipolla 1980; Goubert 1973; Laslett 1983).

THE RELATIONSHIP BETWEEN BODY SIZE AND THE RISK OF DEATH AT MIDDLE AND LATE AGES

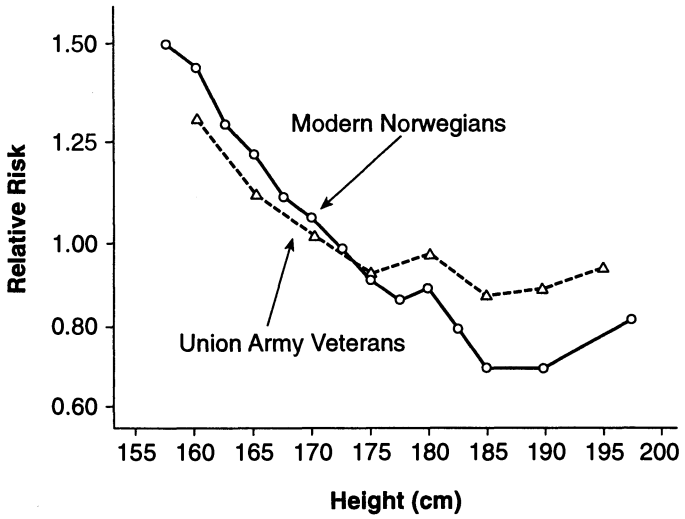
A number of recent studies have established the predictive power of height and body mass with respect to morbidity and mortality at later ages. The results of two of these studies are summarized in Figures 3 and 4. Figure 3 displays the relationship between relative mortality risk and height among Norwegian men ages 40–59 measured in the 1960s from data used by Waaler (1984) and among Union Army veterans measured at ages 23–49 and at risk between ages 55 and 75.[4] Short men, whether modern Norwegians or nineteenth century Americans, were much more likely to die than tall men. Height also has been found to be an important predictor of the relative likelihood that men ages 23–49 would be rejected from the Union Army during the period 1861–1865 because of chronic diseases (Fogel 1993). Despite significant differences in ethnicity, environmental circumstances, the array and severity of diseases, and time, the functional relationship between height and relative risk are strikingly similar in the two cases.

Waaler (1984) also has studied the relationship in Norway between the risk of death and body mass, measured by the Body Mass Index (BMI, weight in kilograms divided by height in meters squared). A curve summarizing his findings for men ages 45–49 is shown in Figure 4. The curve for Union Army veterans measured at ages 45–64 and followed for 25 years is also shown in Figure 4. Among both modern Norwegians and Union Army veterans, the curve is relatively flat within the BMI range 22–28, with the relative risk of mortality hovering close to 1. At BMIs of less than 22 and over 28 (i.e., as BMI moves away from its mean value), however, the risk of death rises quite sharply.

Although Figures 3 and 4 are revealing, they are not sufficient to shed light on the debate over whether moderate stunting impairs health when weight-for-height is adequate. To get at the "small-but-healthy" issue one needs an iso-mortality surface that relates the risk of death to height and weight simultaneously. Such a surface, presented in Figure 5, was fitted to Waaler's (1984) data. Transecting the iso-mortality map are lines that give the locus of each BMI be-

4. Relative risk of dying in a given population in Figures 3, 4, and 5 is defined as the odds of dying at a specific height divided by the mean risk of death over all heights in that population. See Kim (1996) for further details.

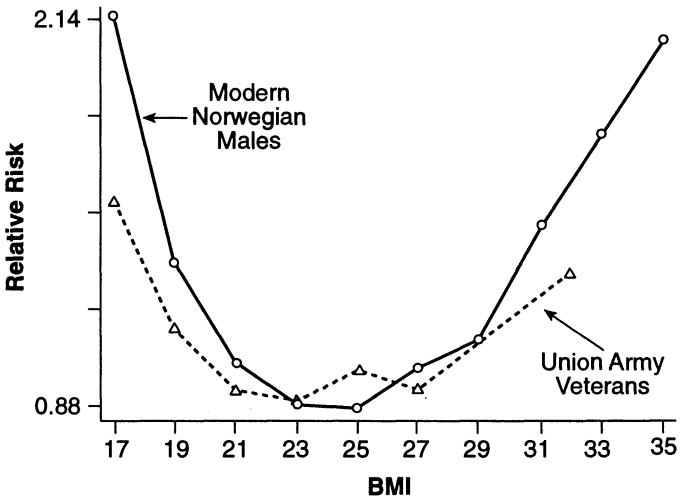
FIGURE 3. COMPARISON OF THE RELATIONSHIP BETWEEN BODY HEIGHT AND RELATIVE MORTALITY RISK AMONG MODERN NORWEGIAN MEN AND UNION ARMY VERERANS AROUND 1900



Source: Costa and Steckel (forthcoming).

Notes: Height for 309,554 modern Norwegians was measured at ages 40–59 and the period of risk was 7 years. Height of 322 Union Army verterans ages 23–49 was measured at enlistment and the period of risk was from ages 55–75.

FIGURE 4. RELATIVE MORTALITY RISK BY BMI AMONG MEN 50 YEARS OF AGE, UNION ARMY VETERANS AROUND 1900 AND MODERN NORWEGIANS



Source: Costa and Steckel (forthcoming).

Notes: In the Norwegian data BMI for 79,084 men was measured at ages 45–49 and the period of risk was 7 years. BMI of Union Army Veterans was measured at ages 45–64 and the observation period was 25 years.

tween 16 and 34 and a curve giving the weights that minimize risk at each height.

Figure 5 shows that even when body weight is maintained at what Figure 4 indicates is an "ideal" level (BMI = 25), short men are at substantially greater risk of death than tall men. Figure 5 also shows that the ideal BMI varies with height. A BMI of 25 is ideal for men about 176 cm (69 in.) tall, but for tall men the ideal BMI is between 22 and 24, whereas for short men (under 168 cm or 66 in.) the ideal BMI is about 26.[5]

Superimposed on Figure 5 are rough estimates of heights and weights in France at four dates. In 1705 the French probably achieved equilibrium with their food supply at an average height of about 161 cm (63 in.) and BMI of about 18. Over the next 270 years the food supply expanded with sufficient rapidity to permit both the height and the weight of adult males to increase. Figure 5 implies that while factors associated with height and weight jointly explain about 90% of the estimated decline in French mortality rates over the period between about 1785 and 1870, they explain only about 50% of the decline in mortality rates during the past century (Fogel et al. 1996).[6]

The analysis in this section points to the misleading nature of the concept of subsistence as Malthus originally used it and as it still is used widely today. Subsistence is not located at the edge of a nutritional cliff, beyond which lies demographic disaster. Rather than one level of subsistence, there are numerous levels at which a population and a food supply can be in equilibrium, in the sense that they can be indefinitely sustained. Some levels, however, will have smaller people and higher "normal" mortality than others.

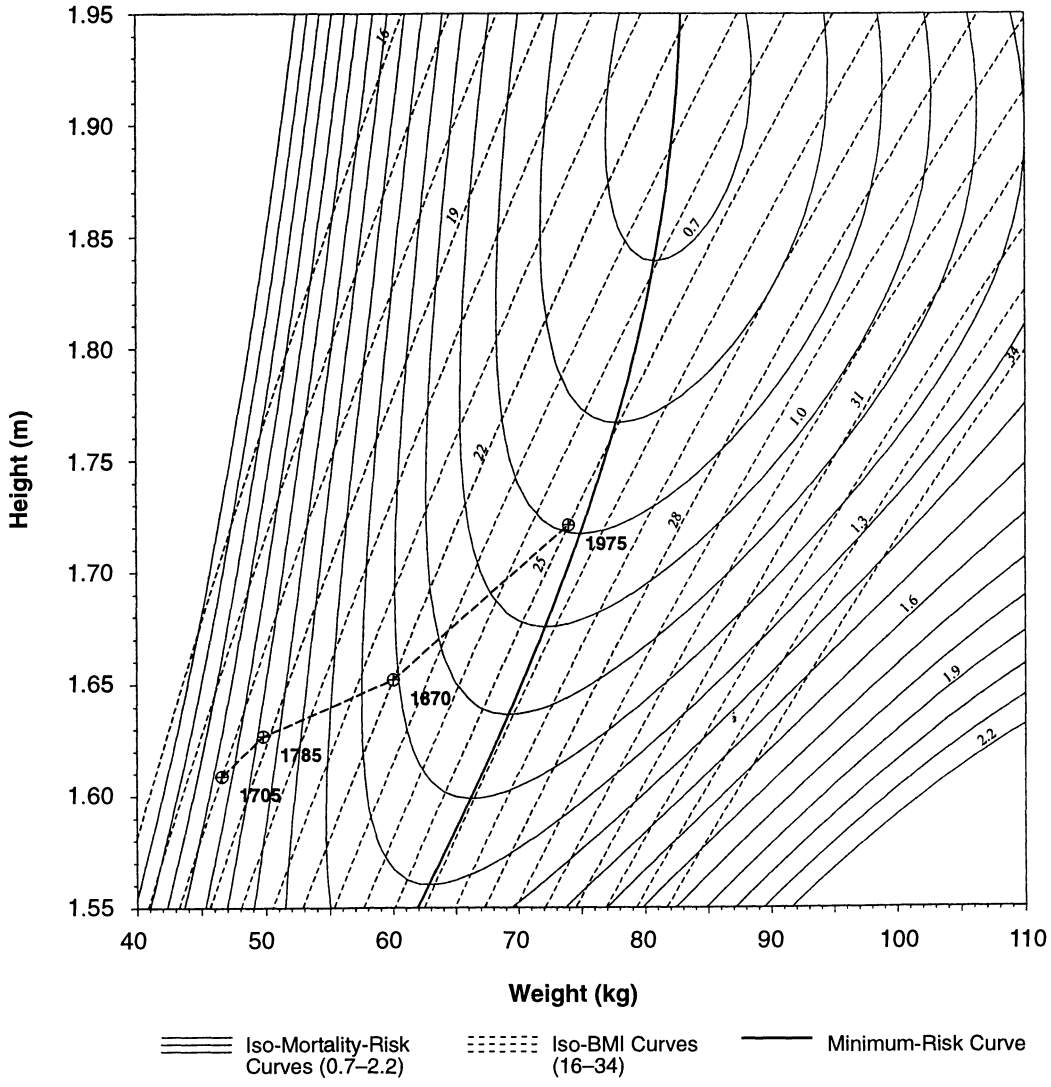
THE RELEVANCE OF WAALER SURFACES FOR PREDICTING TRENDS IN CHRONIC DISEASES

Poor body builds increased vulnerability to both contagious and chronic diseases. This point is demonstrated in Figure 6, which shows that chronic conditions were much more frequent among short young men than among tall young men in the U.S. National Health Interview Surveys (NHIS) for the period 1985–1988. Virtually the same functional relationship was found in the 1860s among young adults and

5. See Kim (1996) for the method of estimating Waaler surfaces.

6. The decreased importance of changes in human physiology that are correlated with height and weight suggest that other factors, such as medical innovations, now matter more. This does not mean, however, that height and weight have lost their predictive value. Average heights are still increasing (see footnote 12) and inability to maintain adequate BMI is life threatening for many elderly. Barker (1992, 1994) reports that anthropometric measures at birth predict BMI and hip-to-waste ratios among persons at late middle-ages.

FIGURE 5. ISO-MORTALITY CURVES OF RELATIVE RISK FOR HEIGHT AND WEIGHT AMONG NORWEGIAN MALES AGES 50–74, WITH A PLOT OF THE ESTIMATED FRENCH HEIGHT AND WEIGHT AT FOUR DATES

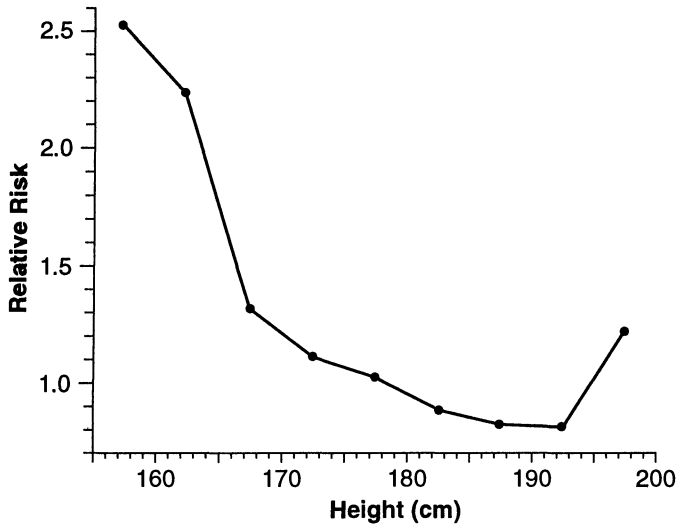


middle-aged men examined by the surgeons of the Union Army. Stunting during developmental ages had a long reach and increased the likelihood that people would suffer from chronic diseases at middle and at late ages (Fogel, Costa, and Kim 1993).

American males born during the second quarter of the nineteenth century were not only stunted by today's standards, but had BMIs at adult ages that were about 15% lower than current U.S. levels (see Figure 7). Their combined muscle and bone mass was also lower. Lean BMI at adult ages, as estimated from anthropometric measures, was about 7% lower than among men today (Costa and Steckel forthcoming). The difference in average BMI between adult males today and those born in the nineteenth century widened with age, perhaps because of the accumulated effects of differences in nutritional intakes and physical activity and because of the increased prevalence of chronic conditions at older ages. The implication of combined stunting and low BMI is brought out in Figure 8, which shows a Waaler surface for morbidity estimated by Kim (1993) from NHIS data for the period 1985–1988 that is similar to the Norwegian surface for mortality (see Figure 5).

Figure 8 also shows the coordinates in height and BMI of Union Army veterans who were 65 or older in 1910 and of veterans (mainly of World War II) who were the same ages during the period 1985–1988. These coordinates predict a

FIGURE 6. THE RELATIONSHIP BETWEEN HEIGHT AND RELATIVE RISK OF ILL HEALTH IN NHIS VERTERANS AGES 40–59



Source: Fogel, Costa, and Kim (1993).

decline of about 35% in the prevalence of chronic disease among the two cohorts (Fogel et al. 1993).

This prediction is quite close to what actually occurred.[7] Table 3 shows comparisons of the prevalence of chronic diseases among Union Army men ages 65 and older in 1910 with two surveys of veterans of the same ages in the 1980s. That table indicates that among the elderly heart disease was 2.9 times as prevalent, musculoskeletal and respiratory diseases were 1.6 times as prevalent, and digestive diseases were 4.7 times as prevalent among veterans ages 65 or older in 1910 as in the period 1985–1988. Young adults born between 1822 and 1845 who survived the deadly infectious diseases of childhood and adolescence were not, as some have suggested, freer of degenerative diseases than persons of the same ages today; rather they were more afflicted. Hernia rates at ages 35–39, for example, were more than 3 times as prevalent in the 1860s as in the 1980s.

Of special note is the much higher incidence of clubfoot in the 1860s—a birth anomaly which suggests that the uterus was far less safe for those awaiting birth in the 1860s than it is today. The provisional findings thus suggest that chronic conditions were far more prevalent throughout the life cycle for those who reached age 65 before World War I than is suggested by the theory of the epidemiological transition (Fogel et al. 1993). Reliance on causes-of-death information to characterize the epidemiology of the past has led to a significant misrepresentation of the distribution of health conditions among the living. It also has promoted the view that the epidemiology of chronic diseases is more separate from that of contagious diseases than now appears to be the case.

PHYSIOLOGICAL FOUNDATIONS FOR WAALER SURFACES AND CURVES

What is the basis for the predictive capacity of Waaler surfaces and curves? Part of the answer resides in the realm of human physiology. Variations in height and weight appear to be associated with variations in the chemical composition of the tissues that make up these organs, in the quality of the electrical transmission across membranes, and in the functioning of the endocrine system and other vital systems.

Research in this area is developing rapidly and some of the new findings are yet to be confirmed. The exact mechanisms by which malnutrition and trauma in utero or during early childhood are transformed into organ dysfunctions are still unclear. What is agreed upon is that the basic structure of most organs is laid down early, and it is reasonable to infer that poorly developed organs may break down earlier than well developed ones (Tanner 1990, 1993). The principal evidence so far is statistical and, despite agreement on certain specific dysfunctions, there is no generally accepted theory of cellular aging.

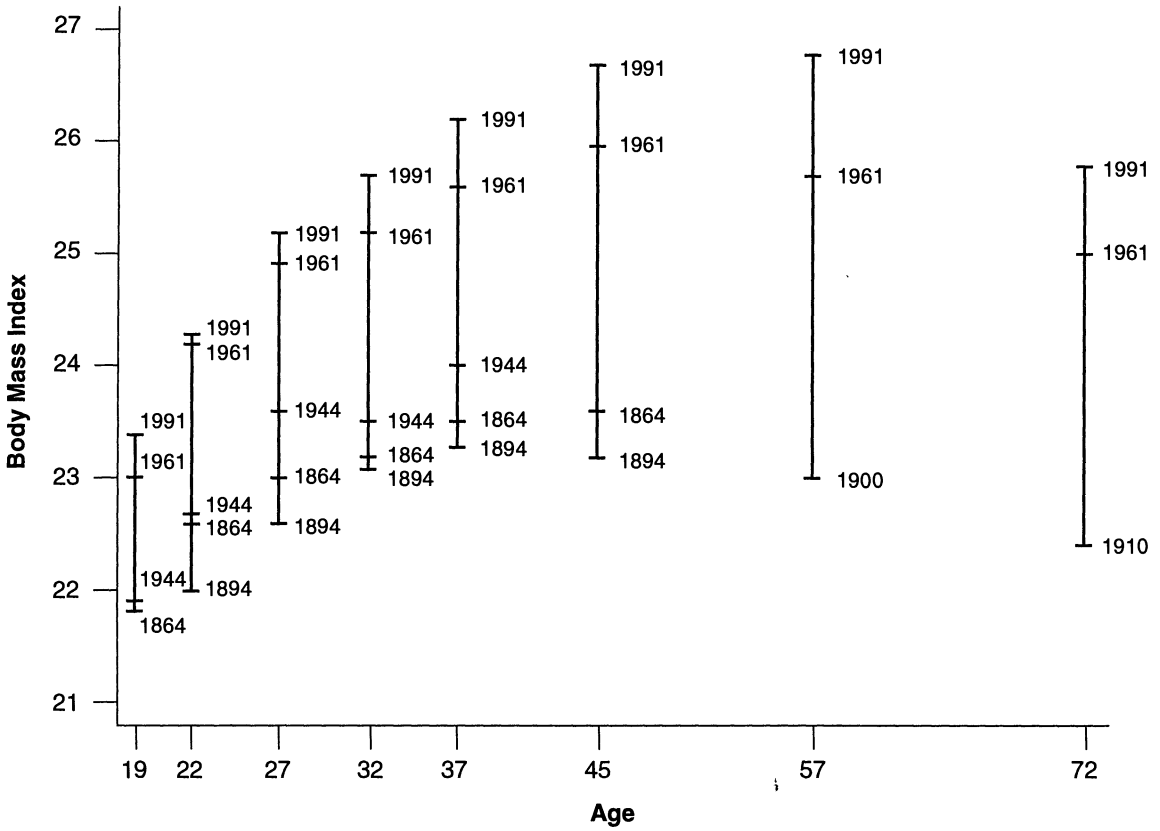
With these caveats in mind, recent research bearing on the connection between malnutrition and body size and the later onset of chronic diseases can be divided conveniently into three categories. The first category involves forms of malnutrition (including the ingestion of toxic substances) that cause permanent, promptly visible physiological damage, as is seen in the impairment of the nervous systems of fetuses due to pregnant women's smoking or excess consumption of alcohol. It appears that protein-calorie malnutrition (PCM) in infancy and early childhood can lead to a permanent impairment of central nervous system function. Iodine deficiency *in utero* and moderate to severe iron deficiency during infancy also appear to cause permanent neurological damage.[8]

Not all damage due to retarded development in utero or during infancy caused by malnutrition shows up immediately. In a recent series of studies D.J.P. Barker and his colleagues (Barker 1992, 1994) have reported that conditions such as coronary heart disease, hypertension, stroke, noninsulin-dependent diabetes, and autoimmune thyroiditis begin *in utero* or in infancy, but do not become apparent until midadult or late ages. In these cases, individuals appear to be in good health and function well in the interim. Early onset of the degenerative diseases of old age, however, appears to be linked to inadequate cellular development early in life.

7. The fact that height and weight predict the decline in chronic conditions does not mean that they caused the decline: These variables may be merely proxies for more fundamental physiological changes that are not directly measured (see the next section).

8. On fetal alcohol syndrome, see Robbins, Cotran, and Kumar (1984). On protein-caloric malnutrition in infancy and early childhood, see Chavez, Martinez, and Soberanes (1995), Martorell, Rivera, and Kaplowitz (1990), and Scrimshaw and Gordon (1968); but cf. Volpe (1987). On the effect of iodine and iron deficiency in utero and infancy, see Lozoff, Jimenez, and Abraham (1991) and Scrimshaw (1993).

FIGURE 7. MEAN BODY MASS INDEX BY AGE GROUP AND YEAR, 1863–1991



Source: Costa and Steckel (forthcoming).

Note: The age groups, which are centered at the marks, are ages 18–19, 20–24, 25–29, 30–34, 35–39, 40–49, 50–64, and 65–79. For some years BMI is not available for a specific age group.

Some of these cases are associated with low birth weight. Some babies are born in the normal weight range but experience below average infant weight gains. In other instances babies are small relative to the size of their placentas, short in relation to the size of their head, or long but thin.

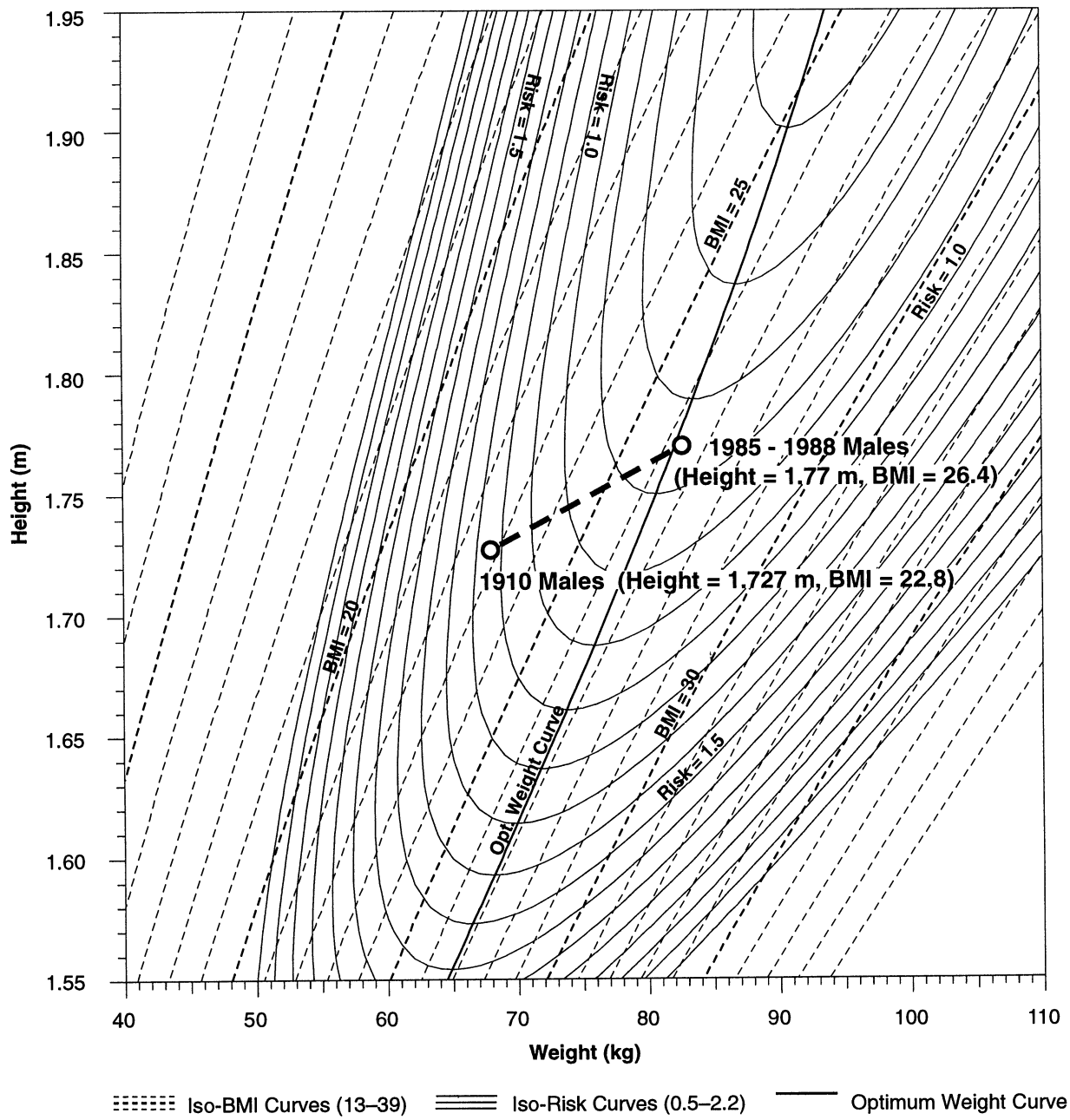
Certain physiological dysfunctions incurred by persons suffering from malnutrition, in principle, can be reversed by improved dietary intake, but they often persist because the cause of the malnutrition persists. If the malnutrition persists long enough these conditions can become irreversible or fatal. This category of dysfunctions includes the degradation of tissue structure, especially in vital organs such as the lungs, the heart, and the gastrointestinal tract. In the case of the respiratory system, for example, there is not only decreased muscle mass and strength but also impaired ventilatory drive, biochemical changes in connective tissue, and electrolyte abnormalities. Malnutrition also has been related to the atrophy of the mucosal cells of the gut, the inhibition of wound healing, increased likelihood of traumatic shock and of sepsis, impaired functioning of the endocrine system, increased tendency to edema, electrical instability that can provoke acute arrhythmias, and degenerative joint diseases (Hill 1990; Idiaquez 1988; McMahon and Bistrian 1990; Saba, Dillon, and Lanser 1983; Fisler 1992; cf. Manton 1993).

Also relevant is the discovery of the relationship between birth weight and the probability of neonatal death. The curves in Figure 9 are "U" shaped, indicating that in each population babies significantly heavier than the optimal weight also incur high mortality risks. Moreover, the optimal birth weight in the two populations with small mothers was significantly lower than that of the U.S. population where mothers were relatively large (Hytten and Leitch 1971; but cf. Chandra 1975). In other words, it appears that before high-technology interventions, the size of the mother's pelvis constituted an intergenerational constraint on the rate at which babies could escape from the effects of malnutrition as fetal nutrition improved.

THE INSTABILITY OF TECHNOPHYSIO EVOLUTION

Available data on stature and mortality from a number of countries indicate that during the eighteenth and nineteenth centuries, technophysio evolution did not proceed steadily in

FIGURE 8. HEALTH IMPROVEMENT PREDICTED BY NHIS 1985–1988 HEALTH SURFACE



Source: Kim (1993).

Note: All risks are measured relative to the average risk of morbidiy (calculated over all heights and weights) among NHIS 1985–1988 white males ages 45–64.

either Europe or the United States. Figure 10 summarizes the available data on U.S. secular trends in both stature and mortality since 1720 (Costa and Steckel forthcoming; Fogel 1986). The series on both stature and period life expectancy at age 10 contain striking cycles. Both series rise during most of the eighteenth century, attaining substantially greater heights and life expectations than prevailed in England during the same period. Life expectancy began to decline during the 1790s and continued to do so for about half a century. There may have been a slight decline in the heights of

TABLE 3. PREVALENCE OF CHRONIC CONDITIONS AMONG UNION ARMY VETERANS IN 1910, VETERANS IN 1983 AND VETERANS IN NHIS 1985–1988, AGES 65 AND ABOVE

Disorder	Union Army Veterans	1983 Veterans[a]	Age-Adjusted 1983 Veterans	NHIS 1985–1988 Veterans[b]
SKIN OR MUSCULOSKELETAL (%)	68.4	48.1	47.5	45.8
Musculoskeletal	67.7	47.9	47.2	42.5
DIGESTIVE (%)	84.0	49.0	48.9	8.0
Hernia	34.5	27.3	26.7	6.6
Diarrhea	31.9	3.7	4.2	1.4
GENITO-URINARY (%)	27.3	6.3	32.2	8.9
CENTRAL NERVOUS, ENDOCRINE, METABOLIC, OR BLOOD (%)	24.2	29.9	9.1	12.6
CIRCULATORY (%)	90.1	42.9	39.9	40.0
Heart	76.0	38.5	39.9	26.6
Varicose veins	38.5	8.7	8.3	5.3
Hemorrhoids	44.4			7.2
RESPIRATORY (%)	42.2	29.8	28.1	26.5
NEOPLASMS (%)	2.2	13.1	11.5	9.2

Source: Fogel et al. (1993).

[a] Includes veterans in 1983 reporting whether they had specific chronic conditions.

[b] Includes veterans in NHIS from 1985–1988 reporting whether they had specific chronic conditions during the preceding 12 months.

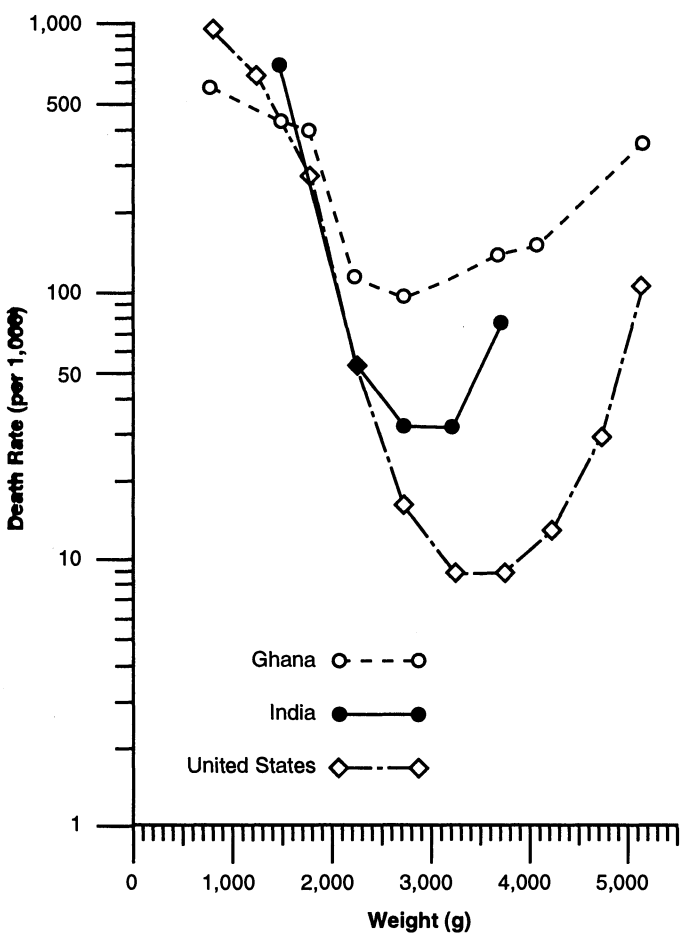
cohorts born between 1785 and 1820, but the sharp decline, which probably lasted about half a century, began with cohorts born around 1830. At the same time, cohort life expectancy at age 20 began an accelerated decline (Pope 1992).[9] A new rise in heights, the one with which we long have been familiar, probably began with cohorts born during the last decade of the nineteenth century and continued for about 60 years. Similar cycles in height occurred in Hungary, Sweden, and Britain.

Evidence on trends in BMI and morbidity is sparser, but the time pattern agrees with that of stature and mortality. BMI of Citadel cadets age 18 declined from the 1870s to the 1890s before rising by 1920 (Coclanis and Komlos 1995). Figure 7 shows declines in BMI between 1864 and 1894 among men ages 20–24, 25–29, and 30–34. Prevalence rates at older ages for chronic conditions among cohorts who were born in the period 1840–1849 were higher than those for cohorts who were born in the periods 1830–1839 and 1820–1929. At ages 65–74 men who were born in the period 1820–29 were significantly less likely than men born between 1840 and 1849 to suffer from rheumatism, hemorrhoids, respiratory disorders, hernias, and stomach disorders. They were significantly less likely to have respiratory and stomach disorders than were men born in the period 1830–1839, who, in turn, were less likely than men born in the period 1840–1849 to suffer from circulatory conditions (Costa and Steckel forthcoming). The stunting of the 1840–1849 cohort relative to the 1830–1839 and 1820–1829 cohorts appears to be reflected in health outcomes at older ages.

Although the mix of factors tending to retard improvements in nutritional status and health varied from one country to another, one factor stands out more than any other: rapid urbanization. In both Europe and the United States the urban population during the nineteenth century grew far more rapidly than at any other time in history, exceeding the capacity of the cities to supply clean water, to remove waste, and to contain the spread of infection (Bairoch 1988). The mortality rate appears to have been influenced both by the size of the city and by the rapidity of its growth rate. In the case of the United States around 1830, cities with 50,000 or more persons had more than twice the death rates of rural areas; similar patterns have been observed for Europe. The exact threshold at which city size began to affect mortality rates varied with time, place, and circumstance, but in the United States during the mid-nineteenth century, cities of about 25,000 persons appear to have been

9. We use period rather than cohort life expectancies in Figure 10 because less complete information is available for cohort life expectancies.

FIGURE 9. PRENATAL RATE BY BIRTH WEIGHT IN GHANA, INDIA, AND THE UNITED STATES



Source: Hytten and Leitch (1971).

the threshold of significant elevation in mortality rates (Fogel et al. 1978).

THERMODYNAMIC AND PHYSIOLOGICAL FACTORS IN ECONOMIC GROWTH

So far we have focused on the contribution of technological change to physiological improvements. The process has been synergistic, however, with improvement in nutrition and physiology contributing significantly to the process of economic growth and technological progress.

We alluded to the thermodynamic contribution to economic growth when we pointed out that individuals in the bottom 20% of the caloric distributions of France and England around 1790 lacked the energy for sustained work and were excluded effectively from the labor force. Moreover, even those who participated in the labor force had only relatively small amounts of energy for work.

Because the first law of thermodynamics applies as much to human engines as to mechanical ones, it is possible to use energy cost accounting to estimate the increase in energy available for work over the past two centuries. In the British case the thermodynamic factor explains 30% of the British growth rate since 1790 (Fogel et al. 1996). The increase in the amount of energy available for work had two effects. It raised the labor force participation rate by bringing into the labor force the bottom 20% of consuming units in 1790 who had had, on average, only enough energy for a few hours of slow walking. Moreover, for those in the labor force, the intensity of work per hour has increased because the number of calories available for work each day increased by about 50%.

The physiological factor pertains to the efficiency with which the human engine converts energy input into work output. Changes in health, in the composition of diet, and in clothing and shelter can affect significantly the efficiency with which ingested energy is converted into work output. Reductions in the incidence of infectious diseases increase the proportion of ingested energy available for work both because of savings in the energy required to mobilize the immune system and because the capacity of the gut to absorb nutrients is improved, especially as a consequence of a reduction in diarrheal diseases.

Thermodynamic efficiency also has increased because of changes in the composition of the diet, including the shift from grains and other foods with high fiber content to sugar and meats. These dietary changes raised the proportion of ingested energy that can be metabolized (i.e, increased the average value of the "Atwater Factors," to use the language of nutritionists). Improvements in clothing and shelter also have increased thermodynamic efficiency by reducing the amount of energy lost through radiation (Dasgupta 1993).

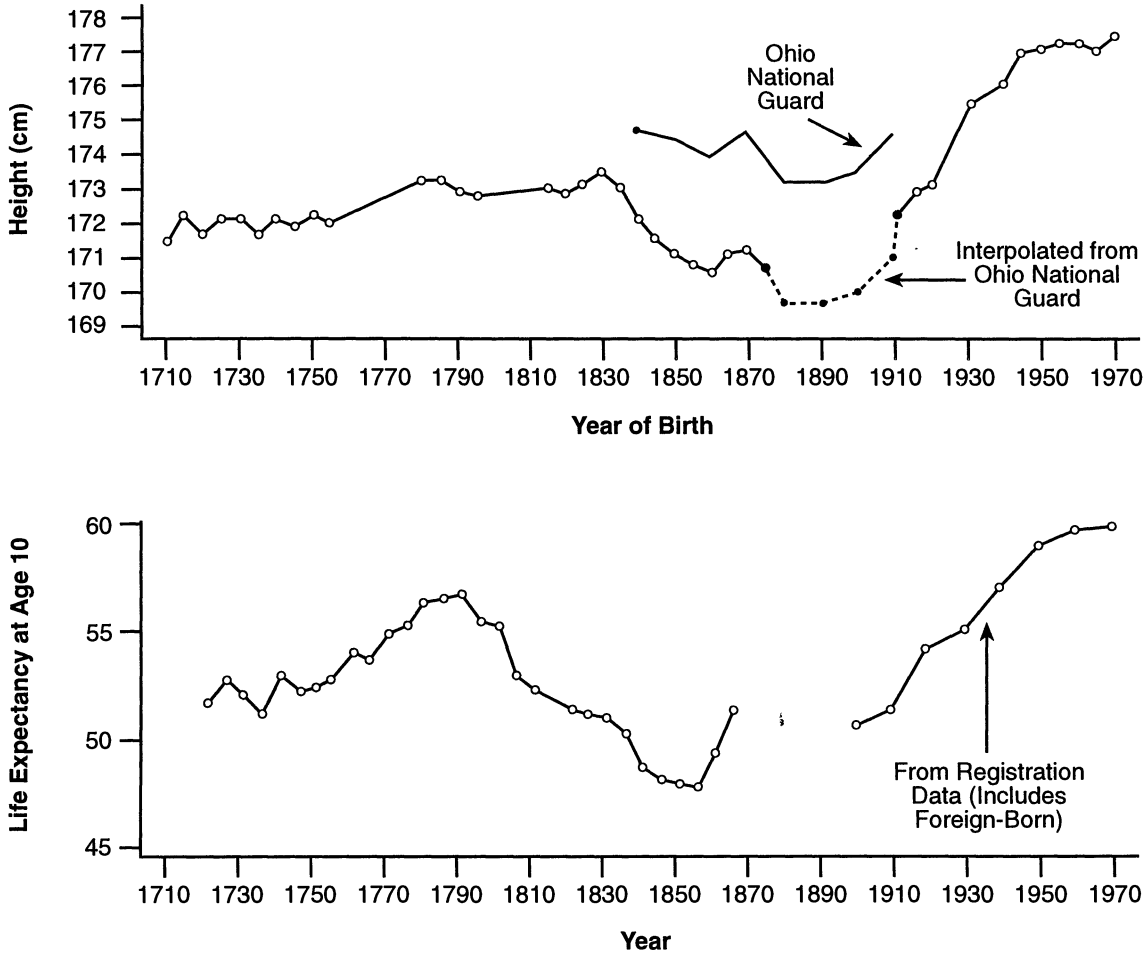
Moreover, individuals who are stunted but otherwise healthy at maturity will be at an increased risk of incurring chronic diseases and of dying prematurely. In other words, when considered as work engines, they wear out more quickly and are less efficient at each age. The available data suggest that the average efficiency of the human engine in Britain increased by about 53% between 1790 and 1980. The combined effect of the increase in dietary energy available for work and the increased human efficiency in transforming dietary energy into work output appears to account for about 50% of the British economic growth since 1790.[10]

SOME IMPLICATIONS FOR FORECASTING AND FOR CURRENT POLICY

Between 1850 and 1950 U.S. life expectation at birth increased from about 40 to 68 years. Then for the next two decades further progress in longevity came to a virtual halt. During and following this interregnum investigators who reviewed the progress in mortality over the preceding century tended toward a consensus on three propositions:

10. See Fogel (1994) for computational procedures.

FIGURE 10. TREND IN MEAN FINAL HEIGHT OF NATIVE-BORN, WHITE AMERICAN MALES AND TREND IN THEIR LIFE EXPECTANCY AT AGE 10



Sources: Fogel (1986) and Costa and Steckel (forthcoming).

Note: Height is by birth cohort and life expectancy at age 10 is by period.

(1) The century-long decline in mortality rates was unique and could not be repeated because virtually all of the gains made through the elimination of death from contagious diseases below age 60 had been made.
(2) Deaths, now concentrated at older ages, were due to degenerative diseases that were unrelated to the contagious diseases they superseded. The degenerative diseases were caused by accelerated organ losses that were part of the natural process of aging.
(3) There was an upper limit to life expectation that was genetically determined. One influential paper put that limit at 85 ±7 years (Fries 1980, 1989).

More recent studies, responding to the renewed decline in mortality for those ages 65 and older, have uncovered evidence that militates against the notion of a genetically fixed life span or, if it is fixed, suggests that the upper limit is well above 85 years. McGue, Vaupel, and Holm's (1993) study of Danish twins indicates that genetic factors account for only about 30% of the variance in age at death. Their study of Swedish males who lived to age 90 indicates that the death rate at that age has declined by about 1% per annum since 1950—a finding that is contradictory to the rectangularization of the survivorship curve.[11] Two recent studies of insect populations indicated that variation in environmental conditions had a much larger effect on the life span than genetic factors

11. Wilmoth and Lundström (1996) indicate that in five countries for which adequate data are available, the upper tail of the age distribution of death has been moving steadily higher for a century or more.

and revealed no pattern suggestive of a fixed upper limit (Carey et al. 1992; Curtsinger et al. 1992; Kannisto et al. 1994; Thatcher 1992; Vaupel 1991; Vaupel and Lundström 1994). Collectively these studies do not rule out genetic factors, but they suggest something much less rigid than the genetic programming of absolute life spans: An emerging theory combines genetic susceptibility of various organs with cumulative insults as a result of exposure to risk.

Recent studies also indicate that age-specific rates of chronic conditions above age 65 generally are falling. According to Manton, Corder, and Stallard (1993) the rate of disability among the elderly in the U.S. declined by 4.7% between 1982 and 1989. Viewed on a decade basis, this rate of decline is quite similar to the long-term rates of decline between 1910 and 1985–1988 in chronic conditions among elderly veterans. The finding is consistent with the growing body of evidence indicating that chronic diseases at later ages are, to a considerable degree, the result of exposure to infectious diseases, malnutrition, and other types of biomedical and socioeconomic stress early in life. It is also consistent with the predicted decline of about 6% per decade in chronic diseases based on the Waaler surface in ill health displayed in Figure 6 (Blair et al. 1989; Fogel et al. 1993; Manton et al. 1993; Manton and Soldo 1992; Manton, Stallard, and Singer 1992).

Much current research is now focused on explaining the decline in chronic conditions. Part of the emerging explanation is a change in life styles: Reduced smoking, improved nutrition, and increased exercise appear to be involved in reducing the prevalence of coronary heart disease and respiratory diseases. Another part of the explanation is the increasing effectiveness of medical intervention. This point is demonstrated strikingly by comparing the second and last columns of the row for hernias in Table 3. Before World War II, hernias (once they occurred) were generally permanent and often exceedingly painful conditions. By the 1980s, however, about three-quarters of all veterans who ever had hernias were cured of them. Similar progress over the seven decades is indicated in the row for genito-urinary conditions. Other areas where medical intervention has been highly effective include control of hypertension and reduction in the incidence of stroke, surgical removal of osteoarthritis, replacement of knee and hip joints, curing of cataracts, and chemotherapies that reduce the incidence of osteoporosis and heart disease.

The success in medical interventions combined with rising incomes has naturally led to a huge increase in the demand for medical services. Econometric estimates suggest a long-run income elasticity of around 1.5 in the demand for medical services across Organization of Economic Cooperation and Development (OECD) nations. The rapidly growing level of demand—combined with the egalitarian policy of providing medical care at highly subsidized prices—has created the crisis in health care costs now a major focus of public policy debates across OECD nations, with various combinations of price and governmental rationing under consideration (The Economist 1990; Newhouse 1992; Schieber, Poullier, and Greenwald 1993; Schwartz and Aaron 1991).

Growing opportunity to improve health at young ages, to reduce the incidence of chronic diseases at late ages, and to cure or alleviate the disabilities associated with chronic diseases raises two other post-Malthusian population issues. One is the impact of improved health on population size. In a recent paper Ahlburg and Vaupel (1990) pointed out that if mortality rates at older ages continue to decline at 2% per annum, the U.S. elderly population in 2050 will be 36 million larger than forecast by the Census Bureau (cf. Preston 1993). That possibility poses policy issues with respect to health care costs (because total medical costs may rise sharply even if cure rates continue to improve) and to pension costs (because the number of persons eligible for benefits under present proposed rules, and the projected compensation levels will become so large that outpayments will exceed planned reserves).

Some policymakers have sought to meet the pension problem by delaying retirement. Such schemes are based on the proposition that improved health will make it possible for more people to work past age 65. The recent findings on the secular improvement in health at older ages, however, make it clear that worsening health is not the explanation for the steep decline in labor force participation rates of males over 65 since 1890. Not only has average health improved, but health has become less important to the labor supply decision (Costa 1996a). As Costa (1995) has reported, the U.S. decline in participation rates of the elderly over the past century is explained largely by the secular rise in income and a decline in the income elasticity of the demand for retirement. It also is related to the vast increase in the supply and the quality of leisure-time activities for the laboring classes.

In Malthus's time, and down to the opening of this century, leisure was in very short supply in OECD countries and, as Veblen (1934) pointed out, it was conspicuously consumed by a small upper class. Over the course of the twentieth century, hours of work have fallen by nearly half for typical workers. Ironically, those in the top decile of the income distribution have not shared much in this gain of leisure; the highly paid professionals and businessmen who populate the top decile work closer to the nineteenth century standard of 3,200 hours per year than to the working class standard of about 1,800 hours.

There also has been a vast increase in the supply of leisure-time activities. The hours between dinner and bedtime now can be spent on either live entertainment, spectator sports, movies, movie rentals, television, listening to the radio or to compact discs, or reading. Weekends can be spent at distant or nearby ski resorts or beaches, at the backyard or neighbor pool or park, or on hobbies. The goods that enhance leisure time have become increasingly accessible. Estimated expenditure elasticities for recreational goods have fallen from around 2 at the beginning of the century to slightly more than 1 today (Costa 1996b). Recreational goods have become less and less of luxury, suggesting that the additional amount of income needed to enjoy leisure has fallen. As a result, the typical worker spends two-thirds as much time in

leisure activities as in work and looks forward to retirement (Costa 1996b; Fogel 1992a, 1992b).

Given the growing and income-inelastic demand for leisure that characterizes the post-Malthusian milieu of the OECD nations, the extent to which the demand for leisure and retirement can be throttled remains to be seen. Policymakers may encounter as much resistance to efforts to reduce the implicit subsidies for leisure as they have recently in raising the taxes of work.

CONCLUDING COMMENTS

In setting forth this theory of technophysio evolution we have sought to emphasize the extent to which human physiology has been altered by environmental change over the past 10 generations, and particularly over the past three to four generations. Although technophysio changes have been very rapid from an evolutionary perspective, little of this pattern of change is visible over a couple of decades which is half the length of a typical biomedical career. Over such relatively brief spans human physiology may seem to be more fixed than in fact it has been intergenerationally.

We know from fly and other animal models that controlling the environment can greatly alter physiological functioning, and expand the average length of life by as much as threefold. Of course, flies cannot control their environment, so their life span under natural circumstances has not changed. Humans, however, control their own environment to a large degree, and they have greatly altered their own physiology and average life span in relatively few generations. The current pace of technological change within and beyond the biomedical sciences, as well as the continuing growth in stature and the reduction in mortality rates at older ages, suggest that technophysio evolution has not yet run its course.[12]

12. Consistent with Figure 10, the increase in the mature stature of males has leveled off in several rich countries (cf. Schmidt, Jorgensen, and Michaelson 1995). This finding does not necessarily imply that the secular growth is over or that stature no longer will be useful in forecasting trends in mortality. The United States and other nations previously have experienced renewed growth in final heights that followed periods of leveling-off and even periods of reversal.

Although a biological theory suitable for establishing an upper limit on human height has not emerged yet, several factors suggest that average final heights will continue to increase in the United States. First, in recent years an increasing share of the gross increase in population, up to nearly one-third, has been due to immigration of persons with substantially lower average adult stature than natives of native parents. This factor accounts for part of the recent leveling-off in the increase in stature shown in Figure 10.

The more or less simultaneous leveling-off of height in several developed countries may be due to common environmental insults. One leading candidate is the spread of smoking among women of childbearing ages during the 1930s and 1940s. Several studies have shown that smoking by pregnant women not only retards fetal development but is associated with the subsequent stunting of children during developmental ages and at maturity (cf. Barker et al. 1991; Eskenazi and Bergmann 1995; Fogelman and Manor 1988; Gidding et al. 1995; Kaplan and Salonen 1990; Rantakallio 1988; Wheeler et al. 1995; Zaren et al. 1996).

Even if there is no further increase in stature at maturity in native-born persons of native-born parents, the cohort born in 1970 will be substantially taller at older ages than the cohorts currently ages 60 and older, who were born mainly between 1905 and 1930 and who were shorter at maturity. Because mortality now is concentrated overwhelmingly at ages 60 and older, it is the increase in stature at these ages that is relevant for forecasting mortality declines (cf. Kim 1996).

For evidence on the persistence of the secular trend in height among the Dutch children see Gerver, De Bruin, and Drayer (1994); on Sweden see Albertson-Wikland and Karlberg (1994).

REFERENCES

Ahlburg, D A. and J.W. Vaupel. 1990. "Alternative Projections of the U.S. Population." *Demography* 27:639–52.

Albertson-Wikland, K. and J. Karlberg. 1994. "Natural Growth in Children Born Small for Gestational Age with and without Catch-Up Growth." *Acta Paediatrica Supplement* 399:64–70.

Allen, R. 1992. *Enclosure and the Midlands, 1450–1850. Yeoman: The Agricultural Development of the South.* Oxford: Oxford University Press

———. "Agriculture During the Industrial Revolution." Pp. 96–122 in *The Economic History of Britain Since 1700*, 2nd edition, edited by R. Floud and D. McCloskey. Cambridge: Cambridge University Press.

Bairoch, P. 1988. *Cities and Economic Development from the Dawn of History to the Present*, translated by C. Brauder. Chicago: University of Chicago Press.

Barker, D.J.P., ed. 1992. *Fetal and Infant Origins of Adult Disease.* London: British Medical Journal.

———. 1994. *Mothers, Babies and Disease in Later Life.* London: BMJ Publishing Group.

Barker, D.J.P., K.M. Godfrey, C. Fall, C. Osmond, P.D. Winter, and S.O. Shaheen. 1991. "Relation of Birth Weight and Childhood Respiratory Infection to Adult Lung Function and Death from Chronic Obstructive Airways Disease." *British Medical Journal* 303:671–75.

Bengtsson, T. and R. Ohlsson. 1984. "Population and Economic Fluctuations in Sweden, 1749–1914." Pp. 277–97 in *Pre-Industrial Population Changes*, edited by T. Bengtsson, G. Fridlizius, and R. Ohlsson. Stockholm: Almquist and Wiksell.

Bingham, S.A. 1994. "The Use of 24–h Urine Samples and Energy Expenditure to Validate Dietary Assessments." *American Journal of Clinical Nutrition* 59:227S–31S.

Black, A.E., W.A. Coward, T.J. Cole, and A.M. Prentice. 1996. "Human Energy Expenditure in Affluent Societies: An Analysis of 574 Doubly-Labelled Water Measurements." *European Journal of Clinical Nutrition* 50:72–92.

Blair, S.N., H.W. Kohl, III, R.S. Paffenbarger, D.G. Clark, K.H. Cooper, and L.W. Gibbons. 1989. "Physical Fitness and All-Cause Mortality: A Prospective Study of Healthy Men and Women." *Journal of the American Medical Association* 262:2395–401.

Bowley, A.L. and A.R. Burnett-Hurst. 1915. *Livelihood and Poverty.* London: Ratan Tata Foundation.

Carey, J.R., P. Liedo, D. Orozco, and J.W. Vaupel. 1992. "Slowing of Mortality Rates at Older Ages in Large Medfly Cohorts." *Science* 258:457–91.

Case, R.A.M., C. Coghill, J.L. Harley, and J.T. Pearson. 1962. *Chester Beatty Research Institute Abridged Serial Life Tables,*

England and Wales 1841–1960, Part I. London: Chester Beatty Research Institute.

Chandra, R.K. 1975. "Antibody Formation in First and Second Generation Offspring of Nutritionally Deprived Rats." *Science* 190:289–90.

———. 1992. "Nutrition and Immunoregulation. Significance for Host Resistance to Tumors and Infectious Diseases in Humans and Rodents." *Journal of Nutrition* 122:754–57.

Chavez, A., C. Martinez, and B. Soberanes. 1995. "The Effect of Malnutrition on Human Development: A 24-Year Study of Well-Nourished and Malnourished Children Living in a Poor Mexican Village." Pp. 79-124 in *Community Based Longitudinal Studies of the Impact of Early Malnutrition on Child Health and Development: Classical Examples from Guatemala, Haiti and Mexico*, edited by N.S. Scrimshaw. Boston: International Nutrition Foundation for Developing Countries.

Cipolla, C.M. 1974. *The Economic History of World Population*, 6th edition. Harmondsworth, Middlesex: Penguin Books.

———. 1980. *Before the Industrial Revolution: European Society and Economy, 1000–1700*, 2nd edition. New York: Norton.

Clark, J.G.D. 1961. *World Prehistory: An Outline*. Cambridge: Cambridge University Press.

Coclanis, P. and J. Komlos. 1995. "Nutrition and Economic Development in Post-Reconstruction South Carolina: An Anthropometric History." *Social Science History* 19:91–116.

Costa, D.L. 1995. "Pensions and Retirement: Evidence from Union Army Veterans." *Quarterly Journal of Economics* 110:297–320.

———. 1996a. "Health and Labor Force Participation of Older Men, 1900–1991." *Journal of Economic History* 56:540–52.

———. 1996b. "The Rise of the Leisured Class: The Growth of Recreation Since 1888." Unpublished manuscript. Economics Department, Massachusetts Institute of Technology.

Costa, D.L. and R. Steckel. Forthcoming. "Long-Term Trends in Health, Welfare, and Economic Growth in the United States." In *Health and Welfare During Industrialization*, edited by R.H. Steckel and R. Floud. Chicago: University of Chicago Press for National Bureau of Economic Research.

Curtsinger, J.W., H.H. Fukui, D.R. Townsend, and J.W. Vaupel. 1992. "Demography of Genotypes: Failure of the Limited Life-Span Paradigm in *Drosophila melanogaster*." *Science* 258:461–63.

Dasgupta, P. 1993. *Inquiry Into Well-Being and Destitution*. Oxford: Clarendon.

Dublin, L.I. and A.J. Lotka. 1936. *Length of Life: A Study of the Life Table*. New York: Ronald Press Company.

Dublin, L.I., A.J. Lotka, and M. Spiegelman. 1949. *Length of Life: A Study of the Life Table*, revised edition. New York: Ronald Press Company.

DuPâquier, J. 1989. "Demographic Crises and Subsistence Crises in France, 1650–1725." Pp. 189–99 in *Famine, Disease and the Social Order in Early Modern Society*, edited by J. Walter and R. Schofield. Cambridge: Cambridge University Press.

Eckstein, Z., T.P. Schultz, and K.I. Wolpin. 1984. "Short-Run Fluctuations in Fertility and Mortality in Pre-Industrial Sweden." *European Economic Review* 26:295–317.

The Economist. 1990. "Squeezing in the Next Five Billion." 314(7638):19–20, 22.

Eskenazi, B. and J.J. Bergmann. 1995. "Passive and Active Maternal Smoking During Pregnancy, as Measured by Serum Cotinine, and Postnatal Smoke Exposure. I. Effects on Physical Growth at Age 5 Years." *American Journal of Epidemiology* 142:S10–18.

Fagan, B.M. 1977. *People of the Earth*, 2nd edition. Boston: Little, Brown and Co.

Fisler, J.S. 1992. "Cardiac Effects of Starvation and Semistarvation Diets: Safety and Mechanisms of Action." *American Journal of Clinical Nutrition* 56:230S–34S.

Floud, R.C. and K.W. Wachter. 1982. "Poverty and Physical Stature." *Social Science History* 6:422–52.

Fogel, R.W. 1986. "Nutrition and the Decline in Mortality Since 1700: Some Preliminary Findings." Pp. 439–555 in *Long-Term Factors in American Economic Growth*, edited by S.L. Engerman and R.E. Gallman. Chicago: University of Chicago Press.

———. 1987. "Biomedical Approaches to the Estimation and Interpretation of Secular Trends in Equity, Morbidity, Mortality, and Labor Productivity in Europe, 1750–1980." Unpublished Manuscript. Center for Population Economics, University of Chicago.

———. 1992a. "Egalitarianism: The Economic Revolution of the Twentieth Century." The 1992 Kuznets Memorial Lecture presented at Yale University, April 22–24.

———. 1992b. "Second Thoughts on the European Escape from Hunger: Famines, Chronic Malnutrition, and Mortality." Pp. 243–86 in *Nutrition and Poverty*, edited by S.R. Osmani. Oxford: Clarendon.

———. 1993. "New Sources and New Techniques for the Study of Secular Trends in Nutritional Status, Health, Mortality and the Process of Aging." *Historical Methods* 26:5–43.

———. 1994. "Economic Growth, Population, Theory, and Physiology: The Bearing of Long-Term Processes on the Making of Economic Policy." *American Economic Review* 84:369–95.

Fogel, R.W., D.L. Costa, and J. Kim. 1993. "Secular Trends in the Distribution of Chronic Conditions and Disabilities at Young Adult and Late Ages." Presented at the National Bureau of Economic Research Summer Institute, Economics of Aging Program, July 26–28.

Fogel, R.W., S.L. Engerman, J. Trussell, R. Floud, C.L. Pope, and L.T. Wimmer. 1978. "The Economics of Mortality in North America, 1650–1910: A Description of a Research Project." *Historical Methods* 11:75–108.

Fogel, R.W., R. Floud, D.L. Costa, J.M. Kim, and C.J. Acito. 1996. "A Theory of Multiple Equilibria Between Populations and Food Supplies: Nutrition, Mortality and Economic Growth in France, Britain and the United States, 1700–1980." Unpublished manuscript. National Bureau of Economic Research, University of Chicago.

Fogelman, K.R. and O. Manor. 1988. "Smoking in Pregnancy and Development into Early Adulthood." *British Medical Journal* 297:1233–36.

Fraker, P.J., M.E. Gershwin, R.A. Good, and A. Prasad. 1986. "Interrelationships Between Zinc and Immune Function." *Federation Proceedings* 45:1474–79.

Fridlizius, G. 1984. "The Mortality Decline in the First Phase of

the Demographic Transition: Swedish Experiences." Pp. 71–114 in *Pre-Industrial Population Changes*, edited by T. Bengtsson, G. Fridlizius, and R. Ohlsson. Stockholm: Almquist and Wiksell.

Friedman, G.C. 1982. "The Heights of Slaves in Trinidad." *Social Science History* 6:482–515.

Fries, J.F. 1980. "Aging, Natural Death, and the Compression of Morbidity." *New England Journal of Medicine* 303:130–36.

———. 1989. "The Compression of Morbidity: Near or Far?" *Milbank Quarterly* 67:208–32.

Galloway, P.R. 1986. "Differentials in Demographic Responses to Annual Price Variations in Pre-Revolutionary France: A Comparison of Rich and Poor Areas in Rouen, 1681–1787." *European Journal of Population* 2:269–305.

———. 1987. "Population, Prices and Weather in Preindustrial Europe." Unpublished doctoral dissertation. Economics Department, University of California, Berkeley.

Gerver, W.J., R. De Bruin, and N.M. Drayer. 1994. "A Persisting Secular Trend for Body Measurements in Dutch Children. The Oosterwolde II Study." *Acta Paediatrica* 83:812–14.

Gidding, S.S., X. Xie, K. Liu, T. Manolio, J.M. Flack, and J.M. Gardin. 1995. "Cardiac Function in Smokers and Nonsmokers: The CARDIA Study. The Coronary Artery Risk Development in Young Adults Study." *Journal of the American College of Cardiology* 26:211–16.

Gille, H. 1949. "The Demographic History of Northern European Countries in the Eighteenth Century." *Population Studies* 3:1, 3–70.

Goubert, P. 1973. *The Ancien Regime*, translated by S. Cox. New York: Harper.

Gould, B.A. 1869. *Investigations in the Military and Anthropological Statistics of America Soldiers*. New York: Hurd and Houghton.

Hill, G.L. 1990. "Some Implications of Body Composition Research for Modern Clinical Management." *Infusionstherapie* 17S:79–80.

Hytten, F.E. and I. Leitch. 1971. *The Physiology of Human Pregnancy*, 2nd edition. Oxford: Blackwell.

Idiaquez, J. 1988. "Nutritional Status and Autonomic Nervous System Function." *Functional Neurology* 3:205–209.

Kannisto, V., J. Lauristen, A.R. Thatcher, and J.W. Vaupel. 1994. "Reductions in Mortality at Advanced Ages: Several Decades of Evidence from 27 Countries." *Population and Development Review* 20:793–810.

Kaplan, G.A. and J.T. Salonen. 1990. "Socioeconomic Conditions in Childhood and Ischaemic Heart Disease During Middle Age." *British Medical Journal* 301:1121–23.

Kim, J. 1993. "Economic and Biomedical Implications of Waaler Surfaces: A New Perspective on Height, Weight, Mortality, and Morbidity." Unpublished manuscript. Center for Population Economics, University of Chicago.

———. 1996. "The Economics of Nutrition, Body Build, and Health: Waaler Surfaces and Physical Human Capital." Unpublished doctoral dissertation. Economics Department, University of Chicago.

Laslett, P. 1983. *The World We Have Lost: England Before the Industrial Age*, 3rd edition. New York: Scribner.

Lee, R. 1981. "Short-Term Variation: Vital Rates, Prices, and Weather." Pp. 356–401 in *The Population of England, 1541–1871: A Reconstruction*, edited by E.A. Wrigley and R.S. Schofield. Cambridge: Harvard University Press.

Lozoff, B., E. Jimenez, and A.W. Abraham. 1991. "Long-Term Developmental Outcome of Infants with Iron Deficiency." *New England Journal of Medicine* 325:687–95.

Manton, K.G. 1993. "Biomedical Research and Changing Concepts of Disease and Aging: Implications for Long-Term Forecasts for Elderly Populations." Pp. 319–65 in *Forecasting the Health of Elderly Populations*, edited by K.G. Manton, B.H. Singer, and R.M. Suzman. New York: Springer-Verlag.

Manton, K.G., L.S. Corder, and E. Stallard. 1993. "Estimates of Change in Chronic Disability and Institutional Incidence and Prevalence Rates in the U.S. Elderly Population from the 1982, 1984, and 1989 National Long-Term Care Survey." Mimeographed document. Center for Demographic Studies, Duke University.

Manton, K. and B.J. Soldo. 1992. "Disability and Mortality among the Oldest Old: Implications for Current and Future Health and Long-Term Care Service Needs." Pp. 199–250 in *The Oldest Old*, edited by R.M. Suzman, K.G. Manton, and D.P. Willis. New York: Oxford University Press.

Manton, K.G., E. Stallard, and B. Singer. 1992. "Projecting the Future Size and Health Status of the U.S. Elderly Population." *International Journal of Forecasting* 8:433–58.

Martorell, R., J. Rivera, and H. Kaplowitz. 1990. "Consequences of Stunting in Early Childhood for Adult Body Size in Rural Guatemala." *Annales Nestlé* 48:85–92.

Mayr, E. 1982. *The Growth of Biological Thought; Diversity, Evolution, and Inheritance*. Cambridge: Belknap Press.

McGue, M., J.W. Vaupel, and N. Holm. 1993. "Longevity is Moderately Heritable in a Sample of Danish Twins Born 1870–1880." *Journal of Gerontology* 48:273–44.

McMahon, M.M. and B.R. Bistrian. 1990. "The Physiology of Nutritional Assessment and Therapy in Protein-Calorie Malnutrition." *Disease-a-Month* 36:373–417.

McNeill, W. 1971. *A World History*, 2nd edition. New York: Oxford University Press.

Meinhold H., A. Campos-Barros, B. Walzog, R. Kohler, F. Muller, D. Behne, and K. Steglitz. 1993. "Effects of Selenium and Iodine Deficiency on Type I, Type II, and Type III Iodothyronine Diodinases and Circulating Thyroid Hormones in the Rat." *Experimental and Clinical Endocrinology* 101:87–93.

Newhouse, J.P. 1992. "Medical Care Costs: How Much Welfare Loss?" *Journal of Economic Perspectives* 6:3–21.

Perrenoud, A. 1984. "The Mortality Decline in a Long-Term Perspective." Pp. 41–69 in *Pre-Industrial Population Changes*, edited by T. Bengtsson, G. Fridlizius, and R. Ohlsson. Stockholm: Almquist and Wiksell.

———. 1991. "The Attenuation of Mortality Crises and the Decline of Mortality." Pp. 18–37 in *The Decline of Mortality in Europe*, edited by R. Schofield, D. Reher, and A. Bideau. Oxford: Clarendon.

Piggott, S. 1965. *Ancient Europe from the Beginnings of Agriculture to Classical Antiquity*. Chicago: Aldine.

Pope, C.L. 1992. "Adult Mortality in America before 1900: A View

from Family Histories." Pp. 276–96 in *Strategic Factors in Nineteenth Century American Economic History*, edited by C. Goldin and H. Rockoff.

Preston, S.H. 1993. "Demographic Changes in the United States, 1970–2050." Pp. 19–48 in *Demography and Retirement: The Twenty-First Century*, edited by A.M. Rappaport and S.J. Schieber. Westport, CT: Praeger.

Rantakallio, P. 1988. "The Longitudinal Study of the Northern Finland Birth Cohort of 1966." *Paediatric Perinatal Epidemiology* 2:59–88.

Richards, R.J. 1992. "Evolution." Pp. 95-105 in *Keywords in Evolutionary Biology*, edited by E.F. Keller and E.A. Lloyd. Cambridge: Harvard University Press.

Richards, T. 1984. "Weather, Nutrition, and the Economy: The Analysis of Short Fluctuations in Births, Deaths, and Marriages, France 1740–1909." Pp. 357–89 in *Pre-Industrial Population Change*, edited by T. Bengtsson, G. Fridlizius, and R. Ohlsson. Stockholm: Almquist and Wiksell.

Robbins, S.L., R.S. Cotran, and V. Kumar. 1984. *Pathologic Basis of Disease*, 3rd edition. Philadelphia: Saunders.

Rowntree, B.S. 1901. *Poverty: A Study of Town Life*. London: Macmillan.

Saba, T.B., B.C. Dillon, and M.E. Lanser. 1983. "Fibronectin and Phagocytic Host Defense: Relationship to Nutritional Support." *Journal of Parenteral and Enteral Nutrition* 7:62–68.

Schieber, G.J., J.-P. Poullier, and L.M. Greenwald. 1993. "Must We Ration Health Care?" *Best's Review* 10:22–38.

Schmidt, I.M., M.H. Jorgensen, and K.F. Michaelsen. 1995. "Height of Conscripts in Europe: Is Postneonatal Mortality a Predictor?" *Annals of Human Biology* 22:57–67.

Schoeller, D.A. 1990. "How Accurate is Self-Reported Dietary Energy Intake?" *Nutrition Review* 48:373–79.

Schwartz, W.B. and H.J. Aaron. 1991. "Must We Ration Health Care?" *Best's Review* 91:37–41.

Scrimshaw, N.S. 1993. "Malnutrition, Brain Development, Learning and Behavior." The Twentieth Kamla Puri Sabharwal Memorial Lecture presented at Lady Irwin College, New Delhi, November 23.

Scrimshaw, N.S. and J.E. Gordon, eds. 1968. *Malnutrition, Learning and Behavior*. Cambridge: Massachusetts Institute of Technology Press.

Slicher van Bath, B.H. 1963. *The Agrarian History of Western Europe A.D. 500–1850*. London: Edward Arnold.

Stolnitz, G. 1955/1956. "A Century of International Mortality Trends: I." *Population Studies* 9:24–55.

———. 1956/1957. "A Century of International Mortality Trends: II." *Population Studies* 10:17–42.

Tanner, J.M. 1990. *Fetus into Man: Physical Growth from Conception to Maturity*, revised edition. Cambridge: Harvard University Press.

———. 1993. "Review of D.J.P. Barker's Fetal and Infant Origins of Adult Disease." *Annals of Human Biology* 20:508–509.

Thatcher, A.R. 1992. "Trends in Numbers and Mortality at High Ages in England and Wales." *Population Studies* 46:411–26.

Trewartha, G.T. 1969. *A Geography of Populations: World Patterns*. New York: John Wiley & Sons.

United Nations. 1953. "The Determinants and Consequences of Population Trends." *Population Studies,* Series A, No. 17.

U.S. Department of Health and Human Services. 1987. *Anthropometric Reference Data and Prevalence of Overweight*. Vital and Health Statistics, Series 11, No. 238. Washington, DC: U.S. Government Printing Office.

Vaupel, J.W. 1991. "Prospects for a Longer Life Expectancy." Presented at the annual meeting of the Population Association of America, Washington, DC.

Vaupel, J.W. and H. Lundström. 1994. "Prospects for Longer Life Expectancy." Pp. 79–94 in *Studies in the Economics of Aging*, edited by D. Wise. Chicago: University of Chicago Press.

Veblen, T. 1934. *The Theory of the Leisure Class: An Economic Study of Institutions*. New York: Modern Library.

Volpe, J.J. 1987. "Hypoxic-Ischemic Encephalopathy—Clinical Aspects." Pp. 236–79 in *Neurology of the Newborn*, 2nd edition. Philadelphia: Saunders.

von Meerton, M.A. 1989. "Croissance économique en France et accroissement des Française: Une analyse Villermetrique." Unpublished manuscript. Center voor Economische Studiën, Louvain, Belgium.

Waaler, H.T. 1984. "Height, Weight, and Mortality: The Norwegian Experience." *Acta Medica Scandinavica* 679S:1–51.

Weir, D.R. 1982. "Fertility Transition in Rural France, 1740–1829." Unpublished doctoral dissertation. Department of Economics, Stanford University.

———. 1993. "Parental Consumption Decisions and Child Health During the Early French Fertility Decline, 1790–1914." *Journal of Economic History* 53:259–74.

Wheeler, T., T. Chard, F. Anthony, and C. Osmond. 1995. "Relationships Between the Uterine Environment and Maternal Plasma Placental Protein 14 in Early Pregnancy." *Human Reproduction* 10:2700–704.

Wilmoth, J.R. and H. Lundström. 1995. "Extreme Longevity in Five Countries." *European Journal of Population/Revue Européenne de Démographie* 12:63–93.

Wrigley, E.A. 1987. 'Urban Growth and Agricultural Change: England and the Continent in the Early Modern Period." Pp. 157–93 in *People, Cities and Wealth: The Transformation of Traditional Society*, edited by E.A. Wrigley. Oxford: Basil Blackwell.

Wrigley, E.A. and R.S. Schofield, eds. 1981. *The Population of England, 1541–1871: A Reconstruction*. Cambridge: Harvard University Press.

Zaren B., G. Lindmark, and M. Gebre-Medhin. 1996. "Maternal Smoking and Body Composition of the Newborn." *Acta Paediatrica* 85:213–19.