Health, Nutrition, and Economic Growth*

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In his Ely lecture, D. Gale Johnson pointed out that improvements in agricultural productivity were the foundation of modern economic growth, which resulted in an increase in per capita income by more than 17-fold in Western Europe and by more than 51-fold in the United States between 1700 and 1998. Prior to 1800 about 80% of the population in these regions was engaged in agriculture and barely 11% of the population lived in cities of 5,000 or more persons. Life was nasty and short. As much as a third of infants born in Europe during the eighteenth century died, and life expectancy was probably between 25 and 30 years at birth. However, the enormous increase in agricultural productivity since 1700 not only permitted the population of the world to increase from 600 million to 6 billion but also rescued the majority of humankind from chronic malnutrition, greatly reduced the incidence of nutrition-related disabilities, and increased life expectancy at birth by more than twofold (Johnson 1997; Maddison 2001). In the remainder of this article I want to elaborate on these issues.

Today the most remarkable instances of rapid economic growth are in Asian and Latin American countries, which together represent more than half of the population of the world. This vast expansion of modern economic growth did not get under way until after World War II but since then has proceeded with lightning speed. These changes first became evident in the improvement in life expectancy. In the case of India, for example, life expectancy at birth increased from 29 years in 1930 to 60 years in 1990. An increase of that magnitude required two and a half centuries in England and France. The sharp drop in the death rates of developing nations led to an enormous surge in the growth of population, which in turn led to widespread fears that the food supply could not keep up with population growth and that industrialization would be thwarted.

None of these forecasts has turned out to be true. Quite the contrary, as table 1 shows, both worldwide and in the major subregions, the supply of

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food has exceeded population growth. Between 1961 and 2000 the per capita supply of food has grown by 24% worldwide. The increase in China, 61% between 1964 and 2000, has been the most remarkable achievement. However, despite some growth, the low food supplies of India and sub-Saharan Africa are still a cause of greatly elevated mortality. On the other hand, rich nations such as the United States and the members of the EU are consuming too much food, and many people, even among these countries’ lower-income classes, are dangerously overweight.

Of course, excessive food consumption on a wide scale is a recent phenomenon. As late as the middle of the nineteenth century, the British per capita caloric supply was at or below the average calorie supply of nations that the World Bank designates as “low-income economies.” The situation in France during the early nineteenth century was even worse. Its per capita caloric supply put it at the level of poorest nations today, such as Mozambique, Ethiopia, and Somalia. It was not until well into the twentieth century that France and Great Britain achieved the type of food abundance that is currently characteristic of rich nations (3,500 or more kcals).

Thus the escape from chronic malnutrition is a recent phenomenon even for the rich countries of the world, belonging mainly to the twentieth century. Recent bioclimatic studies have revealed that poor body builds increase vulnerability to diseases—not just contagious diseases, but chronic diseases as well. American males born during the second quarter of the nineteenth century were the first generation to reach age 65 in the twentieth century. They were not only stunted by today’s standards, but their average adult body mass index (BMI, a measure of weight standardized for height, usually calculated as weight in kilograms divided by height in meters squared) was about 15% lower than current U.S. levels. The implication of such poor body builds

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TABLE 1
CHANGES IN THE SUPPLY OF FOOD 1961–2000 (in Calories per Capita per Day)

<table>
<thead>
<tr>
<th>Region</th>
<th>1961</th>
<th>2000</th>
<th>% Increase</th>
</tr>
</thead>
<tbody>
<tr>
<td>World</td>
<td>2,255</td>
<td>2,805</td>
<td>24</td>
</tr>
<tr>
<td>Developed</td>
<td>2,947</td>
<td>3,266</td>
<td>11</td>
</tr>
<tr>
<td>Developing</td>
<td>1,929</td>
<td>2,677</td>
<td>39</td>
</tr>
<tr>
<td>Africa</td>
<td>2,097</td>
<td>2,428</td>
<td>16</td>
</tr>
<tr>
<td>Sub-Saharan</td>
<td>2,063</td>
<td>2,214</td>
<td>7</td>
</tr>
<tr>
<td>Asia</td>
<td>1,893</td>
<td>2,708</td>
<td>43</td>
</tr>
<tr>
<td>China</td>
<td>1,852*</td>
<td>2,979</td>
<td>61</td>
</tr>
<tr>
<td>India</td>
<td>2,073</td>
<td>2,489</td>
<td>20</td>
</tr>
<tr>
<td>Europe</td>
<td>3,023</td>
<td>3,265</td>
<td>8</td>
</tr>
<tr>
<td>EU</td>
<td>2,984</td>
<td>3,505</td>
<td>17</td>
</tr>
<tr>
<td>North America</td>
<td>2,713</td>
<td>3,431</td>
<td>26</td>
</tr>
<tr>
<td>United States</td>
<td>2,883</td>
<td>3,814</td>
<td>32</td>
</tr>
<tr>
<td>Mexico and Central America</td>
<td>2,297</td>
<td>2,935</td>
<td>28</td>
</tr>
<tr>
<td>South America</td>
<td>2,322</td>
<td>2,850</td>
<td>23</td>
</tr>
<tr>
<td>Oceania</td>
<td>2,852</td>
<td>2,941</td>
<td>3</td>
</tr>
</tbody>
</table>

SOURCE.—FAOSTAT.
* Consumption in 1964 is used because 1961 was a famine year in China. However, the world and regional averages are based on China’s 1961 consumption of 1,641 calories.
is brought out by table 2, which shows the average decline in the number of chronic diseases by age between 1900 and the 1990s.1 The American escape from hunger during the course of the twentieth century reduced the number of chronic conditions by about two-thirds at each age interval.2

Toward a Theory of Physiological Capital

Individuals who are stunted and wasted (i.e., have a very low BMI) are at a much higher risk of dying prematurely. Over the past 3 centuries, human beings in Organisation for Economic Cooperation and Development countries have increased their average body weight by over 50% and their average longevity by over 100%, and they have greatly improved the robustness and capacity of vital organ systems. I shall refer to this enhanced physiological capacity as physiological capital.

Physiological capital is a relatively new concept for economists. It differs from, but is related to, the better known concepts of human capital and health capital. The human capital concept was developed to explain differences in earnings by occupation, over the life cycle, by industries, and by regions. It focuses especially on the contribution of education (including on-the-job training) to an individual’s stock of human capital and to the rate of return on investments in education. The health capital concept was developed to explain the demand for goods and services that offset the depreciation in the initial endowment of health over the life cycle. Although health capital presupposes physiological capital, it does not deal with it explicitly. Health capital assumes the health stock that each individual is born with and considers how investment in health care can reduce the rate of depreciation in that stock. It does not address why some people have a greater initial stock than others, and it does not recognize the relationship between the size of the initial stock and its rate of depreciation. Nor does it take notice of the effect of the date of birth on the size of the initial stock or on the rate of depreciation. In other words, it does not confront the issue of how the average initial stock of physiological capital has been changing from one generation to another. However, all of these issues are central to the concept of physiological capital.

Since the beginning of the eighteenth century, physiological capital has been accumulating very rapidly. Much of this improvement is the result of a process that Dora Costa and I have called “technophysio evolution,” a syn-

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**TABLE 2**

<table>
<thead>
<tr>
<th>Age</th>
<th>c. 1900</th>
<th>1992–96</th>
<th>Average Annual Rate of Decline (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>50–54</td>
<td>3.3</td>
<td>1.0</td>
<td>1.3</td>
</tr>
<tr>
<td>55–59</td>
<td>4.5</td>
<td>1.4</td>
<td>1.2</td>
</tr>
<tr>
<td>60–64</td>
<td>5.6</td>
<td>1.6</td>
<td>1.3</td>
</tr>
<tr>
<td>65–69</td>
<td>6.2</td>
<td>1.9</td>
<td>1.3</td>
</tr>
</tbody>
</table>

Note.—Computed from the Union Army data base (http://www.cpe.uchicago.edu) and the Health and Retirement Study, or HRS (http://hrsonline.isr.umich.edu).
ergism between technological advances 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 still ongoing in both rich and developing countries (Fogel and Costa 1997).

The rapid accumulation of physiological capital is tied to long-term reductions in environmental hazards and to the conquest of chronic malnutrition (made possible by technophysio evolution). By environmental hazards I refer not just to greenhouse gases, which today are produced by automobile emissions and other uses of fossil fuels. Fossil fuels, mainly charcoal and soft coal, were also a severe problem at the turn of the twentieth century, but other forms of pollution were then perceived as even more severe. In New York City there were more than 200,000 horses in 1890, and pulverized horse manure that contained high concentrations of bacteria filled the air, contributing to respiratory and other infectious diseases. Indeed, the automobile was greeted by reformers of that age as a solution to the horse problem. Other severe environmental problems were the contamination of drinking water by fecal waste and the contamination of the milk and food supply, all of which promoted acute diseases with high death rates at young ages and, for survivors, early onset of chronic diseases at middle ages. It was advances in technology that solved many of these bygone environmental problems and laid the basis for the rapid accumulation of physiological capital during the twentieth century. This accumulation of physiological capital is reflected in the improvements in stature and BMI. Variations in height and weight are associated with variations in the chemical composition of the tissues that make up vital organs, in the quality of the electrical transmission across membranes, and in the functioning of the endocrine system and other vital systems. Nutritional status, as reflected in height and weight, thus appears to be a critical link connecting improvements in technology to improvements in human physiology (Fogel 2004b).

Research on this connection is developing rapidly, but the exact mechanisms by which malnutrition and trauma in utero or in 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. The principal evidence so far is statistical, and, despite agreement on certain specific dysfunctions, there is no generally accepted theory of cellular aging (Tanner 1990, 1993).

With these caveats in mind, recent research bearing on the connection between malnutrition and body size and the later onset of chronic diseases can conveniently be divided 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 excessive smoking or consumption of alcohol by pregnant women. Protein calorie malnutrition in infancy and early childhood can lead to a permanent impairment of central
nervous system function. Folate and iodine deficiency in utero and moderate-to-severe iron deficiency during infancy also appear to cause permanent neurological damage (Scrimshaw and Gordon 1968; Chávez, Martínez, and Sobreries 1995).

But not all damage due to retarded development in utero or infancy caused by malnutrition shows up immediately. D. J. P. Barker and his colleagues (1998) have reported that such conditions as coronary heart disease, hypertension, stroke, non–insulin-dependent diabetes, and autoimmune thyroiditis begin in utero or in infancy but do not become apparent until middle age or later. In these cases, individuals appear to be in good health and function well in the interim. However, early onset of the degenerative diseases of old age is linked to inadequate cellular development early in life.

Certain physiological dysfunctions incurred by persons suffering from malnutrition can, in principle, 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 consequences includes the degradation of tissue structure, especially in such vital organs as the lungs, the heart, and the gastrointestinal tract. In the case of the gastrointestinal system, atrophy of the mucosal cells and intestinal villi results in decreased absorption of nutrients. Malnutrition also has been related to impairment of immune functions, increased susceptibility to infections, poor wound healing, electrolyte imbalances, endocrine imbalances, and in adults, dangerous cardiac arrhythmias and increased chronic rheumatoid disorders (McMahon and Bistrian 1990).

Implications of Technophysio Evolution for Analysis and Measurement

Technophysio evolution implies that some theoretical propositions that underlie some current economic models are misspecified. For example, it is frequently assumed that individuals are born with a specific amount of health capital that depreciates over time. It is also assumed that the rate of depreciation depends on gross investments in health and on the level of health technology (which is assumed to be both exogenous to the individual and independent of the date of birth, i.e., neglects cohort effects; cf. Grossman 1972; Wagstaff 1986; Wagstaff and Dardanoni 1986). While these assumptions greatly simplify estimating procedures, they are inconsistent with accumulating evidence that successive birth cohorts are experiencing later onset of chronic diseases and disabilities, lower age-specific prevalence rates, and less severe conditions (Freedman and Martin 1998; Jette et al. 1998; Crimmins, Reynolds, and Saito 1999; Larson 1999).

The theory of technophysio evolution implies that individuals' initial endowments of health capital increased over the course of the twentieth century. This implication has been supported by recent research which has demonstrated that the curve of age-specific prevalence rates of chronic diseases has been shifting downward over the course of the twentieth century at what appears to be an increasing rate (Manton, Stallard, and Corder 1997; Reynolds,

If the theory of technophysio evolution is correct, some of the assumptions currently used by health economists and others to measure and analyze the contribution of health interventions to improvements in life expectancy are misleading. In the standard models, endowments of individuals at birth are assumed to be the same, regardless of the year of birth. Without investments in improving health capital, different birth cohorts are assumed to experience the same average rates of decline in their original health endowments (i.e., no allowance is made for the slower average rates of decline in the untreated endowments of different “vintages” of health capital). Another problem is that a single health technology is presumed to exist that is exogenous to the individual.

The 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.⁴

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 1990.⁵

The same set of considerations applies to efforts to explain the decline in disabilities during the twentieth century. The discovery that the average age of onset of disabilities is more than a decade later today than it was in 1900 focuses attention on factors that might have improved the health endowments of successive cohorts or might have slowed down rates of depreciation before remedial medical interventions became necessary.⁶

The theory of technophysio evolution is also useful in circumstances where the standard models of health capital provide a useful first approximation. Improvements in health capital by date of birth have income effects that would lead individuals to make greater investments in health services. They also have substitution effects because they reduce the relative price of an additional year of life expectancy. The amount that you need to spend on health care to add one year to life at later ages declines, as wear and tear
during developmental ages declines. Thus, life at later ages becomes relatively more attractive, holding prices constant, because the later onset of chronic disabilities and a slower natural rate of deterioration in health increases the discounted present value of a year of consumption at later ages.

It must be kept in mind that at the beginning of the twentieth century, disparities in health between rich and poor neighborhoods were much greater than they are today. If, for example, we use the infant death rate as an index of exposure to pathogens, the difference between the infant death rate in the worst and best wards of six large cities in 1900 was 226 per thousand. By 1950 this differential had declined to 38 per thousand. Thus in just half a century the differential in infant mortality between the healthiest and most sickly districts declined by 83% (Fogel 2004a).

**Implications of Enhanced Physiological Capital for Egalitarian Health Policies**

The more egalitarian health status that exists in rich countries today than existed in 1900 is probably due primarily to improved diets and other environmental improvements that enhanced the physiological capital of successive cohorts rather than to greater or more equal access to health care services. Support for this proposition is to be found in a series of recent studies that have linked events early in life, including the intrauterine period, to the onset of chronic conditions at middle and late ages. The strongest evidence for such links pertain to hypertension and coronary heart disease (Cresswell et al. 1997; Scrimshaw 1997; Barker 1998). A review of 32 papers dealing with the relationship between birth weight and hypertension (Law and Shiell 1996) showed a tendency for blood pressure in middle ages to increase as birth weight declines. Evidence of a connection between birth size and later coronary heart disease has been found in England, Wales, Sweden, India, and Finland (Frankel et al. 1996; Forsén et al. 1997; Koupilová, Leon, and Vågerö 1997; Stein et al. 1996).

The theory of a nexus between nutritional and environmental insults in utero or at early postnatal ages—and the risk of chronic health conditions half a century or more later—calls attention to the rapid improvement in the environment between 1890 and 1950. This period also witnessed improvements not only in the quantity but also in the diversity of the food supply throughout the year and the beginnings of dietary supplements that increased year-round consumption of vitamins and other trace elements. Two recent studies have shown strong correlations between month of birth and the longevity of middle-aged men in Austria, Denmark, Australia, and the United States that reflect differences in the availability of food to pregnant mothers over the seasons (Dobhammer and Vaupel 2001; Kanjanapipatkul 2001). These developments largely preceded the revolution in health care services that followed World War II.

Further evidence that a substantial part of the improvement in health status in the U.S. during the twentieth century was due more to better nutrition
and improved public sanitation than to direct medical care is provided by table 3. It shows that the proportion of white males who were still free of chronic conditions during ages 50–69 was substantially higher in the mid-1990s than a century earlier. This improvement was not due primarily to the curative interventions of health care providers with persons already afflicted with chronic diseases, because we are looking only at the subset of people who were not yet afflicted by chronic diseases. Table 4 shows that, for those who did develop four common chronic diseases, the average age of onset was between 7 and 11 years later for men who reached age 65 in the 1980s or later than for those who reached age 65 in 1910 or earlier (Helmchen 2003; cf. Costa 2000).

Although the more extensive and more effective medical interventions of the last third of the twentieth century in rich countries have contributed to the enhancement of the physiological capital of successive birth cohorts, their main contribution has been to slow down the rate of depreciation in the stock of the enhanced physiological capital that the members of these cohorts accumulated during developmental ages. This distinction between the increasingly enhanced physiological capital of successive cohorts and health care interventions that retard the rate of depreciation in the initial stocks of physiological capital in a specific set of cohorts is what is overlooked in the conventional theory of health capital.

The evidence currently in hand points to the importance of prenatal care

<table>
<thead>
<tr>
<th>Condition</th>
<th>Men Born 1830–45</th>
<th>Men Born 1918–27</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart disease</td>
<td>55.9</td>
<td>65.4</td>
</tr>
<tr>
<td>Arthritis</td>
<td>53.7</td>
<td>64.7</td>
</tr>
<tr>
<td>Neoplasm</td>
<td>59.0</td>
<td>66.6</td>
</tr>
<tr>
<td>Respiratory</td>
<td>53.8</td>
<td>65.0</td>
</tr>
</tbody>
</table>

Source.—Helmchen 2003.

Note.—Capacity to engage in manual labor was assessed by the surgeons who examined the veterans to determine their eligibility for pension. The size of the pension varied with the extent to which veterans’ disabilities reduced their capacity to perform manual labor (as defined by Congress and presidential directives). For further details, see the Center for Population Economics of the University of Chicago (CPE) Web site at http://www.cpe.uchicago.edu.
and environmental issues in both the enhancement of physiological capital and in affecting the rate of depreciation in that capital. Used in this context the word environment has a dual meaning. The uterus is a crucial environment for the developing embryo and fetus. And the quality of the external environment affects the environmental quality of the uterus. Pregnant women who are malnourished and exposed to high levels of pathogens and suffering, for example, from frequent bouts of diarrhea, will have intrauterine environments that retard the development of the children they are bearing. Various studies have revealed that the neonates of women who suffer from diarrhea are lighter—and at higher risk of perinatal mortality—than those of mothers who escape such diseases during their pregnancies (Mata 1978; Martorell and Habicht 1986).

Providing nutritionally stressed pregnant women with such supplements as folate, iron, and iodine will not only reduce the rate of spontaneous abortions but will also reduce the risk of permanent damage to the central nervous system of their fetuses. Counseling pregnant women to avoid smoking and alcohol consumption during pregnancy can reduce a major cause of low birth weight and of perinatal mortality. Effective measures to promote breast-feeding can substantially reduce morbidity and death rates among infants who are bottle-fed (Forste, Weiss, and Lippincott 2001).

The Contribution of Nutrition to Economic Growth: A First Approximation

The foregoing considerations suggest that to measure the impact of nutrition on economic growth it is necessary to incorporate both thermodynamic and physiological factors into the theory of economic growth. Viewed in the human growth context, both factors may be thought of as labor-enhancing technological changes that were brought about by developments in the agricultural, public-health, medical-services, and household sectors. They may also be thought of as adjustments for the mismeasurement of the labor input, when labor is measured only in person-hours.

The neglect of the relationship between body size and the food supply has obscured one of the principal sources of the long-term growth in labor productivity. The combined effect of improvements in the environment and in biomedical interventions over the past century has greatly improved the health of the population at middle and late ages. This proposition is supported by table 5, which shows the capacity of Union Army veterans circa 1900 to engage in manual labor. By ages 60–64, capacities had declined to about a third of what they had been at their peak, which is about half the proportions shown by current age-earnings profiles. It is also worth noting that peak earnings were reached at about age 35, which is about 15 years earlier than today.

Reconsideration of the issue starts with the first law of thermodynamics, which applies as strictly to the human engine as to mechanical engines. Since, moreover, the overwhelming share of calories consumed among malnourished
TABLE 5

<table>
<thead>
<tr>
<th>Age</th>
<th>Capacity to Engage in Manual Labor</th>
</tr>
</thead>
<tbody>
<tr>
<td>50–54</td>
<td>.75</td>
</tr>
<tr>
<td>55–59</td>
<td>.56</td>
</tr>
<tr>
<td>60–64</td>
<td>.34</td>
</tr>
<tr>
<td>65–69</td>
<td>.17</td>
</tr>
<tr>
<td>70–74</td>
<td>.08</td>
</tr>
<tr>
<td>75 and over</td>
<td>.04</td>
</tr>
</tbody>
</table>

Note.—Capacity at prime ages = 100.

populations is required for the basal metabolic rate (the amount of energy needed per unit of time to maintain temperature and the function of vital organs when the body is fully at rest) and essential maintenance, it is quite clear that in energy-poor populations, such as those of Europe at the beginning of the nineteenth century, the typical individual in the labor force had relatively small amounts of energy available for work. This observation does not preclude the possibility that malnourished French or English farmers worked hard for relatively long hours at certain times of the year, such as at harvest time. Such work could have been sustained either by consuming more calories than normal during such periods or by drawing on body mass to provide the needed energy.

It is quite clear, then, that the increase in the amount of calories available for work over the past 200 years must have made a significant contribution to the growth rate of the per capita income of countries such as France and Great Britain. That contribution had two effects—a thermodynamic effect and a physiological effect. The thermodynamic effect increased the labor force participation rate by bringing into the labor force the bottom 20% of the consuming units, who, even if highly stunted individuals with a low BMI, had only enough energy above maintenance for a few hours of strolling each day—about the amount needed for just 1 hour of heavy manual labor. Consequently, merely the elimination of the large class of paupers and beggars, which was accomplished in England mainly during the last half of the nineteenth century, contributed significantly to the growth of national product. The increase in the labor force participation rate made possible by raising the nutrition of the bottom fifth of consuming units above the threshold required for work, by itself, contributed 0.12% to the annual British growth rate between 1800 and 1980 (1.25^{0.00056} - 1 = 0.00124).3

In addition to raising the labor force participation rates, the supply of calories per equivalent adult male available for work increased from about 848 per day in 1800 to about 1,793 in 1980. However, not all of these calories contributed to output, as output is defined by the national income and product accounts (NIPA). Over the past 2 centuries there has been a considerable increase in the share of the discretionary day devoted to leisure. Hence, al-
though the calories available for work in England increased by about 109% per equivalent adult, the increases in calories used to produce output, as that term is defined by NIPA, is only about 41.7%. It follows that the increase in the amount of energy available for work contributed about 0.19% per annum to the annual growth rate of per capita income \( (1.417^{0.00556} - 1 = 0.00194) \).

Between 1800 and 1980, British per capita income grew at an annual rate of about 1.14%. Thus, through bringing the ultra poor into the labor force and through raising the energy available for work by those in the labor force, the thermodynamic effect explains about 30% of the British growth in per capita income over the past 2 centuries.

The contention that the British intensity of effort increased over time may seem dubious since the work day, week, and year (measured in hours) declined significantly over the past 2 centuries. However, the British (and other Europeans) could not have worked at the same average intensity per hour in 1800 as they do today, since that would have required a considerably larger supply of dietary energy per capita than was actually available. Increases in the intensity of labor per hour were also a factor in the American case, where food supplies were far more abundant than in Europe. Even if it is assumed that the daily number of calories available for work was the same in the United States in 1860 as today, the intensity of work per hour would have been well below today’s levels, since the average number of hours worked in 1860 was about 1.75 times as great as today. During the mid-nineteenth century, only slaves on southern gang-system plantations appear to have worked at levels of intensity per hour approaching current standards.

The physiological effect pertains to the efficiency with which the human engine converts energy input into work output. Nutritionists, physiologists, and development economists have contributed to the extensive literature on this topic. Since some important issues are unresolved, a firm assessment of the physiological contribution to economic growth is not yet possible. However, some aspects of the contribution can be indicated.

Changes in health, in the composition of diets, and in clothing and shelter can significantly affect the efficiency with which ingested energy is converted into work output. Reductions in the incidence of infectious diseases increase the proportion of ingested energy that is 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 has also 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 (increased the average value of the “Atwater factors,” to use the language of nutritionists). Improvements in clothing and shelter have also increased thermodynamic efficiency by reducing the amount of energy lost through radiation.

Individuals who are stunted but otherwise healthy at maturity will be at
an increased risk of incurring chronic diseases and of dying prematurely. To evaluate the significance of changes in the rate of deterioration of the capacity to work over the life cycle, one needs to calculate the effect of changes in stature and weight on the discounted present value of the difference between earnings and maintenance over the life cycle. A procedure for estimating this effect is set forth elsewhere, along with illustrative estimates of the key variables (Fogel 1994). The exercise indicates that the discounted revenues would have increased by about 37%. This last figure, combined with a guess on the effect of the shifting of Atwater factors, suggests that the average efficiency of the human engine in Britain increased by about 53% between 1800 and 1980. The combined effort of the increase in dietary energy available for work, and of the increased human efficiency in transforming dietary energy into work output, appears to account for about 50% of the British economic growth since 1800. In other words, the impact of nutrition on long-term economic growth accounts for most of the previously unmeasured increase in British total factor productivity.

This first approximation of the contribution of nutrition to economic growth probably errs on the low side. It neglects, for example, the contribution of nutrition to the impact of education on economic growth. Nutritionists and epidemiologists have demonstrated that the capacity of individuals to benefit from education depends on overcoming malnutrition, since malnutrition diminishes the effective operation of the central nervous system. Much remains to be done in measuring the contribution of nutrition to economic growth.

In this connection, the CPE has launched a new project, EXDID, to explain the decline in age-specific disability rates during the course of the twentieth century. This project requires the creation of a new data set which contains not only information on disability rates by small neighborhoods but on a variety of factors that influence changes in the process of aging, including the relative impact of improvements in the environment and of increasingly sophisticated biomedical intervention. EXDID will focus on the 24 largest cities in 1900 that collected information on key variables by wards and by census districts, which averaged about 20,000 and 8,000 individuals, respectively, at the beginning of the century. The focus on small neighborhoods is necessary because most of the decrease in disabilities took the form of closing the gap between prevalence rates in the sickliest and healthiest wards.

The EXDID database will consist of three components: (1) cross-sections of ward-level data throughout the century on acute and chronic diseases, bacterial counts in water, and food supplies (especially milk), the state of public sanitation, housing density, birth rates, and age-specific death rates by cause; (2) successive cross-sections of samples of birth records from hospitals, containing information on the health of mothers, prior pregnancy outcomes, the characteristics of the neonate at birth, and the health of both mothers and neonates during confinement, and various socioeconomic status variables (including income) of the parents. Anthropometric characteristics, illness, and perinatal mortality will be related to environmental characteristics of the wards.
in which the mothers lived; and (3) longitudinal samples of veterans of the Spanish-American War (SA) and of World War I (WWI), covering nearly all of their life cycles. These linked military, pension, and health histories will be drawn randomly from enlistees who resided in the wards of the 24 cities. It is worth noting that the SA veterans reached age 65 mainly between 1929 and 1946 and that WWI veterans reached age 65 mainly between 1948 and 1965. Consequently, in combination with the Union Army sample, EXIDID will make it possible to study differences in the process of aging during most of the twentieth century.8

References
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Notes
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1. Chronic diseases or conditions are long lasting and change gradually both at onset and subsequently. Chronic diseases may or may not be disabling or may or may not be controlled by medical interventions. The national Center for Health Statistics defines a chronic condition as one of 3 months' duration or longer.

2. For a more detailed discussion of these issues, see Fogel (2004b).

3. Costa has noted that the annual rate of decline in functional limitations between 1900 and 1980 was substantially below the rate of decline since 1980. That point is important because it bears on forecasts of the likely improvements in functional limitations during the twenty-first century. Another question arises: how much of the total decline in the burden of disease and functional limitation that occurred in the United States during the twentieth century took place before 1980 and how much since then? A reliable answer to that question requires new data sets that will provide a more detailed picture of the temporal pattern of changes in the burden of chronic disabilities for cohorts who turned age 65 between 1915 and 1980 than we now have. An illustration of what that division might be is suggested by Costa's estimate that functional limitation declined at 0.6% per annum between 1900 and 1980, and Manton and Gu's estimate that during the 1980s and 1990s the average rate of decline in disability was 1.7% per annum. Together these estimates suggest a total decrease of 56% in the burden of disability after age 65. About two-thirds of the decline took place before 1980 and one-third after 1980. This computation illustrates some of the problems that will need to be overcome in measuring and explaining the decline in disabilities during the twentieth century. The measure of functional limitations needs to be consistent over the century (Costa and Manton and Gu used different measures of disability). There is also an issue of how to define the severity of different sets of conditions in different social and economic contexts. It is likely that several alternative indexes will
have to be constructed, involving issues similar to those encountered in the construction of indexes of prices over long periods.

4. Among the exceptions are Rosenzweig and Schultz (1988) and Dasgupta (1993).

5. 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 Scrimshaw (1997), and Barker (1998).

6. Although I have focused on new technology for treatment, much has been done to prevent early onset of chronic diseases by promoting better nutritional habits and lifestyles.

7. 0.00556 = 1 ÷ 180.

8. For further information on the EXDID project, see the CPE Web site at http://www.cpe.uchicago.edu.