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9 Nutrition and the Decline in Mortality since 1700: Some Preliminary Findings

Robert William Fogel

9.1 The Issues

Between 1700 and 1980 there was a decline of about 35 points in the standardized American death rate (see table 9.1). Between the same years, the British rate declined by about 21 points. About 70% of the American decline and about 50% of the British decline took place before 1911.

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This paper is a progress report on two projects jointly sponsored by the National Bureau of Economic Research and by the Center for Population Economics of the University of Chicago. Aspects of the research reported here were supported by grants from the National Science Foundation; the Social Science Research Council, London; the British Academy; the Exxon Educational Foundation; the Walgreen Foundation; Brigham Young University; the University of California at Berkeley; Harvard University; Ohio State University; the University of Pennsylvania; Princeton University; the University of Rochester; and Stanford University. I have drawn on the work of fellow collaborators in the two projects including S. L. Engerman, R. Floud, G. Friedman, C. D. Goldin, R. A. Margo, C. Pope, K. Sokoloff, R. H. Steckel, T. J. Trussell, G. Villaflor, K. W. Wachter, and L. Wimmer. J. Bourne Wahl, C. Ford, M. Fishman, J. Moen, and J. Walker have been effective research assistants. C. Miterko efficiently typed and corrected the various drafts. A. M. John has generously made material from her study of Trinidad available to me, and D. Levy permitted me to cite some of the results of his study of life expectancy in colonial Maryland. I am especially indebted to J. M. Tanner for his encouragement and advice since the beginning of both projects and to P. H. Lindert for insightful comments and criticisms and for the correction of several errors in the draft presented at the Williamsburg conference.

An earlier version of this paper was commissioned by Gunter Steinmann and other organizers of the "Conference on Economic Consequences of Population Change in Industrialized Countries," which was held in Paderborn, West Germany, during June 1983. Successive versions of the paper were presented to seminars at Caltech, the London School of Economics, the Graduate Institute of International Studies (Geneva), Harvard,

Table 9.1 **The Probable Decline in Standardized Death Rates between 1700 and 1980 in the United States and Great Britain**

Approximate Date	United States	Great Britain
	A. Standardized Death Rates (per thousand)	
1. 1700	40	28
2. 1850	23	24
3. 1910	15	17
4. 1980	5	7
	B. Percentage of the Total Decline Which Occurred between ca. 1700 and the Specified Date	
5. 1850	49	19
6. 1910	71	52
7. 1980	100	100

Sources:
United States: The age distribution is standardized on the weights computed from persons alive in 1700 in the pilot sample of genealogies that is described in the next section of this paper. *Line 1*, Fogel et al. 1978, p. 76, with the New England and Chesapeake rates weighted by the New England and Southern populations for 1700 as given in United States Bureau of the Census 1975, p. 1168. *Line 2*, unpublished mortality tables for whites in 1850, cited in Haines, 1979. *Line 3*, Preston et al. 1972, pp. 728, 730. *Line 4*, United States National Center for Health Statistics 1983, p. 12.
Great Britain: The age distribution is standardized on the weights given in Wrigley and Schofield 1981, p. 529, for 1701–5; male and female death rates were equally weighted. *Line 1*, ibid. *Lines 2 and 3*, Case 1963, pp. 41, 53, 65, 76. *Line 4*, Great Britain Central Statistical Office 1983, p. 43.

The causes of this remarkable decline remain a puzzle. Until the mid-1950s it was widely attributed to improvements in medical technology. During the past 3 decades Thomas McKeown vigorously disputed that view in a series of highly influential papers and books. McKeown agreed that there had been a considerable expansion of hospital services and important advances in medical knowledge during the eighteenth and nineteenth centuries, but he argued that such ad-

Chicago, Birkbeck College, Minnesota, Northwestern, Pennsylvania, Princeton, Toronto, Rochester, and Indiana. Numerous revisions were made as a consequence of points raised during these sessions. I have also benefited from comments and criticisms by M. J. Bailey, R. K. Chandra, M. G. Coopersmith, E. Crimmins, J. Cropsey, P. A. David, L. E. Davis, N. Davis, G. R. Elton, A. Fishlow, R. A. Easterlin, F. Furet, D. Galenson, R. E. Gallman, H. Goldstein, M. R. Haines, R. Hellie, J. A. Henretta, S. Horton, T. A. Huertas, H. C. Johansen, D. G. Johnson, W. Kruskal, P. Laslett, E. P. Lazear, S. E. Lehmberg, M. Livi-Bacci, W. H. McNeill, L. Neal, D. C. North, G. H. Pelto, S. Peltzman, S. H. Preston, M. G. Reid, J. C. Riley, A. Sen, W. C. Sanderson, R. S. Schofield, T. W. Schultz, N. S. Scrimshaw, S. G. Scrikantia, J. L. Simon, S. Stigler, C. E. Taylor, B. Thomas, R. H. Tilly, E. van de Walle, S. C. Watkins, S. B. Webb, E. A. Wrigley, and W. Zelinsky.

The findings presented in this paper are tentative and subject to change. They do not necessarily reflect the views of the NBER or any of the other cooperating institutions or funding agencies.

vances had little effect on the decline in death rates until the twentieth century. An epidemiologist, McKeown gained prominence for biomedical research, including his studies of the relationship between birthweight and perinatal mortality rates in Birmingham after World War II (Gibson and McKeown 1950, 1951; McKeown and Gibson 1951), before turning his attention to long-term changes in medical practices and demographic rates.

9.1.1 The Nutritional Contribution: The English Experience

McKeown's explanation for the decline in mortality rates after 1700 is most fully set forth in his book on *The Modern Rise of Population* (1976a), and he subsequently restated and cogently summarized his argument in 1978 and 1983. In the place of medical technology, McKeown substituted improvement in nutrition as the principal factor affecting the decline in mortality. He does not make his case for nutrition directly but largely through a residual argument in which he rejects the other principal explanations. The alternatives to nutrition are advances in medical technology; reductions in the virulence of pathogens; human acquisition of immunity through natural selection, genetic drift, or acquired immunities; personal hygiene; and public sanitation.

McKeown's analysis turns on a careful consideration of the British pattern of decline in death rates due to specific infectious diseases between 1850 and 1971. During this period the standardized death rate attributable to infectious diseases declined from 13.0 per thousand to 0.7 per thousand. About 54% of the decline was associated with airborne diseases, 28% with water- and food-borne diseases, and 18% with diseases spread by other means (McKeown 1976a, pp. 54–63). This simple classification permits McKeown to assess the probable impact of public health measures and personal sanitation. Cleaning up the public water supply and improving sewage systems, he argues, would have had little effect on the airborne diseases. Moreover, as long as water supplies were polluted, individuals could not protect themselves against such waterborne diseases as typhoid and cholera by washing regularly. Under such circumstances "the washing of hands is about as effective as the wringing of hands" (McKeown 1978, p. 540). In his view public health measures did not become effective until the very end of the nineteenth century. The sharp declines in food- and water-borne diseases (which he dates in England and Wales with the start of the eighth decade) were not only due to better water and sewage systems but to improvements in food hygiene, especially pasteurization. He attributes the rapid decline of infant mortality between 1900 and 1931 mainly to the development of a "safe milk supply" (McKeown 1976a, p. 122; 1978, p. 540). McKeown argues that improvements in personal or public hygiene would not have reduced deaths from air-

borne diseases unless they reduced crowding, and crowding generally increased during the nineteenth century.

McKeown's skepticism about the efficacy of early medical measures is based on his study of the temporal pattern of decline in the death rates of the most lethal diseases of the nineteenth century. Tuberculosis, the leading killer in England and America during much of the nineteenth century, is a case in point. During 1848–54 tuberculosis caused nearly one out of every six English deaths from all causes, and one out of every four due to infectious diseases. It was not until 1882 that the tubercle bacillus was identified, and an effective chemotherapy for this disease was not developed until 1947. Nevertheless, the death rate of respiratory tuberculosis declined to just 43% of its 1848–54 level by 1900 and to just 10% of that level before the introduction of streptomycin in 1947. Similarly, the major decline in the death rates from bronchitis and pneumonia, whooping cough, measles, scarlet fever, and typhoid all preceded the development of effective chemotherapies. McKeown also doubts the efficacy of the lying-in hospitals which were established during the eighteenth and nineteenth centuries, noting that well into the third quarter of the last century "hospital death rates were many times greater than those of related home deliveries" (McKeown 1976a, p. 105).

McKeown is skeptical of the contention that the decline in mortality rates was due to a decline in the virulence of pathogens. He notes that scarlet fever and influenza have fluctuated in their severity in short periods of time and acknowledges that these fluctuations were due to changes in the character of these diseases. He lists typhus as another disease that might have declined due to changes in the pathogens. However, the fraction of the total decline attributable to these three diseases is small. On a more general plane he notes that infectious diseases that are now relatively benign in developed nations are still quite virulent in less developed countries and argues that it is quite unlikely that pathogens would have lost their virulence only in developed countries. McKeown also minimizes the impact of natural selection, arguing that in the case of tuberculosis too much of the population had been exposed to the bacillus for too long a period before the decline, and the decline itself was too rapid, to be consistent with natural selection.

McKeown's arguments in favor of a nutritional explanation fall into two categories. First, he cites evidence that per capita food supplies in England increased sporadically during the late eighteenth and early nineteenth centuries and then regularly in the late nineteenth and in the twentieth centuries. Second, he emphasizes findings of medical researchers currently working in the developing countries who have concluded that there is a synergistic relationship between malnutrition

and infection, and that malnutrition significantly increases the likelihood that a victim will succumb to an infection. In this connection he cites a report of the World Health Organization which concluded that malnutrition was an associated cause in 57%–67% of the deaths of children under age 5 in Latin America (1976a, p. 136).

9.1.2 The Nutritional Contribution: The American Experience

McKeown's argument has been extended to the American experience by Meeker (1972) and by Higgs (1973, 1979). According to Meeker, the period from 1880 to 1910 witnessed both a substantial rise in per capita income and a decline in mortality rates. In cross-sectional regressions for 1890–1900, city mortality rates are significantly related to housing density variables and state mortality rates are significantly related to income. In his 1973 paper Higgs estimated the decline in rural mortality rates for the period from 1870 to World War I. Despite the absence of direct observations on rural mortality, Higgs was able to infer a series by making use of three other series (the aggregate crude death rate, the urban crude death rate, and the share of the population that was urban) and an identity that related the rural crude death rate to these series. This procedure produced a rural crude mortality series which declined at approximately the same rate as the urban mortality series, the total decline over 50 years amounting to 30%–40%. Higgs argues that whatever role public sanitation and medical care might have played in the urban context, they were of minor consequence in rural areas which were undersupplied with physicians, and which continued to draw water mainly from wells, springs, and cisterns, continued to rely on privies, and continued to consume unpasteurized milk. Like McKeown, Higgs concluded that "the great bulk of the decline in rural mortality before 1920 is probably attributable to rising levels of living among the rural population" (1973, p. 189).

9.1.3 Objections to the Nutritional Argument

Virtually all those who are attempting to explain the secular decline in mortality rates in Europe and America agree that improvements in nutrition made a contribution. But some scholars believe that McKeown and others have greatly exaggerated the case (Livi-Bacci 1983). The doubts arise partly because of major gaps in the evidence. Razzell, for example, doubts McKeown's claim that the food supply in England grew more rapidly than the population before 1840. He argues that at least for the eighteenth century the evidence is "much more consistent with a reversed hypothesis—that the standard of the diet was a function of population change" (Razzell 1973, p. 8). Even more basic is the absence of adequate evidence on mortality rates. Before 1837 in Great Britain and before 1900 in the United States information on death rates

is so sparse that historical demographers are at odds not only on the levels of mortality but even on the direction of change (Vinovskis 1972; Easterlin 1977; Lindert 1983).

In the American case, for example, fragments of evidence led Thompson and Whelpton (1933) to believe that mortality rates declined fairly steadily from the middle of the eighteenth century to 1900. On the other hand, Yasuba's (1962) examination of available urban death registrations and some scattered registrations from rural communities led him to conclude that mortality rates increased between 1800 and 1860. More recently, a study of Deerfield, which has vital records that extend back to the early eighteenth century, revealed that mortality was low and stable within this rural town of western Massachusetts until the turn of the nineteenth century. Between 1795–99 and 1840–44, however, mortality rates nearly doubled (Meindl and Swedlund 1977, p. 398).

It is not merely the evidential gaps in the argument of McKeown and others that aroused the concern of critics. Certain facts seemed to contradict the case for nutrition. The absence of a significant gap between the mortality rates of the peerage and those of the laboring classes in England before 1725 was particularly vexing. "If the food supply was the critical variable," Razzell argued (1973, pp. 6–7), mortality reductions should have been "concentrated almost exclusively amongst the poorer" classes and the mortality rates of the aristocracy should have been "unaffected." Yet as table 9.2 shows, between the fourth quarter of the sixteenth century and the beginning of the second quarter of the eighteenth century, the mortality rates of the aristocracy were about as high as those of the general population. Both the high mortality rates of the nobility before 1725 and the rapid fall in these rates thereafter, although there was no apparent change in the diet of the peerage, predisposed Razzell "to look at the food supply hypothesis very critically."[1]

Efforts to relate both short- and long-term variations in the mortality rates to variations in bread or wheat prices have also undermined the nutritional explanation. Appleby's (1975) regressions, which related London deaths from specific diseases to bread prices over the period from 1550 to 1750, led him to conclude that there was no correlation between the supply of food and deaths due to plague, smallpox, or tuberculosis and only slight correlations between bread prices and deaths due to typhus and "ague and fever." More sophisticated analysis by Lee (1981) revealed statistically significant but weak relationships between short-term variations in death rates and in wheat prices. According to Schofield (1983, p. 282), short-run variations in English mortality were "overwhelmingly determined" by factors other than the food supply and the long-run trend in mortality was unaffected by the trend in food prices.

Table 9.2 **Cohort Life Expectancy (e_0^o) in the English Peerage and in the English Population as a Whole**

Birth Cohort (century and quarter)	Peerage (both sexes)	England and Wales (both sexes)
16th:		
III	38.0	35.6
IV	37.2	38.0
17th:		
I	34.7	37.3
II	33.0	35.5
III	31.9	34.2
IV	34.2	33.5
18th:		
I	36.2	35.1
II	38.1	33.8
III	40.2	36.3
IV	48.1	37.0
19th:		
I	50.6	41.5
II	55.3	44.6
III	58.6	
IV	60.2	
20th:		
I	65.0	

Sources: Column 1: Hollingsworth 1977, table 3. *Column 2:* The observations for 16–III through 18–IV are from Wrigley and Schofield 1981, p. 530; the observations for 19–II and 19–III are computed from the cohort life tables in Case et al. 1962, pp. 1–28, which were derived from registration data.

Lindert's (1983) examination of the work of Lee, Wrigley, and Schofield confirmed their conclusions on the absence of a notable relationship between food prices and mortality rates. Nevertheless, he was discontented with results that implied that living standards "left little or no mark on mortality." The puzzle, he acknowledged, extended to his own work with Williamson, since they have not yet been able to "find a firm casual link behind the obvious correlation between income and life expectancy after 1820." He suggested that the resolution to "the mystery of independent mortality" trends might require more complex attacks on the issue. That would be the case if the "life-extending" effect of income "was hidden behind the shift toward earlier death in the growing unhealthy cities." He also suggested that diets may "have improved in ways unmeasured by income" (pp. 147–48).

Other investigators have found evidence which suggests that McKeown underestimated the impact of public health measures on the decline in mortality during the nineteenth century. Estimates of the cause of mortality rates in the three largest urban areas of France during

the nineteenth century by Preston and van de Walle (1978) led them to the conclusion that water and sewage improvements played a major role in the urban mortality decline. Not only were the declines concentrated in the waterborne diseases, but the rate of decline was much more rapid in the two cities that introduced vigorous sewage and pure water programs than in the one that did not. On the other hand, deaths due to tuberculosis did not decline in Paris over a 33-year period, although deaths due to other airborne diseases showed small declines. Even these declines could have been due to the cleanup of the water supply. Preston and van de Walle stress that diarrheal and other waterborne diseases have important nutritional consequences because they "reduce appetite, reduce the absorption of essential nutrients, increase metabolic demands and often lead to dietary restrictions" (p. 218). Thus, cleaning up the water systems not only reduced deaths caused by waterborne diseases but also contributed to the reduction in deaths due to airborne diseases because the reduction in waterborne diseases improved the nutritional status of the population, especially of infants and young children.

9.1.4 The Concepts of "Nutritional Status" and "Nutritional Adequacy"

The last point calls attention to a terminological issue that has confused the debate over the contribution of improvements in nutrition to the decline in mortality. Although some investigators have equated the term *nutritional status* with the amount of food that is consumed, epidemiologists and nutritionists use the term in a different way. To them *nutritional status* denotes the balance between the intake of nutrients and the claims against it. It follows that adequate levels of nutrition are not determined solely by the level of nutrient intake but vary with individual circumstances. Whether the diet of a particular individual is nutritionally adequate depends on such matters as his level of physical activity, the climate of the region in which he lives, and the extent of his exposure to various diseases. As Nevin S. Scrimshaw put it, the adequacy of a given level of iron consumption depends critically on whether an individual has hookworm.[2] Thus, it is possible that the nutritional status of a population may decline even though that population's consumption of nutrients is rising if the extent of exposure to infection or the degree of physical activity is rising more rapidly. It follows that the assessment of the contribution of nutrition to the decline in mortality requires measures not only of food consumption but also of the balance between food consumption and the claims on that consumption. To avoid confusion, in the remainder of this paper I will use the terms "diet" and "gross nutrition" to designate nutrient intake

only. All other references to nutrition, such as "nutritional status," "net nutrition," "nutrition," "malnutrition," and "undernutrition" will designate the balance between nutrient intake and the claims on that intake.

9.2 New Sources of Evidence

The major obstacle to a resolution of the debate on the causes of the decline in mortality is the absence of data rather than the absence of analytical ingenuity or credible theories. Recognition of this point has led to numerous attempts to find sources of data that could fill the gap. The most impressive of these undertakings have been the work with parish records in England and France which have produced important new series on population and vital events that reach back to the first half of the sixteenth century. The publication of *The Population History of England* and of a summary of the second volume in the series (Wrigley and Schofield 1983) reveal that we are now coming into possession of a new long-term series that will greatly illuminate the evolution of demographic processes in England. Similar promise for French demography resides in the parish data assembled by INED, a part of which has been insightfully analyzed by Weir (1982), and in the new project based on the collection of a random sample of genealogies that has been launched by J. Dupâquier and his colleagues. Despite the demonstration by Henripin (1954) and his colleagues that genealogies could be used to reconstruct the population history of French Canada during the eighteenth century, historical demographers made little use of this type of evidence during the three decades following the publication of that study. The situation now appears to be changing. In Germany samples of genealogies are also being employed as the principal source of evidence in attempts to reconstruct long-term series on population and vital events in that nation (Imhof 1977).

It has been far more difficult to obtain data on standards of living and nutrition that could be used in conjunction with the demographic series that are now coming on line. Wrigley and Schofield (1981), for example, were forced to rely on a wage series of a small class of workers in a single region and to treat the price of wheat as a proxy for the consumption of food (cf. Thirsk 1983). This difficulty is also being addressed, and promising new sources of data on economic variables are now being exploited. It has recently been demonstrated that probate records, bailiffs' accounts, tax lists, and similar archival records can provide data on economic information suitable for both cross-sectional and time-series analysis. From these sources scholars have been able to measure such variables as grain yields, meat supplies, rental prices

of housing, changes in occupational structure, income, and wealth (Overton 1979, 1980; Lindert 1980; Schuurman 1980; Lindert and Williamson 1983a; Campbell 1983).

Although European scholars have led the way in the exploitation of many of the new data sources, Americans have not been far behind. Much of the work on this side of the Atlantic has been pioneered by historians of the colonial period who have exploited the full array of these difficult but now highly valued documentary sources to produce evidence on demographic, economic, and social behavior (Lockridge 1966; Demos 1970; Greven 1970; McCusker 1970; Smith 1972; Walsh and Menard 1974; Menard 1975; Kulikoff 1976; Fischer and Dobson 1979; Rutman and Rutman 1979; Carr and Walsh 1980; J. Gallman 1980; Jones 1980; McMahon 1981; Galenson 1981; R. Gallman 1982; Main 1982; Rothenberg 1984; Levy 1984). Although for the most part these studies have focused on local communities and particular periods, collectively they adumbrate regional and national patterns and demonstrate the feasibility of extending this approach to the national level and to the entire span of United States history.

In 1977 the NBER launched a new Program in the Development of the American Economy (DAE) which is investigating long-term changes in the United States economy that have occurred at the microeconomic level. To facilitate this objective the DAE has organized several studies of the feasibility of creating representative data sets consisting of intergenerationally linked households. Such data sets could open up entirely new possibilities for examining the interaction of economic and cultural factors and their mutual influence on such variables as the saving rate, the rate of female entry into the labor force, fertility and mortality rates, the inequality of the wealth distribution, migration rates, and rates of economic and social mobility. These data sets cannot be created from a single set of records but require the linking of several different types of records. The pilot studies have been aimed at determining whether the creation of the projected data sets is economically feasible and whether it is likely that such data sets will yield the desired information. The results to date have been encouraging on both counts.

9.2.1 The DAE/CPE Genealogical Sample

One of the projects in the DAE program is called "The Economics of Mortality in North America, 1650–1910." Jointly sponsored by the Center for Population Economics of the University of Chicago, this project turns on the collection of a large sample of genealogical records. The demographic information in the genealogical sample is being linked, on an individual or household basis, to economic information contained in probate records, tax lists, manuscript schedules of federal and local censuses, military and pension records, and eventually with medical

records. The projected size of the ultimate sample is approximately 1 million individuals in 200,000 families that will be linked intergenerationally for up to 10 generations (see table 9.3).

During the past 5 years we have retrieved a sample of approximately 80,000 persons who were born or entered the United States between 1640 and 1910. Our objective during this phase has been to investigate the various categories of genealogical records in order to determine which types of records would yield the most desirable properties and which are most cost effective. Of the various categories of genealogies that we have examined the two most promising are *published family histories* and *family group sheets*.

There are at least 40,000 published histories of families that contain information on over 20 million people who have lived in North America. The largest collection, with 24,000 volumes, is in the Library of Congress, but the New York Public Library, the Library of the American Antiquarian Society, the Genealogical Society Library in Salt Lake City, and the Newberry Library have extensive collections. We have surveyed the resources in these and other collections and have put information from a sample of the family histories into machine-readable form. At the present time the sample of published histories consists of about 65,000 individuals drawn from about 275 books.

Most of the family histories begin with an immigrant to North America or some other individual who may be viewed as a patriarchal or matriarchal figure. The book then records the descendants of this initial

Table 9.3 **Tentative Estimates of the Temporal Distribution of Observations in the Completed Genealogical Sample**

	Families Established during the Period		Persons Born or Entering United States during the Period	
Period	Number (1)	Percent (2)	Number (3)	Percent (4)
Before 1700	1,000	1	8,000	1
1700–1750	3,000	2	26,000	3
1751–1800	6,000	3	51,000	5
1801–50	34,000	18	246,000	26
1851 or after	144,000	77	608,000	65
Totals	188,000	100	939,000	100

Source: Fogel et al. 1978. A family is defined by a marriage of a bloodline individual, whether or not that marriage produces progeny. See Fogel et al. 1978, app. B, for a description of the simulation model on which this table is based. It should be kept in mind that children in one family are parents in the next one. Since col. 3 does not count such individuals twice, the ratio of col. 3 to col. 1 for a generation is not equal to the average size of completed families during the period covered by that row.

individual so that a descending tree or a pyramid is described within the family history. Dates of birth, death, and marriages are recorded in the family history, along with the place of each vital event, although omission of some vital information is common. The typical family history in the pilot sample covers six to eight generations and contains about 2,000 individuals. Families of New England are overrepresented in the histories but a significant number of books exist for each region of the country. The paucity of black family histories is the most serious shortcoming of this source. But the source is sufficiently diverse with respect to religion, European origins, places of settlement in North America, and period of immigration to be useful for studies of the white population.

We have experimented with a variety of strategies in sampling from these books. An initial concern was the distribution of the sample over the largest feasible number of books in order to insure geographic and other forms of diversity. More recently we have been experimenting with the recording of all of the information in a book, which may be the most cost-effective procedure. This new approach was encouraged by the discovery that whatever the initial location of the patriarch, subsequent generations were so mobile that each book generally had wide geographic coverage.[3]

Of the 75,000 observations in machine-readable form, only two-thirds have been integrated into the two files currently employed for demographic analysis. For the analysis of fertility we created an intergenerationally linked file of about 10,000 families embracing about 41,000 unduplicated individuals. The subsample currently being used to investigate mortality consists of about 19,000 individuals at risk from birth. About 15% of the individuals in these two working subsamples have been linked so far to economic information obtained from probate records and from the manuscript schedules of the federal censuses.[4]

Family group sheets are also family histories, but each sheet consists of just three generations. It is possible to link successive group sheets together in order to form longer genealogies, but we have not yet attempted to do so. So far we have used them mainly for the period between 1830 and 1900 when foreign immigration was heavy. The group sheets are well suited for that purpose since patriarchs who arrived during the second half of the nineteenth century would only have had one or two generations of eligible descendants. The family group sheets were constructed by Mormons, and there are about 10 million of these records in the files of the Genealogical Library in Salt Lake City. Although the compilers were Mormons, the ancestors included in the group sheets usually were not.[5] Much of our work with the group sheet sample has been concerned with whether its members are similar enough to the members of the published family histories to consider both sam-

ples as constituting a single pool of information. So far the results of our tests indicate that they do, and for many of our runs we have been pooling the two samples, although we continue to test for differences. The current working sample of group sheets consists of about 9,500 individuals who belonged to 1,500 families.

A priori considerations suggest that genealogies are likely to be a biased source of information on demographic and other socioeconomic characteristics. For example, it seems reasonable to assume that the probability that a family history will be constructed is proportional to the fertility of the family and inversely proportional to its mortality. It follows that genealogies may yield upward-biased estimates of fertility rates and downward-biased estimates of mortality rates. Whether the magnitude of such biases is large or small and whether they are correctable cannot, however, be determined on a priori grounds and the investigation of the direction and magnitude of various biases has been at the center of our work.

One approach to this problem has been to run a series of regressions of the form:

$$D_j = g_j(X_{ij}, B_{ij}), \tag{1}$$

where

D_j = a dichotomous variable for persons in the jth age group that takes the value one in the event of a death,
X_{ij} = the ith behavioral factor affecting the mortality rate of the jth group,
B_{ij} = the ith distortion in the data set which spuriously affects the probability of dying in the jth group.

The regressions described by equation (1) can be used to produce values of ${}_nQ_x$ corrected for the biases measured by the B_{ij}. At the present time only a proportion of the X_{ij} variables that we intend to consider have been brought into analysis. Still missing are the main economic variables, which we are now in the process of linking to the demographic variables. Nevertheless, the initial runs on age-specific risk of death (for each sex, on each of the seven age intervals, for each of four birth cohorts) are rather promising. Birth order is statistically significant and has a relatively large impact on the probability of dying in most of the age intervals, with first and last births having a higher probability of dying than intermediate births in families with at least four live births. Place of birth has a significant impact on the probability of dying, and the high-risk regions change over time.

The bias variables (B_{ij}) indicate that practices by the compilers of genealogies had a small but statistically significant effect on the measured level of risk. So far these biases do not appear to have had much

effect on the coefficients of the X_{ij}, generally changing the values only of the second or third significant digit. Much remains to be done, however, on investigating alternative ways in which the bias variables may be introduced into the regressions. But so far the impact of the various biases identified on a priori grounds appears to be small in well chosen and carefully screened genealogies. Even in the case of wealth, the upward bias in the genealogies is smaller than had been conjectured. Adams and Kasakoff have collected a sample of genealogies for northern states which they linked with the manuscript schedules of the 1850 census. They then computed mean wealth of the men in their sample who were age 20 or over in 1850, by occupation. Table 9.4 compares their results with the means reported by Soltow (1975) for his random sample from the 1850 census schedules. Table 9.4 shows that although the means in the sample of Adams and Kasakoff (1983) are biased upward as one would expect, the differences in means are not very large. Moreover, the large standard deviations indicate that the genealogies cover virtually the whole range of wealth holders.[6] Consequently, by including wealth as an argument of equation (1), it is possible to adjust for errors in estimates of mean mortality rates due to the overrepresentation of rich individuals and underrepresentation of poor ones.

Potential biases in the mortality rates because of the nature of different categories of genealogies raise more troublesome issues. One of the first issues we investigated was whether the family histories were truly family histories or merely pedigrees. Family histories include all of the descendants of the patriarch but pedigrees include only the direct ancestors of the compiler (his father, his grandfather, his great grandfather, and so on). Quite clearly pedigrees would bias mortality rates downward severely since the individuals in a pedigree had to live at least long enough to have procreated. The creation of a pedigree is the first step in the compilation of a family history since the compiler must

Table 9.4 **The Mean Value of Real Estate of Native-Born Males Age 20 and over in 1850 in Two Samples (in Dollars)**

	Genealogical Sample			Random Sample of 1850 Census		
	N	Mean	S.D.	N	Mean	S.D.
Farmers	325	1,547	1870	n.a.[a]	1,401	n.a.
Nonfarmers	276	1,037	2803	n.a.	805	n.a.

Source: Adams and Kasakoff 1983.

[a]Not available.

trace his lineage to the patriarch. Only then can he come forward in time to construct a complete family history. The simplest test of whether a book is a family history or a pedigree is to observe the fullness of the tree. Some books can be discarded because it is obvious that the compiler traced only a few lines. Such inspection will not, however, reveal more subtle omissions. To get at these we devised other tests, such as whether the number of lines that died out in a given genealogy was consistent with the predictions generated by reasonable guesses at appropriate life tables and fertility schedules. The results of the various tests have indicated that the majority of the family histories in our sample are indeed what they purport to be. It appears that once they determined who their patriarchs were, the compilers usually sought to fill in the entire family tree, although they were not always completely successful.

Other tests of potential bias have involved evaluation of the behavior of various fertility and mortality statistics in order to determine if they conform to patterns observed in comparable populations. We have, for example, compared age-specific fertility schedules and the mean birth intervals at various parities with those obtained from a variety of family reconstitutions and found them to be normal for noncontraceptive populations. We are currently comparing the age structure of the individuals in the sample who are alive at given dates with the age structure in censuses performed at the same date but have not yet completed these tests. We have also computed both period and cohort life tables from the data in our sample in order to determine whether the internal structures of these tables are consistent with known characteristics of life tables and these are (Bourne et al. 1984).

One such life table has been constructed for 920 native-born white males in the pilot sample who were at risk to die during the decade of the 1850s (see panel A of table 9.5). It should be emphasized that during the pilot phase of data collection the individuals included in the sample have been chosen in such a way as to be representative of the collections of genealogies that have been the focus of our concern. Consequently, the observations in the pilot sample are not necessarily representative of the national population to which the final sample will pertain. Although the individuals at risk during the 1850s come from all of the major regions, the Northeast is overrepresented and the South and Midwest are underrepresented. The rural areas are also overrepresented and urban areas are underrepresented. In principle the deficiencies in the sample could be remedied by reweighting each of the cells in an appropriate manner. I have not engaged in such an exercise for two reasons. First, the current sample is too small; on average there are only about 13 observations for each of the 70 cells that need to be

Table 9.5 **A Comparison of a Period Life Table for United States Males Derived from the Genealogical Sample with Two Other Period Life Tables**

Age (years)	A Native-Born Whites, 1850–60, Derived from the Genealogical Sample			B All Whites, 1850–60, Average of Haines's Tables for 1850 and 1860			C All Whites, 1900, Registration States		
	$1000Q_X$	l_x	e_x	$1000Q_X$	l_x	e_x	$1000Q_X$	l_x	e_x
10	29.4	1000	46.7	48.6	1000	46.6	38.3	1000	49.5
20	253.7	971	37.9	192.8	951	38.7	155.2	962	41.3
40	280.2	724	27.4	328.2	763	25.8	289.9	812	27.1
60	344.3	521	14.2	360.0	512	13.5	346.3	577	14.0
70	539.7	342	9.0	656.9	328	8.3	603.6	377	8.8
80	1000.0	157	4.5	1000.0	113	4.4	1000.0	149	4.7

Sources and notes: Panel A: See the text and n. 7 for the sources. The number of observations on which each ${}_nQ_x$ value was computed ranged between 126 and 212. The value of e_{80} was computed from the approximation in Coale and Demeny (1966, p. 20). *Panel B:* This table was built up from the average of the ${}_nQ_x$ values in the unpublished tables for 1850 and 1860 of Haines (1979). Since Haines did not estimate the ${}_{10}Q_{70}$, I used the value of ${}_{10}Q_{70}$ in Model West (Coale and Demeny 1966) consistent with $e_{10} = 46.6$. *Panel C:* Constructed from the ${}_nQ_x$ values in the 1900 life table in Preston et al. 1972. The use of longer age intervals in the estimation of l_x resulted in a value of e_{10} slightly below that reported in the source.

reweighted. Second, the life tables which are available for comparison suffer from sample selection biases which cannot, at present, be defined with the precision required for reweighting.

Panel B of table 9.5 presents the average of the 1850 and 1860 life tables recently estimated by Haines (1979) from the data in the censuses of mortality for these two years. Because these censuses suffer from substantial underreporting, Haines fitted model life schedules to data for persons aged 5–19, ages during which the reporting tends to be most complete. Nevertheless, it is still likely that the mortality rates in his tables are to some degree biased downward. The downward bias is likely to be present even at the ages he focused on because underreporting was greater in the South than in the North and because underreporting was severe in urban areas at all ages (cf. Kahn 1978; Condran and Crimmins 1980). Panel C presents the life table for 1900 constructed by the Bureau of the Census for the 10 original death registration states. Unlike the Haines tables it is difficult to know the direction of bias in this table because the biases run in both directions. The exclusion of the South from the original registration states tends to bias mortality rates downward. It was not until 1933 that all 48 states were included in the death registration system (United States Bureau

of the Census, 1975 p. 44). On the other hand, the states included in the original registration area are overrepresented, in comparison with the nation as a whole, in two high-risk groups: the foreign born and residents of large cities.

Table 9.5 indicates that life expectation at age 10 during the decade of the 1850s in the genealogical sample is 46.7 years, which is almost identical with the corresponding figure in the average of the Haines tables for 1850 and 1860 (46.6 years) and about 3 years less than that indicated by the 1900 table (49.5 years). These results are generally consistent with what is known about the extent of improvement in mortality between the 1850s and 1900. There are some differences in the ${}_nQ_x$ values between the genealogical sample and the average of the Haines tables, but because of the relatively small sample sizes, these are within the range of sampling variability. Sampling variability can, however, be reduced by further aggregation, and for this reason the preliminary findings presented in section 9.3 turn on 25-year averages (averages of five quinquennial intervals). All in all, the life tables derived from the genealogies conform well to those derived from registration data and other sources even before adjusting the sample for the underrepresentation of various sections of the reference population. The prospect for further improving the genealogical sample by both fuller sampling of underrepresented groups and various statistical adjustments is quite good.

9.2.2 The Height-by-Age Samples

One of the variables that we wanted to include in the regressions run on equation (1) was a measure of nutritional status. Measures of the mean consumption of various foods are so scarce for modern populations that it is unlikely that even the most assiduous search of archival documents would produce reliable annual estimates of the consumption of the principal nutrients for any significant number of individuals, certainly not for the whole span of time that we wish to consider. Moreover, since nutritional status depends not only on the amount of nutrients that are consumed but also on the claims against that consumption, a measure of food intake alone would be insufficient.

Fortunately, there is a class of measures that are relatively abundant, that reach far back into time, and that are sensitive to variations in nutritional status. Both laboratory experiments on animal populations and observational studies of human populations have led physiologists and nutritionists to conclude that anthropometric measurements are reliable indexes of the extent of malnutrition among the socioeconomic classes of particular populations. Measures of height and weight at given ages, the age at which growth of stature terminates, attained final height, and the rate of change in height or weight during the growing

ages "reflect accurately the state of a nation's public health and the average nutritional status of its citizens" (Eveleth and Tanner 1976, p. 1). Consequently, these measures are now widely used by the World Health Organization and other agencies to assess the nutritional status of the population of underdeveloped nations.

The use of anthropometric measures as measures of nutrition rests on a well-defined pattern of human growth between childhood and maturity. The average annual increase in height (velocity) is greatest during infancy, falls sharply up to age 3, and then falls more slowly throughout the remaining preadolescent years. During adolescence, velocity rises sharply to a peak that is approximately one-half of the velocity achieved during infancy, then falls sharply and reaches zero at maturity. In girls the adolescent growth spurt begins about 2 years earlier, and the magnitude of the spurt is slightly smaller than in boys.

This growth pattern reflects the interaction of genetic, environmental, and socioeconomic factors during the period of growth. According to Eveleth and Tanner (1976, p. 222),

> Such interaction may be complex. Two genotypes which produce the same adult height under optimal environmental circumstances may produce different heights under circumstances of privation. Thus, two children who would be the same height in a well-off community may not only both be smaller under poor economic conditions, but one may be significantly smaller than the other. . . . If a particular environmental stimulus is lacking at a time when it is essential for the child (times known as 'sensitive periods'), then the child's development may be shunted, as it were, from one line to another.

The relative importance of environmental and genetic factors in explaining individual variations in height is still a matter of some debate. For most well-fed contemporary populations, however, systematic genetic influences appear to have very little impact on mean heights. Thus, the mean heights of well-fed West Europeans, North American whites, and North American blacks are nearly identical. There are some ethnic groups in which mean adult heights of well-fed persons today do differ significantly from the West European or North American averages, presumably due to genetic factors. However, since such ethnic groups have represented a minuscule proportion of American and European populations, they are irrelevant to an explanation of the secular trends in mean adult heights in the United States and in the various European nations since 1750. Nor do they contribute significantly to differences, at various points of time, between the height means of the United States population and of the principal populations from which the United States population was drawn. In this connection,

it should be noted that today the mean final heights of well-fed males in the main African nations from which the United States black population is derived also fall within the narrow band characteristic of Western Europe (Eveleth and Tanner 1976; Fogel et al. 1983).

Biologists, epidemiologists, and nutritionists have charted the effect of nutritional deficiencies on the human growth profile. Nutritional insults in utero are reflected in birth length and birthweight. Short periods of severe undernutrition or prolonged periods of moderate undernutrition merely delay the adolescent growth spurt; severe, prolonged undernutrition may diminish the typical growth-spurt pattern and contribute to substantial permanent stunting. If undernutrition is both prolonged and moderate, growth will continue beyond the age at which the growth of well-fed adolescents ceases. Hence, average length at birth and in early childhood, the average age at which the growth spurt peaks, the average age at which growth terminates, the mean height during adolescent ages, and the mean final height are all important indicators of mean nutritional status (Frisancho 1978; Tanner 1978; Kielmann et al. 1983). Any one of these factors can be used to identify secular trends in nutrition. The more of these measures that are available, the more precise the determination of the severity and duration of periods of malnutrition.

In considering the relationship between nutrition and height, it is important to keep in mind that height is a net rather than a gross measure of nutrition. Moreover, although changes in height during the growing years are sensitive to current levels of nutrition, mean heights reflect the accumulated past nutritional experience of an individual over all of his growing years including the fetal period. Thus, it follows that when the final heights are used to explain differences in adult mortality rates, they reveal the effect, not of adult levels of nutrition on adult mortality rates, but of nutritional levels during infancy, childhood, and adolescence on adult mortality rates. Similarly, when heights at age 8 are related to mortality at age 8, the exercise reveals the effect of nutritional experience up to that age.

The measure of net nutrition represented by mean heights depends on the intake of nutrients, on the amount of nutrients available for physical growth after the necessary claims of work and other activities (including recovery from infections), and on the efficiency with which the body converts nutrients into outputs. The body's ability to generate a surplus for growth will vary with such factors as age, the climate, the nature of the available food, clothing, and shelter, the disease environment, the intensity of work, and the quality of public sanitation. In other words, the same nutritional input can have varying effects on physical growth, depending on environmental conditions. Consequently, mean height corresponds quite well to the type of measure of

nutritional status called for in section 9.1.4: it is a measure of the balance between food consumption and the claims on that consumption.

Some social scientists have suggested that height (or weight) at given ages should not be called measures of "nutritional status," but "generalized indexes of health" or "non-specific indicators of health status" (cf. Moseley and Chen 1983).[7] The definition of "nutritional status" that I have set forth here is not my own, but rather the definition employed by medical nutritionists, epidemiologists, and physiologists. It is an unfamiliar concept to most economists (and other social scientists) because we are not steeped in the medical literature. Moreover, "nutritional status" sounds too much like "diet" to most of us, although medical nutritionists and epidemiologists draw a sharp distinction between the two terms. To some social scientists the use of the term "nutritional status" seems to be a subtle way of supporting the oversimplified view that low levels of nutritional intake are the only sources of malnutrition, and so use of the term appears to give covert support to oversimplified theories of the relationship between diet and mortality.

Will the use of such alternative terms as "health" or "non-specific indicators of health status" avoid the problems? These terms have been advanced as though their meaning were unambiguous; yet as F. K. Taylor (1979) has pointed out, "health" is difficult to define rigorously. We can try to give it rigor by using available information on morbidity and mortality rates. Then "healthy" populations may be defined as populations having rates in these dimensions that fall within a "normal" range. The difficulty with this procedure is not only that reliable measures of mortality and, especially, of morbidity are often lacking; there is the more fundamental issue that "normal" ranges of morbidity and mortality vary so widely with time, place, and circumstances that epidemiologists often turn to anthropometric measures in order to determine what is "normal" in a given environment (Meredith 1970; Hytten and Leitch 1971; Goldstein 1976; Thomson and Billewicz 1976; Waterlow et al. 1977; Habicht et al. 1979; Naeye 1981; Raman 1981). So the mere substitution of one term for another, of "generalized indexes of health status" for "nutritional status," will not remove conceptual ambiguities or prevent oversimplified characteristics of the empirical relationships we seek to uncover.[8]

Quite the contrary, striking out on our own, without adequate attention to and connection with the extensive medical investigations on which we must base our own work, is far more likely to be misleading than acceptance of medical terminology that now seems strange or even questionable.[9] Long experience with the problems of the interrelationship between nutritional status and infectious diseases has led medical specialists to the conclusion that anthropometric measures are

the best single index of the average nutritional status of a population and of the relationship between undernutrition and the outcome of a significant range of infections (Habicht et al. 1979). Both clinical studies and laboratory experiments have shown that body wasting, retardation in the rate of physical development, and stunting are usually caused by undernutrition during the developmental years, which reduces both the rate of cell accumulation and the size of cells (Winick and Brasel 1980). These findings do not, however, imply that inadequate nutrient intake is the primary source of undernutrition or that undernutrition can be remedied merely, or even primarily, by increasing nutritional intake, since the source of the undernutrition may be a disease which makes it impossible for the body to assimilate those nutrients which are ingested.

Moreover, both laboratory experiments on animal populations and field studies of human populations have identified a set of infections whose outcome is sensitive to the nutritional status of the population at risk. The most carefully controlled of these field studies has been underway in the Narangwal district of India for more than a decade (Kielmann et al. 1982, 1983; C. E. Taylor 1982). The Narangwal project has revealed that perinatal mortality is particularly sensitive to the nutritional status of mothers. Perinatal mortality rates were reduced by more than 40% when the diets of pregnant women were supplemented by various nutrients, particularly iron and folic acid. Public health measures, such as the immunization of mothers for neonatal tetanus and improved delivery procedures, also were effective, contributing about half as much to the reduction in perinatal mortality rates as did nutritional supplementation. Infant mortality during the balance of the first year and mortality between ages 1 and 3 were also reduced by both measures. Interestingly, public health measures were more effective than nutritional supplementation in curbing late infant mortality, and the two types of intervention were about equally effective in curbing mortality at ages 1–3. Both nutritional supplementation and public health measures had marked effects on height and weight at given ages, and deviations from the 50th percentile of prevailing standards for height and weight were strongly correlated with death rates. Whatever the nature of the infections to which they were exposed, children whose diets were supplemented grew more rapidly up to age 3 (the final age of the test) than children in the control group.

In other words, improvements in nutritional status, whether the consequence of nutritional supplementation (which enables the body to resist infections) or medical intervention (which reduces the virulence of infections) were associated with reductions in morbidity and mortality rates. However, these associations are not identical or even symmetrical. Increases in nutritional intake and medical or public health

measures often have different effects on particular diseases (and these effects vary with different stages in the life cycle) as well as on the pattern of human growth. For the range of issues that we are exploring it is particularly important to stress that every infection affects nutritional status; the survivors of an infection suffer a deprivation of nutrients required for growth which slows down the rate of cell accumulation and reduces cell mass (Winick and Brasel 1980). It does not follow, however, that improvements in nutritional status necessarily reduce morbidity and mortality rates. Not all infections are nutritionally sensitive, and the body's capacity to resist a nutritionally sensitive infection may be inconsequential if the pathogen is sufficiently virulent. In other words, nutritional status is likely to be "a determining factor" in the outcome of an infection when that infection is both nutritionally sensitive and of an intermediate degree of virulence (*Journal of Interdisciplinary History* 1983, p. 506). Physiologists have also identified the "mechanisms responsible for the increased number and severity of infections in the malnourished host" (Feigin 1981, p. 18).

Furthermore, although mean height is a good measure of nutritional status, it does not by itself indicate whether fluctuations in net nutrition are due to fluctuations in the consumption of food, in the claims on the food intake, or in the efficiency with which food is converted into outputs. Such decomposition is possible because of the asymmetries to which I have referred. Although both infection and inadequate nutritional intake retard the process of growth, they do not do so in precisely the same way. Because the body draws more heavily on nutritional stores when it is fighting an infection than when it is not, an infection may cause growth to cease during a period of infection. However, if a child is normally well fed, and if there is sufficient time between infectious episodes, there will usually be full catch-up in growth when an infection ceases. Normal, well-fed children do not grow at equal daily rates but alternate periods of growth well in excess of the daily average with periods of little or no growth, as disease and other claims on nutritional intake wax and wane. In well-fed children with sufficient time between infectious episodes these lacunae in growth have no effect on final heights, because of full and rapid catch-up, but in malnourished children they contribute to permanent stunting (Fogel et al. 1983).

The more data which are available on heights at each age, the more numerous and disaggregated the links between age- and disease-specific death rates, not only with anthropometric data, but with a variety of other socioeconomic variables, the more complete the decomposition of the determinants of the decline in mortality will be, including the determinants of nutritional status. It is not easy to construct a database as varied and abundant as I have indicated, but the objective is not out

of reach. Some initial stabs at decomposition with the data currently in hand are undertaken for a few specific cases that are considered in sections 9.3.2, 9.4.2, 9.4.3, and 9.4.4.

The collection of a modest sample of height-by-age data was launched in early 1978 as an adjunct of the mortality project in order to produce a measure that could be employed in equation (1). However, it quickly became apparent that this body of evidence was filled with so much useful information on economic behavior that the scope of the sampling effort was enlarged and the work on this body of evidence became the foundation for a new NBER project called "Secular Trends in Nutrition, Labor Welfare and Labor Productivity," which is also cosponsored by the Center for Population Economics.

The nutrition project currently involves a set of 16 samples (see table 9.6) containing information on height by age, weight, and various socioeconomic variables. The samples, which cover the period from 1750 through 1937, reveal aspects of physical development in the United States, Trinidad, Great Britain, Austro-Hungary, and Sweden. Ten of the samples were drawn from military records and hence pertain to males of military age. One of the British samples is composed of poor teen-age boys taken in by the Marine Society, a charitable organization, from 1750 to 1910; another is composed of upper-class boys admitted to Sandhurst. Three of the samples contain information on both sexes from infancy to old age. One sample is of birthweights and lengths in Philadelphia from the 1840s to the end of the 1870s. The data in these samples are being linked with additional data obtained from probate records, tax lists, pension records, and manuscript schedules of censuses. Such linking increases both the range of variables that can be brought into the analysis and the complexity of the interrelationship between height, nutritional status, and economic and social behavior that we can investigate. As of mid-1984, information had been collected on about 400,000 individuals, which is about 70% of the anticipated final number.

Much of our work on the height data between 1977 and 1982 dealt with problems of estimating and correcting biases that arose from using military records to estimate the mean height of the population from which the recruits were drawn. These biases fall into three categories. First, there are the self-selection biases that are peculiar to volunteer armies. Then there are a variety of more general measurement biases, some relating to the accuracy of the age information and others to the accuracy of the height information. Finally, there is the bias that arises because military organizations may have height limits. Most frequently these organizational restrictions produced a jagged truncation of the left tail of the height distribution, but right-tail truncation is also encountered. Since we have published several papers dealing with the

Table 9.6 The Principal Samples in the Nutrition Project

Title of Samples	Number of Observations Originally Planned	Number of Observations Currently on Tape	Main Categories of Information Included	References
Civil War Samples:				
1. Union army, whites	40,000	53,000	Height, age, mortality, cause of death, various socioeconomic characteristics; covers mainly ages 18–45	Margo and Steckel (1983)
2. Union army, blacks	5,000	10,000	Same as 1, plus complexion	Margo and Steckel (1982)
3. Amnesty records, white southern males	5,000	5,000	Height, age, place of residence, occupation, ages 12–80	Steckel (1982a)
4. Union army, rejects	5,000	5,000	Same as 1 (except mortality information) plus reason for rejection	Steckel (1984b)
Other U.S. Samples:				
5. Regular U.S. army, 1790–1910	100,000	43,000	Same as 1	
6. Ohio National Guard, 1870–1925	13,000	13,000	Height, age, birthplace, residence, occupation, marital status, mainly ages 18–49	Steckel (1982b)
7. Coastwise manifests, 1807–62	75,000	51,000	Height, age, color, sex, dates, and points of embarkation and arrival; covers all ages of both males and females	Margo and Steckel (1982)
8. Colonial muster rolls, 1750–83	20,000	14,000	Same as 1, except no mortality information	Sokoloff and Villaflor (1982)

9. Philadelphia Alms House, 1847–77	4,500	3,000	Birthweight and birthlength, gestational age, birth order, characteristics of labor, mortality of child and mother during hospital stay, sex of child, race, characteristics of mother (age, ethnicity, residence, marital status, health status [e.g., venereal disease, drunkenness])	Goldin and Margo (1984)
10. Cost of living surveys, 1934–77	3,000	3,000	Height for all family members by age, sex, and various socioeconomic characteristics including occupation, wages, days ill, education, and family wealth	Goldin (1979)
11. Trinidad, 1813–34	30,000	25,000	Height, age, color, births, deaths, and various other socioeconomic variables for all ages and both sexes	Friedman (1982)
British Samples:				
12. Marine Society, 1750–1910		50,000	Height, age, and various socioeconomic characteristics including occupations of fathers and sons, literacy and vaccination (or inoculation); ages mainly 13–17	Floud and Wachter (1982)
13. Sandhurst boys, 1750–1910	11,000	11,000	Height by age, date of recruitment, fees paid	
14. Military recruitment records, 1750–1910	130,000	130,000	Same as 11, except for father's occupation	Floud (1983a)
Other European Samples:				
15. Swedish conscript rolls, 1750–1910	30,000	30,000	Height by age, years of service, and various other socioeconomic characteristics for males	Sandberg and Steckel (1979)
16. Hapsburg monarchy, 1720–1920	75,000	19,000	Height by age, occupation, residence, mainly ages 19–50	Komlos (1984)

techniques developed for coping with these problems (Trussell and Bloom 1979; Wachter 1981; Wachter and Trussell 1982; Fogel et al. 1982, 1983; Floud 1983a; Floud and Wachter 1983; Trussell and Wachter 1984), I will not attempt to describe them here but merely state that both simulation techniques and practical experience have demonstrated the effectiveness of the procedures.

9.3 Some Preliminary Findings on the Relation between Improvements in Nutritional Status and the Decline in Mortality

At present we have three pieces of evidence linking mortality rates with nutritional status (as measured by height). Before presenting these findings, I wish to reemphasize that they are provisional. Although the work on the height data is fairly advanced, we are still in the pilot phase of the drawing of the genealogical sample. The pilot sample is still relatively small, lacking in the geographical diversity we desire, especially before 1750, and only partly linked to the economic, medical, and social information that we will eventually have. Nevertheless, there are several suggestive findings that have arisen out of these data sets, and which appear to be sufficiently robust to warrant their presentation.

9.3.1 The Secular Trends in the Height and in the Life Expectancy of United States White Males, 1700–1930

Figure 9.1 compares the time series that we have developed so far in both the height and the life expectancy of United States white males. Before considering this diagram some characteristics of the series and their limitations should be kept in mind. First, the secular trend in height is controlled for shifts in the distribution of the region of birth, of occupation, and several other relevant characteristics while the life-expectancy series is not, but merely gives the mean life expectancy at age 10 of all of the individuals at risk during each period.[10] Second, Southerners are underrepresented in both the height and the life-expectancy series. The correction of these deficiencies, which we hope to make in the near future, will probably have a greater effect on the e_{10} series than on the height series, especially before approximately 1750.[11]

It is possible to estimate tentatively the effect of the mortality correction by making use of Levy's (1984) estimates of the life expectancy of Maryland legislators. The value of e_{10} estimated from his data for 1700–1749 and 1750–99 are shown in the lower portion of the diagram.[12] Also shown is the effect of averaging his observations and those in the genealogical sample, using weights that correct for the undercount of Southerners in the genealogical sample.[13] As can be seen, the impact of the correction will be greatest before 1750, partly because the dif-

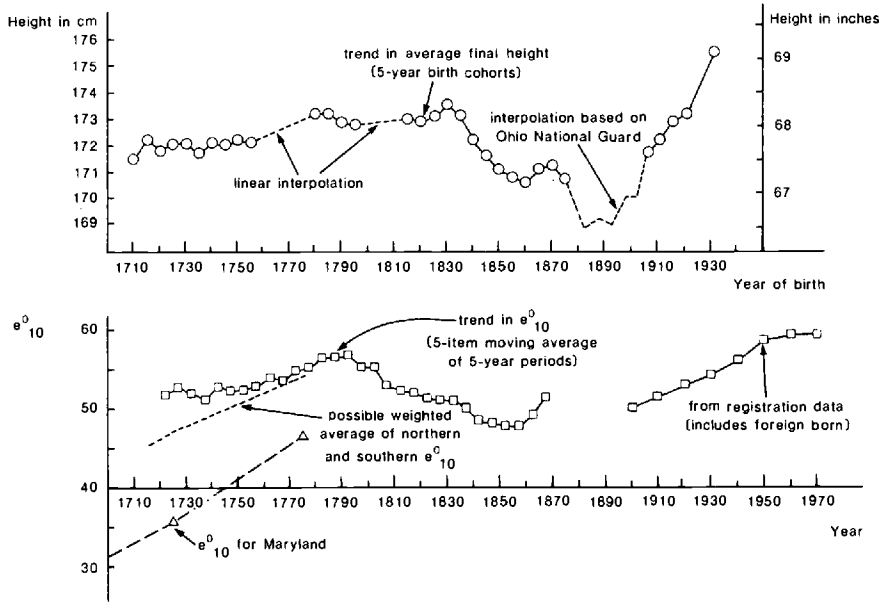

Fig. 9.1 A comparison between the trend in the mean final height of native-born white males and the trend in their life expectancy at age 10 (e^o_{10}) (height by birth cohort; e^o_{10} by period).

ferential in mortality between the regions closed rapidly during the first half of the eighteenth century and partly because the current representation of the South in the sample improves considerably after 1750. Thus, I expect a more representative sample to show a more rapid rise in e_{10} between 1700 and 1750. The beginning of the peak may be shifted by one or two decades and the level of the peak may be lowered slightly.

These corrections will not change the suggestive and unexpected similarities in the two series. Both series appear to be rising during most of the eighteenth century, attaining both substantially greater heights and life expectations than prevailed in England during the same period (Floud 1985a). 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 cohorts born between 1785 and 1820, but the sharp decline, which probably lasted about half a century, began with cohorts born around 1830. A new rise in heights, the one with which we have long been familiar, probably began with cohorts born during the last decade of the nineteenth century and continued for about 60 years.[14]

We do not, at present, have data on final heights in America for cohorts born before 1710, but the relatively flat profile between around 1710 and around 1750 and the tall stature compared with the English

in 1750 suggests that heights were probably rising rapidly for several decades before our series begins. This inference is supported by data on food consumption in Massachusetts discovered by McMahon (1981). Wills deposited in Middlesex county between 1654 and 1830 indicate a sharp rise in the average amount of meat annually allotted to widows for their consumption. Between 1675 and 1750 the average allotment increased from approximately 80 to approximately 168 pounds per annum: about half the increase took place by 1710. The evidence both on stature and on food allotments suggests that Americans achieved an average level of meat consumption by the middle of the eighteenth century that was not achieved in Europe until well into the twentieth century (McMahon 1981; Holmes 1907; Fogel 1986).[15]

Figure 9.1 and table 9.7 reveal not only that Americans achieved modern heights by the middle of the eighteenth century, but that they reached levels of life expectancy that were not attained by the general population of England or even by the British peerage until the first quarter of the twentieth century. Correction of the e_{10} series for the underrepresentation of Southerners may push the period estimate of e_{10} in around 1725 to about 47 years and the estimate for around 1775 to about 54 years, but these would still be remarkably high values for e_{10}. Although a more refined downward adjustment will eventually need to be made to obtain a reliable national average, there is at present no obvious reason for believing that the figures shown were not representative of the Northeast.

The early attainment of modern stature and relatively long life expectancy is surprising, and for that reason alone calls for further verification. Yet in light of the evidence that has accumulated in recent years it is by no means unreasonable. By the second quarter of the eighteenth century, Americans had achieved diets that were remarkably nutritious by European standards, and particularly rich in protein. The American population was low in density, probably below the threshold needed to sustain major epidemics of such diseases as smallpox. The low density probably also reduced exposure to the crowd diseases of the nineteenth century that took a heavy toll of life in both England and America. This is not to say that there were no epidemics in America between 1725 and 1800, but with the exception of a few port cities, outbreaks of epidemic diseases appear to have been much milder than in England.

The discovery of the cycling in both height and e_{10}, especially of the amplitude of the movements, is so new and so surprising that many issues will have to be pursued before doubts about the discovery can be set aside. Not least of the tasks is the need to enlarge the genealogical sample and to investigate characteristics that might be inducing spurious cycles or exaggerating the amplitude of the cycles in the uncon-

Table 9.7 **A Comparison among the Cohort Life Expectations for Native-Born United States White Males, British Peers, and the English Population, 1700–1925**

Birth Cohort (century and quarter)	(1) England and Wales (both sexes) e_0	(2) British Peerage (males) e_0	(3) British Peerage (males) e_{10}	(4) United States Native-Born Whites (males) e_{10}
18th:				
I	35.1	34.9	39.4	50.3
II	33.8	38.8	44.4	55.5
III	36.3	44.6	46.3	55.8
IV	37.0	46.9	46.1	51.9
		e_{10} (Males)		
19th:				
I		49.3	48.3	52.3
II	41.5 47.1	52.2	49.5	48.9
III	44.6 50.6	54.7	51.4	55.3
IV		53.7	47.4	
20th:				
I		60.1	54.0	56.9

Sources: Column 1: table 9.2, above. The two observations of e_{10} (males) for 19–II and 19–III were computed from Case et al. 1962 in the manner described in table 9.2. *Columns 2 and 3:* Hollingsworth 1977, p. 328. *Column 4:* The genealogical sample (N = 4,210) for all observations except 20–I, which is derived from United States registration data in the sources listed in appendix A and from United States National Center for Health Statistics 1983. The ${}_nQ_x$ values for late ages reached after 1980 were projections of the entries in the 1980 life table using the rate of decline in age-specific death rates obtained from medical records during 1968–78 and reported in Wilkin (1981). The entry for 20–I is the average of e_{10} for cohorts born in 1900, 1910, and 1920. This entry includes the foreign born, while all the other entries in col. 4 do not. Consequently, a comparison between 19–III and 20–I may understate the extent of the improvement in e_{10} for cohorts born in the United States during the first quarter of the twentieth century.

trolled trend. In this paper, however, it is the hitherto unsuspected pattern in the height series and its strong correlation with the mortality series that I want to emphasize.

9.3.2 Slaves, Poor London Boys, and Adult English Workers

The second piece of evidence linking mortality and nutritional status comes from data on slaves, on poor London boys, and on a more typical cross-section of English workers. Under abolitionist pressures the British colonial office conducted two registrations of slaves in Trinidad within a 20-month period, the first in 1813 and the second in 1815. Because the aim of the registrations was to prevent smuggling of slaves, physical characteristics, including height, were recorded. The second

registration also included information on the disposition of all the slaves who were registered in 1813. Friedman (1982) was the first to investigate the differences between the height of the slaves who died and those who survived. The difference is evident in table 9.8, which presents the heights of surviving and nonsurviving males under age 26. The extent of the difference is more apparent in a regression format. Table 9.9 shows that Trinidad-born males under age 26 who died between 1813 and 1815 were 1.2 inches shorter than those who survived. The corresponding figure for females is 0.9 inches.

Table 9.8 shows not only that nonsurvivors were shorter than survivors, but that even the survivors were exceedingly short by modern standards. Figure 9.2 indicates how bad their nutritional status was. In this diagram the heights of Trinidad-born male slaves, at ages from

Table 9.8 **Mean Heights by Age and Mortality, 1813–15, Trinidad-Born Males**

	Survivors			Nonsurvivors		
Age	Height	S.D.	*N*	Height	S.D.	*N*
0	23.9	3.29	118	22.2	3.44	26
1	26.6	3.38	159	26.4	2.91	30
2	29.9	3.22	131	28.1	2.96	16
3	33.8	3.09	177	33.1	3.75	11
4	36.2	4.09	158	36.7	2.90	11
5	38.6	3.39	128	37.0	4.38	8
6	41.2	3.72	134	39.9	2.27	7
7	43.0	3.22	119	43.2	4.09	5
8	44.5	3.95	104	45.0	3.16	5
9	46.8	2.70	67	44.5	3.54	2
10	49.7	3.75	110	42.0	—	1
11	49.9	3.25	70	—	—	0
12	52.3	2.75	84	54.0	—	1
13	52.7	3.34	60	52.0	—	1
14	56.1	3.96	68	59.5	0.71	2
15	58.3	3.86	59	60.0	—	1
16	59.4	2.99	43	59.0	1.41	2
17	61.6	4.05	30	—	—	0
18	62.5	3.05	50	61.5	2.12	2
19	63.7	2.87	18	—	—	0
20	64.6	3.29	48	64.0	5.66	2
21	64.8	2.17	16	—	—	0
22	65.0	3.07	40	—	—	0
23	66.3	2.69	9	—	—	0
24	65.3	2.99	20	—	—	0
25	65.2	3.00	33	65.0	—	1

Source: Friedman 1982. Age and height are those recorded in 1813. Owing to a transcription error the standard deviations of nonsurvivors were misreported in the original source. Those shown here, supplied by Friedman, are the correct ones.

Table 9.9 **Height Regression for Slaves under Age 26, Trinidad-Born (Dependent Variable = Slave's Height)**

	Males			Females		
Variable	Mean	Coefficient	*t* Ratio	Mean	Coefficient	*t* Ratio
Intercept	—	9.2	4.64	—	15.8	7.77
Age	6.7374	5.0927	27.05	6.8528	4.9525	26.05
Age^2	77.4127	−0.4148	−11.71	81.0743	−0.3533	−9.93
Age^3	1,155.665	0.0227	9.50	1,243.841	0.0177	7.47
Age^4	20,048.90	−0.0005	−8.90	22,013.99	−0.0004	−7.13
Number of slaves on the unit	75.6142	0.0054	0.95	76.8831	0.0006	0.10
Number of slaves^2	9,109.826	0.000016	0.63	9,349.246	0.000034	1.34
Sugar unit	0.6975	−0.1437	−0.42	0.6722	0.1958	0.56
Sugar × number of slaves	63.9059	−0.0032	−0.78	62.8478	−0.0031	−0.80
Cotton unit	0.04796	−0.0918	−0.22	0.0577	0.4115	1.02
Light child of dark mother	0.0496	0.9285	2.41	0.0432	−0.5840	−1.38
Creole mother	0.2436	2.9238	0.74	0.2803	−8.5058	−2.33
Creole mother × her height	15.0205	−0.0561	−0.87	17.1799	0.1420	2.38
Crude death rate on unit	0.0399	−4.5974	−1.85	0.0373	−3.1587	−1.12
Mother's height	60.8135	0.1983	6.13	60.7987	0.0830	2.49
Died 1813–15	0.0652	−1.1687	−3.33	0.0812	−0.8538	−2.61
Mean of dependent variable	40.3			40.4		
R^2			0.92			0.91
F-ratio			1,358.35			1,296.09
Degrees of freedom			1749			1857

Source: Friedman 1982.

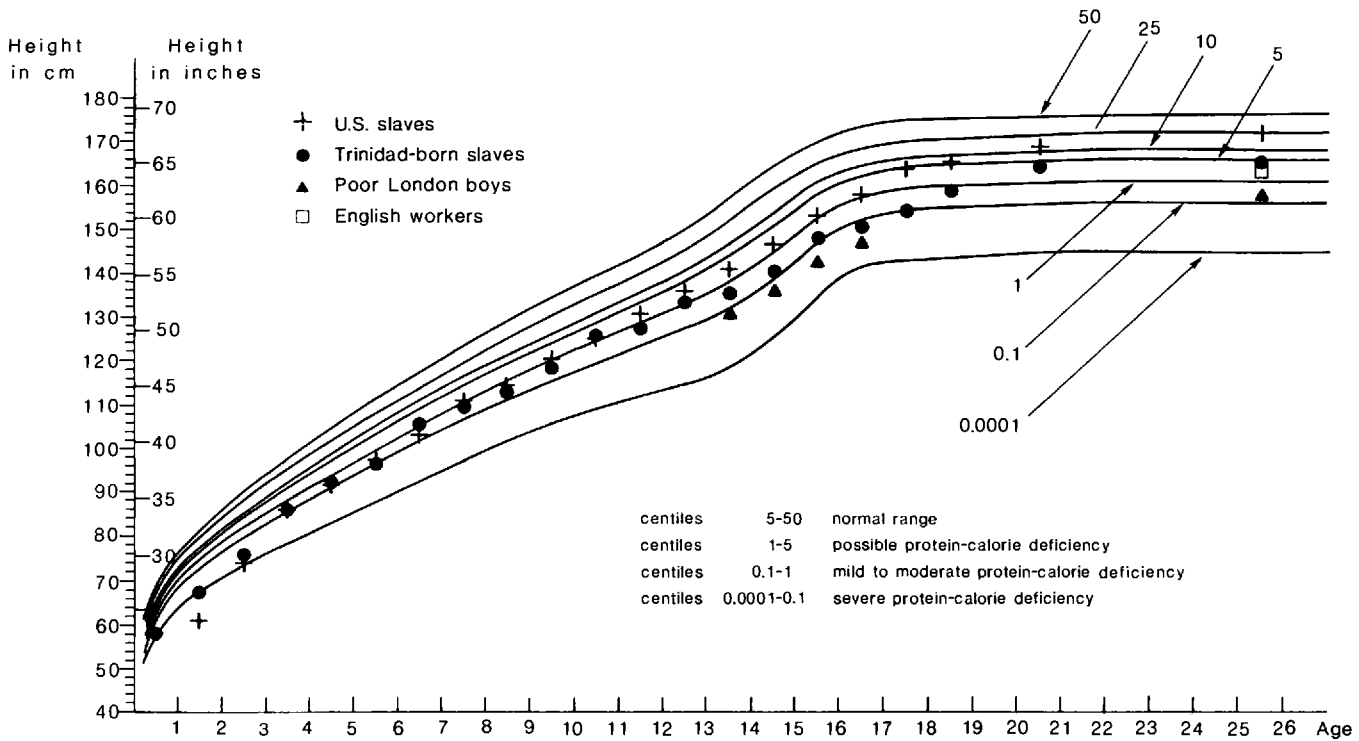

Fig. 9.2 The extent to which mean heights of male slaves, poor London boys, and English workers deviated from the modern height standard.

infancy to maturity, are superimposed on a set of curves which describe the current British standard for assessing the adequacy of physical development. The curve marked "50th centile" gives the average height at each age among generally well-nourished persons in Great Britain today. Also shown on the diagram are the heights of United States male slaves (which come from documents designed to prevent smuggling of slaves into the United States), the height of poor adolescent boys in London during the last half of the eighteenth century, and the height of more typical English workers at maturity about 1800.

Figure 9.2 shows that during early childhood slaves in both Trinidad and the United States were exceedingly malnourished. The figures for ages 0.5 and 1.5 are probably biased downward because the legs of the children were not fully stretched out when they were measured. But at ages 2.5 and 3.5 the children were walking and would have been measured in a standing position. Yet they were still exceedingly short by modern standards, falling at or below the 0.1 percentile. Such poor development is indicative of kwashiorkor and other diseases caused by severe protein-calorie malnutrition (PCM). Although the gap with modern height standards was reduced after age 3, it remained in a range suggesting at least mild to moderate PCM through age 8. Between ages 10 and 17 the growth patterns of United States and Trinidad slaves diverged, with the heights of United States slaves climbing into the normal range, while the heights of Trinidad slaves fluctuated in the range of moderate to severe PCM. By the mid-twenties, United States slaves were well into the normal range and Trinidad-born slaves were borderline normal. Thus, it appears that the diet that United States slaves received when they began working at adult tasks was good enough not only to sustain their work effort but to permit a substantial degree of catch-up growth as well. In the case of Trinidad slaves, however, the diet appears to have been inadequate to permit the same degree of catch-up, given the character of the physical environment.

Figure 9.2 suggests that nutritional deficiencies in utero and in early childhood, rather than the overwork or underfeeding of adults, were the main cause of the relatively high death rate of United States slaves. This possibility is supported by available data on the death rates. Figure 9.3 indicates that it was excess death rates of slave children under 5 that accounted for the difference between the overall death rates of United States slaves and United States whites during the late antebellum era. Moreover, the fact that United States slaves and whites had similar life expectancies after age 20 suggests that it was not the general virulence of the disease environment but conditions specific to young children. Campbell's (1984) examination of a large cotton plantation in Georgia revealed a correlation between the infant death rate and the intensity with which planters worked pregnant women. Steckel (1984a)

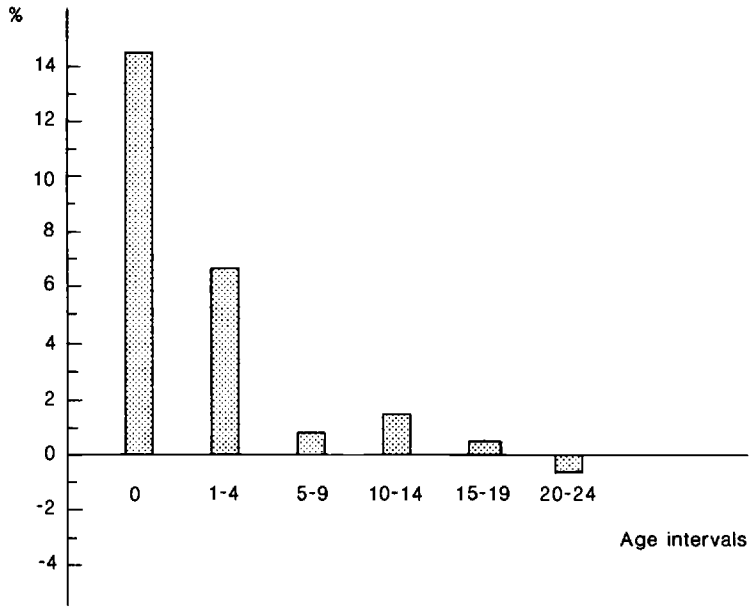

Fig. 9.3 The difference between age-specific death rates of U.S. slaves and whites, estimated for the late antebellum Era (slave death rate minus white death rate). *Sources:* Haines and Avery, 1980; Steckel, 1984a; Fogel, 1986.

has also found evidence that overwork of pregnant women increased the stillbirth and neonatal death rates. His examination of the monthly pattern of a sample of such deaths indicated that these rates were highest among the babies of women whose first trimester coincided with the planting season and who were in their third trimester when the peak period of harvesting occurred.

The small heights at ages 2.5 and 3.5 suggest not only that fetal malnutrition was prevalent but that chronic undernourishment was widespread during infancy and early childhood. Breastfeeding of slave babies was common throughout the South, but its average duration is uncertain. On some of the larger plantations most of the infants may have been at least partially weaned within 3 or 4 months. Plantation records which describe the diets of weaned infants and young children suggest that it was ample in calories but low in protein. Gruels and porridges, usually made with cornmeal and sometimes containing milk, were common fare. After age 3 these were supplemented to some extent by vegetable soups more likely to contain lard than meat, potatoes, molasses, grits, hominy, and cornbread. These more balanced diets contributed to catch-up growth between ages 3 and 8, although even the 8-year-olds were still quite short by modern standards (Fogel 1986).

Both the available descriptions of the diets of young children and the small stature of children, especially those under age 3, are consistent with the evidence on protein deficiency culled from the antebellum medical reports by Kiple and King (1981). They argue that frequent descriptions of the "glistening fat and corpulent paunches" of young children, the frequent listing of "dropsy" and "swelling" as a cause of death, and the concern of Southern physicians with "the distention of slave children's stomachs," suggest that kwashiorkor or prekwashiorkor was prevalent.

In Trinidad as in the United States the exceedingly small stature of slaves under 3 years suggests intrauterine malnutrition of fetuses. But in the case of Trinidad consumption of alcohol during pregnancy, which retards fetal development and induces a number of other abnormalities that are referred to as the Fetal Alcohol Syndrome, may have been a complicating factor. On sugar plantations liberal rations of rum were usually provided to slaves, especially during harvest time. Thus, although the absence of catch-up growth before age 3 may indicate that the early childhood diet was very low in protein, it could also reflect the residual effect of Fetal Alcohol Syndrome. However, since Trinidad slaves had a weaker adolescent growth spurt and a lower final height than United States slaves, the nutrients available for adolescent growth were obviously less in Trinidad than in the slave South. Not only was the nutrient intake of Trinidad slaves relatively low, but the more virulent disease environment of Trinidad undoubtedly exercised relatively greater claims against that intake. It is doubtful that adult slaves in Trinidad could have worked harder than United States slaves; the nutrient value of their diet would not permit it (Sheridan 1985). But in combination, the claims of work and disease and the dysfunctions caused by alcohol appear to have left Trinidad slaves with a lower net nutrition to sustain an adolescent growth spurt than United States slaves.

There are no measurements of the stature of the poor London boys during infancy or early childhood, but their heights between ages 13 and 16 are 1 to 2 inches less than those of Trinidad-born slaves of the same ages (Floud and Wachter 1982). Nor is it likely that much of this gap was made up during the late years of adolescence. These boys appear to have been drawn from the poorest section of the English working class—that one-fifth of English families that were unemployed or at best partially employed. They lived in the most crowded and virulent slums of London, and many were orphans or for other reasons lived with guardians (Floud and Wachter 1982). Evidence that the nutritional status of most English workers was superior to that of the poor London boys is also presented in figure 9.2, which shows the mean final height of the pool of men from which the recruits into the Royal Marines were drawn about 1800. Although this pool included

Londoners, most were residents of the southern and northern counties and of the Midlands. Artisans and craftsmen from both rural and urban areas were well represented in the ranks of the Royal Marines, and so were common laborers from both rural and urban areas. Analysis of this broad cross section indicates that the mean height of the English working class as a whole near the beginning of the nineteenth century was about 64.5 inches (Floud 1985a).

Tanner (1982) has estimated that the height of poor London boys at maturity was just 62 inches, about 3 inches below the adult height of Trinidad slaves. It thus appears likely that some combination of intrauterine malnutrition, poor weaning diet, and an adolescent diet inadequate to sustain catch-up growth (under the conditions of their environment) stunted the physical development of these poor London boys between 1750 and 1800. When Tanner assessed this evidence (1981, p. 158) he said that such short stature, which persisted into adulthood without an acute retardation of the teenage growth spurt, probably stemmed from conditions in utero and in early childhood: "Severe malnutrition of the pregnant mother followed by chronic and severe undernutrition of the infant could cause this result. More likely still is a low birthweight and/or a low weight gain in infancy caused by injurious substances breathed or eaten by the pregnant mother and the newborn child." The substances to which he referred included opium, laudanum, and morphia, which were the ingredients of popular patent medicines for children that are thought to have been widely used by mothers, unaware of their contents, to keep their children quiet while they worked at home or in factories (Pinchbeck and Hewitt 1969; Berridge and Edwards 1981).

Although malnutrition in utero and in early childhood may contribute to severe permanent stunting, it should not be assumed that these early experiences rigidly determine the entire pattern of physical growth. Such an inference is contradicted by the information in figure 9.2. Although both the Trinidad and the United States slaves were severely stunted in early life, their development patterns diverged markedly after age 10. Since the distribution of tribal origins of the United States and Trinidad slaves was similar, differences in their adolescent growth patterns were due principally to environmental rather than genetic factors (Steckel 1984a; Fogel 1986). Correlations between indicators of early childhood experience and later-life morbidity and mortality rates (Forsdahl 1977; Marmot et al. 1984) may thus reflect not so much the long reach of these childhood experiences as the normally strong correlations between childhood and later-life experiences, correlations that appear to have broken down in the case of American slaves. Thus it appears that even severe malnourishment in utero and in early childhood may be largely offset by improved conditions during adolescence.

9.3.3 Evidence from Regressions between Height and Mortality

Table 9.9 revealed a strong correlation between height and mortality in Trinidad. This relationship has been investigated further by John (1984), who ran a series of logit regressions relating the probability of dying between 1813 and 1815 to a number of variables including height. Among adults and children under age 15, the elasticity of the death for believing that the effect of height (or length) on mortality rates would be greatest for infants, especially neonates, although this proposition cannot be tested against the Trinidad sample because both infants and infant deaths were undercounted by margins that render them of little use.

Floud (1983b, 1985b) has assembled data for eight European nations over the years from 1880 to 1970 which permit an examination of the relationship between adult male height and mortality.[17] Equations (2) and (3) present the results of regressions which related both the crude death rates and infant mortality rates to height (numbers in parentheses are *t*-values):

$$(2) \qquad \hat{C} = \underset{(5.292)}{30.7877} - \underset{(-4.534)}{5.3851\hat{H}} - \underset{(-0.382)}{0.0363\hat{Y}} - \underset{(-4.040)}{0.006647T}$$
$$\bar{R}^2 = .85; \; N = 64$$

$$(3) \qquad \hat{I} = \underset{(12.327)}{88.9781} - \underset{(-10.797)}{15.9106\hat{H}} - \underset{(-3.294)}{0.3889\hat{Y}} - \underset{(-4.213)}{0.00837T}$$
$$\bar{R}^2 = .96; \; N = 64$$

where

C = the crude death rate per thousand
I = the infant mortality rate per thousand
H = adult male height measured in centimeters
Y = per capita income measured in United States dollars of 1970
T = time (year 1 = 1880)
$\hat{}$ = a hat over a variable indicates the natural logarithm of that variable

From these equations it can be seen that a 1% increase in height was associated with a change in infant mortality rates that was three times as large as the corresponding change in crude mortality rates. In both equations height has an independent effect on mortality rates, even after controlling for per capita income and time. Indeed, the addition of time to these regressions had virtually no effect on the coefficients of height, although it reduced the coefficient on per capita income in equation (2) by more than half, and in equation (1) it made the coefficient of per capita income statistically insignificant. Equation (2) implies that the rise in heights accounted for 39% of the decline in the infant mortality rate and per capita income accounted for another 27%, leaving

only about 33% attributable to the unknown factors which are measured by time.[18]

There is a question regarding the interpretation that should be placed on the coefficients of height and per capita income when both are included in the regression. Steckel's (1983) analysis suggests that when per capita income is held constant, height becomes a proxy for the degree of inequality in the income distribution. His regression on adult height implies that a 1% change in the Gini ratio (holding the level of income constant) had about four times as large an effect on mean heights as a 1% change in the level of per capita income (holding the Gini ratio constant).

Equations (2) and (3) suggest that height and income together were only about half as important in explaining the decline in the crude death rate between 1880 and 1970 as in explaining the decline in infant death rates in the eight countries covered by these equations. Even this last statement tends to exaggerate the effect of improvements in income and nutrition on the decline in adult mortality rates since infant death rates represent as much as a quarter of the crude death rate in high-mortality regimens such as those which existed in Europe during the nineteenth century. Of course, the crude death rate is a poor proxy for life expectancy since it is so sensitive to variations in age structure. Nevertheless, when considered in conjunction with the Trinidad regressions, equations (2) and (3) add to the evidence that the mortality rates of infants and and very young children are more sensitive to nutritional status than the mortality rates of adolescents and adults.[19]

9.4 Discussion

In combination, the several pieces of evidence make a fairly strong case for the view that nutritional status had a significant impact on mortality rates. Yet even those scholars who are skeptical of nutritional arguments acknowledge that nutrition is a relevant consideration. The real issues are the size of the nutritional contribution to the long-term decline in mortality and the locus of its impact. Much work remains before it will be possible to provide an adequate resolution of these issues. But I believe that a provisional estimate of the nutritional contribution is possible and might be useful.

9.4.1 A Provisional Estimate of Improvements in Nutritional Status on the Long-Term Decline in Mortality

I will first estimate the impact of improvements in nutritional status on noninfants. Because of the absence of data a more indirect approach is required for infants. This illustrative calculation will be applied to the British case.

Let us begin by supposing that the nutrition of working-class English males about 1800 had been improved to the point that they were able

to achieve modern stature. Then their mean final height would have increased from 64.5 to 69.5 inches, which is an increase of 7.75%.[20] How much impact would such an improvement in nutritional status have had on mortality? As previously noted, the Trinidad sample revealed that the elasticity of the death rate of non-infants with respect to height is about -1.4. Hence, a 7.75% increase in the final height of males would have reduced the non-infant death rate by about 11% ($-1.4 \times 7.75 = -10.85$).

The Trinidad registrations undercounted mortality so badly that they cannot be used to estimate reliably the elasticity of infant mortality with respect to height. We can circumvent this problem by using the schedule that relates the neonatal death rates to birthweight.[21] The probability of dying at given birthweights is very high at weights below 2501 grams (5.5 pounds). The schedule which relates the probability of dying to birthweight is stable below 2501 grams. It varies little from one socioeconomic group to another within a nation or even across nations. This stability is evident in figure 9.4.[22]

Mean birthweights vary greatly with the nutritional status of populations (Eveleth and Tanner 1976; WHO 1980). This point is illustrated in figure 9.5. The lines on this graph are normal approximations of the frequency distributions of birthweights.[23] Birthweight is represented on the vertical axis, and the horizontal axis represents z-scores (deviations of birthweight from the mean measured in units of the standard

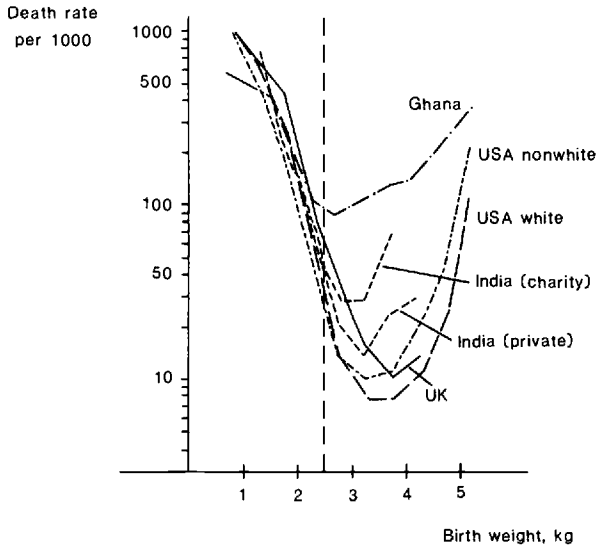

Fig. 9.4 Perinatal mortality by birth weight in Ghana, India, U.K., and United States. *Source:* Hytten and Leitch, *Physiology of Human Pregnancy* 2d ed.) p. 324.

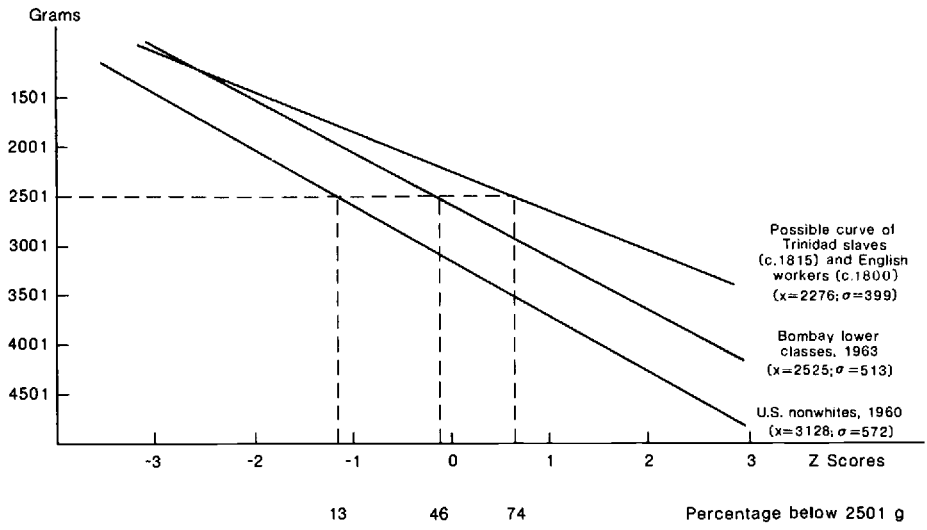

Fig. 9.5 The percentage of male births with weights below 2,501 grams in two modern populations and the possible percentage among Trinidad slaves and English workers during the early nineteenth century.

deviation). Hence, the cumulative frequency distribution is represented by a straight line. The lowest line represents the distribution of United States nonwhites in 1960. They had a mean birthweight of 3128 grams and, as indicated by figure 9.5, about 13% of the neonates weighed less than 2501 grams at birth. The second line is the distribution of birthweights for lower-class women in Bombay (Jayant 1964). Figure 9.5 indicates the mean birthweight in this population was just 2525 grams. In this case nearly half (46%) of the births were below the critical level, although the women in the sample were not the lowest of the low.

The third curve is my estimate of the probable distribution of the birthweights of the children of English workers about 1800.[24] In deriving this distribution I employed established correlations between height and birthweight as well as both published and unpublished information on the final heights of English workers developed by Floud and Wachter. These sources suggest that the distribution of the birthweights in this class around 1800 had a mean of 2276 grams, which is about 249 grams (about half a pound) below the average in the deliveries of the lower-class women in Bombay. It follows that about 79% of the births among English workers around 1800 were at weights below 2501 grams.[25]

The implication of this distribution of birthweights is revealed by table 9.10. Column 2 represents the actual schedule of neonatal death rates by weight for nonwhite United States males in 1950, and column 3 gives the actual distribution of their birthweights. The product of

Table 9.10 **Effects of a Shift in the Distribution of Birthweights on the Neonatal Death Rate, Holding the Schedule of Death Rates (by Weight) Constant**

Weight (grams) (1)	Neonatal Death Rate of Single Nonwhite U.S. Males in 1950 (per 1,000) (2)	Distribution of Birthweights of Single Nonwhite U.S. Males in 1950 ($\bar{x}$ = 3,128 g; σ = 572 g) (3)	Distribution of Birthweights in a Population with $\bar{x}$ = 2,276 g σ = 399 g (4)
1,500 or less	686.7	0.0117	0.1339
1,501–2,000	221.3	0.0136	0.2421
2,001–2,500	62.1	0.0505	0.3653
2,501–3,000	19.7	0.1811	0.2198
3,001–3,500	10.7	0.3510	0.0372
3,501–4,000	12.1	0.2599	0.0017
4,001–4,500	13.0	0.0865	—
4,501 or more	23.2	0.0456	—
Implied neonatal death rate (per 1,000)		26.8	173.0
Possible infant death rate (per 1,000)		48.9	288.3

Sources: Cols. 2 and 3: United States National Office of Vital Statistics 1954; *Column 4:* See nn. 21, 23, 24, and 26.

Note: The infant death rate in the last line of col. 4 is estimated at 1.67 times the neonatal rate.

these two columns yields an implied neonatal death rate of 26.8 per 1,000 which, of course, was also the actual death rate. If, however, this United States population had had the distribution of the birthweights of the English workers about 1800 which I have estimated, their neonatal death rate would have been 173.0 per thousand (see col. 3). The implication of table 9.10 is that improvements in nutrition sufficient to have shifted the mean birthweight from 2276 grams to 3128 grams would have reduced the infant death rate by 83% [1 − (48.9 ÷ 288.3) = 0.83].[26]

Equation (5) can be used to estimate the overall contribution of improvements in nutritional status to the decline in English mortality between 1800 and 1980.[27]

$$\dot{S} = \phi\dot{I} + (1 - \phi)\dot{S}_n, \tag{5}$$

where $\dot{S}$ = the counterfactual percentage decline in the standardized death rate due to improvements in nutritional status

$\dot{I}$ = the percentage change in the infant death rate due to improvements in nutritional status

$\dot{S}_n$ = the percentage change in the standardized non-infant death rate due to improvements in nutritional status

ϕ = the share of infant deaths in total deaths around 1800 as indicated by the data in Wrigley and Schofield.[28]

Substituting into equation (5), we obtain

(6) $$28 = 0.24\,(83) + 0.76\,(11).$$

Since the age-standardized death rate actually declined by about 69%, equation (6) implies that improvements in nutritional status accounted for about 41% of the total decline in the age-standardized English mortality rate since 1800. This figure is neither inconsequential nor everything. It shows that although improvements in nutrition made a substantial contribution to the decline in English mortality, other factors accounted for the majority of the decline. The main impact of the nutritional contribution was on the infant death rate. The reduction in noninfant deaths that may be attributed to nutrition account for just 12% of the total decline in English mortality since 1800.[29] Plausible upper and lower bounds on the variables in equation (5) indicate that 41 ± 10 probably bounds the nutritional contribution at all ages. It should be emphasized that these figures refer not merely to the diet but also to the other factors that affected the nutrients available for growth.[30]

9.4.2. A Possible Explanation for the Peerage Paradox

Although the calculation suggests a more modest role for nutrition than some have argued, other scholars may find even four-tenths is much too high a number, since the question about the peerage is still unanswered. If nutrition was so important, why did the English peerage have virtually the same mortality as the general population until the beginning of the eighteenth century? And why did life expectation of peers improve so rapidly after 1750 when no great change in their diet is apparent?

First, there is a general point which may not be of great quantitative significance in the resolution of the peerage paradox, but which bears on the context in which that issue ought to be considered. Some of the work on the changing epidemiology of Europe suggests that the potential leverage of nutritional status on mortality rates may have increased during the eighteenth and nineteenth centuries. Since nutritional status does not have an equal influence on the outcome of every disease, the prevalence of different diseases among different classes, and within different geographical areas, will obviously affect the impact of nutritional factors on fluctuations in mortality rates. Table 9.11 classifies diseases according to whether nutritional status is likely to influence their outcome. Taken in conjunction with studies of the epidemiology of medieval and early modern Europe by Creighton (1891),

Table 9.11 Nutritional Influence on Outcomes of Infections

Definite	Equivocal or Variable	Minimal
Measles	Typhus	Smallpox
Diarrheas	Diphtheria	Malaria
Tuberculosis	Staphylococcus	Plague
Most respiratory infections	Streptococcus	Typhoid
Pertussis	Influenza	Tetanus
Most intestinal parasites	Syphilis	Yellow fever
Cholera	Systemic worm infections	Encephalitis
Leprosy		Poliomyelitis
Herpes		

Source: JIH 1983.
Note: Outcome includes morbidity and mortality.

Helleiner (1967), McNeill (1976), Hatcher (1977), Appleby (1980), Kunitz (1983), and others, this classification suggests that diseases in which nutritional influence is minimal or equivocal (such as plague, malaria, smallpox, typhus, and influenza) may have had a greater impact on mortality before 1750 than after. For reasons that are still unclear, the prevalence of these diseases declined in Europe between 1700 and the mid-nineteenth century, and there was an increase in the prevalence of those infectious diseases in which the influence of nutrition is large. The possibility that there was a shift in the distribution of diseases suggests that the impact of nutritional status on mortality rates may have increased after 1750. However, whether such a shift actually took place, and its quantitative significance if it did, is still in dispute.[31]

A point more directly relevant to the resolution of the peerage paradox is that investigators searching for the possible influence of nutrition on the longevity of peers appear to have dwelt on the wrong issue: the diet of adults. As we have seen, nutritional status has its greatest impact on the mortality of infants, not on adults. That dukes and earls had an abundance of food as adults does not mean they were well nourished in infancy or in early childhood. Weanling peers of the eighteenth century did not eat joints of beef, but like weanling peasants, dined on a pap or watery gruel. During this era privilege and wealth did not ensure a diet or a nutritional status for the upper-class infants and young children that was better than that experienced by the common people. Although the housing, the clothing, and some aspects of the personal care of upper-class infants probably was better than that received by their lower-class counterparts, these advantages do not appear to have affected the infant and early childhood mortality rates of the peerage during the first half of the eighteenth century. Examination of the Hollingsworth (1977) mortality schedules indicates that 60% of the increase in the life expectancy between the cohorts of 1700–

1724 and of 1900–1924 was due to the decline in deaths under age 10. Indeed, if the peerage had continued to suffer the ${}_{10}Q_0$ value of 1700–1724 in 1900–1924, but experienced the improved mortality rates of the twentieth century at all other ages, then the life expectancy of the peers (both sexes combined) born during 1900–1924 would not have been 65.0 but only 46.4.

Furthermore, the fact the English nobility was rich enough to afford a good diet does not imply that they actually enjoyed a good diet. Considerable evidence suggests that, as G. R. Elton recently put it, the English nobility of the late medieval and early modern eras was often afflicted by "bad nutrition" (an abundant but unhealthy diet) while the lower classes often suffered from an "inadequate diet (hunger)."[32] Studies of per capita rations and annual food expenditures in the estate account books of noble households indicate that although aristocratic diets were abundant in calories and proteins, they were deficient in vitamins A, C, and D, owing partly to a dietary theory that viewed "greenstuff as a danger to health" and partly to a class prejudice against milk, cheese, and eggs (Dyer 1983, pp. 196, 207). Consequently, lower-class diets, though often inadequate in quantity, were often "more varied" than those of the upper classes. Peasants "ate all types of corn, not just wheat" and "the peasant table was more likely to carry the dairy produce and vegetables that the nobility despised" (Dyer 1983, p. 209). The hallmark of aristocratic diets before 1700, aside from their abundance (or superabundance) in calories and meats, was the regular availability of wines and spices.

The huge quantities of wine and ale consumed in aristocratic households, not only in England but throughout Europe, raises still another issue, one that bears on both neonatal and late-age mortality rates. The standard ration of drink for the inferior members of noble households was a gallon of ale per day, while the superior members drank both ale and wine, with wine constituting as much as half of the daily intake of fluids (Drummond and Wilbraham 1939; Pullar 1970; Wilson 1973; Dyer 1983; Thurgood 1984). So prominent were these alcoholic beverages in aristocratic diets that they accounted for one-quarter or more of the daily consumption of calories (Drummond and Wilbraham 1939; Heckscher 1954; Dyer 1983). The lavish consumption of ale and wine was stimulated by the fact that even in aristocratic households much of the fish and meat were heavily salted.[33] A diet heavy in salt and alcohol probably increased the incidence of liver, renal, gastrointestinal, and cardiovascular diseases among peers who survived to middle and late ages and may have contributed to the high mortality rates of peers at ages 40 and over. But it was in utero that dietary habits of the peerage were most deadly, since ladies of the realm were apparently consuming an average of between three and nine ounces of absolute

alcohol per day—more than enough to produce a high incidence of Fetal Alcohol Syndrome and Fetal Alcohol Effects.[34]

Thus, despite their enormous wealth and command over resources, the diet of the English aristocracy during the sixteenth and seventeenth centuries was deleterious to health. Their diet was bad partly because of nutrients that were excluded but mainly because of toxic substances that were included. Alcohol may have been the most lethal of these substances. In the quantities in which it was apparently consumed by pregnant women, it not only increased late fetal deaths and the neonatal mortality rate of the peerage, but also increased the risk to surviving infants into the second and third years of life (Abel 1982). By impairing the ability of the body to assimilate nutrients for 3 years or more after birth, Fetal Alcohol Syndrome severely exacerbated the deficiencies in the weaning and early childhood diets and may have undermined the health of many peers for the balance of the life cycle.

The peerage paradox thus appears to have arisen from the mistaken proposition that because the adult diet of English aristocrats was abundant in grains and meat, it was a "good" diet. That proposition overlooked the role of toxic substances in upper-class diets, substances which were harmful to adult health and disastrous for fetal development as well as for development during infancy and early childhood. The proposition also overlooked the critical nature of the weaning diet, which was as inadequate for peers as for peasants. These neglected factors are certainly relevant to an explanation of the exceedingly high infant and childhood mortality rates of the peerage before 1725. They also help to account for the decline in these mortality rates after 1725. The relevant point here is not merely what was added to the aristocratic diet between 1700 and 1900 but also the gradual elimination from that diet of the toxic substances that were so lethal to the young and the unborn (Mingay 1963; Pullar 1970; Wilson 1973). The children of the upper classes were the beneficiaries of the reform in manners that were in part a lagged response to Puritanism and other austere religious movements of the seventeenth century, in part a reflection of the Enlightenment, and in part a reaction to the devastating consequences of the gin mania of 1720–50 (Trevelyan 1942; Warner and Rosett 1975). One of the by-products of this reform that affected both the nutritional status and mortality rates of infants was the gradual disappearance of the upper-class practice of putting out infants with wet nurses (Mingay 1963; Pullar 1970; Sussman 1975, 1977; Stone 1977; Trumbach 1978; Flinn 1981).

The suggested resolution to the peerage paradox points to the need for further research on past practices that affected nutritional status during infancy, the weaning ages, and in utero. What was the temporal pattern by which the new manners diffused through the upper classes

of England, and how did this pattern affect the consumption of alcohol, opiates, and other toxic substances by pregnant mothers, infants, and young children? Was it fashionable in court during some periods for pregnant women to keep their weight gain low? To what extent did the weaning diet of peers expose them to virulent infections from contaminated water or raw milk, and when did this risk diminish? Research into these issues should, of course, be extended to cover the experience of the lower as well as of the upper classes. Study of the leads and lags between changes in the cultural standards of the upper and lower classes may go a long way toward explaining the leads and lags between the changes in their respective mortality rates.

9.4.3 The Wrigley-Schofield-Lee Paradox

In interpreting the regressions between mortality rates and wheat prices, it has often been assumed that the price of wheat was so highly correlated with all other grain prices that it could serve as a proxy for the price of food. It has also been assumed that food shortages would be reflected in their price. Although the second assumption is quite reasonable, it does not follow that a large rise in prices necessarily implies an equally large decline in the supply of food. That would be the case only if the demand elasticity (ϵ) for food was one. However, if the demand elasticity for grains was only between 0.25 and 0.1, then even the so-called exceptionally high fluctuations in grain prices (20% or more above trend) would imply shortfalls in grain yields that were only between 2% and 5% below the trend (see table 9.12). With a highly inelastic demand for grains, even the weak relationship between mortality and wheat prices found by various investigators would be consistent with the nutritional case. Their regressions would then imply that mortality rose even when declines in the supply of food were quite small. Thus, the critical question raised by the studies of correlations between wheat prices and mortality is the size of demand elasticities for wheat, for grains, and for food as a whole in England during the early modern era.

Table 9.12 **The Changes in the Price of Grains Associated with Changes in the Quantity, for Elasticities of 1.0, 0.5, 0.25, and 0.1**

Percentage Increase in Price	Percentage Decline in Quantity of Grain if			
	$\epsilon = 1$	$\epsilon = 0.5$	$\epsilon = 0.25$	$\epsilon = 0.1$
10	9.1	4.7	2.3	0.9
20	16.7	8.7	4.5	1.8
30	23.1	12.3	6.3	2.6
40	28.6	15.4	8.0	3.3
50	33.3	18.3	9.6	4.0

The analytical framework recently developed by Amaryta Sen (1981) facilitates the marshalling of the evidence needed to answer this question. Sen called attention to the fact that certain recent famines in underdeveloped nations occurred despite abundant harvests. These famines were caused not by natural disasters but by dramatic redistributions of "entitlements" to grain. The mechanism which promoted the redistribution of entitlements was a sharp rise in the price of grain relative to wages or other types of income received by the lower classes. In the "great Bengal famine" of 1943, for example, the exchange rate between wages and foodgrains declined by 86%, despite an "exceptionally high" supply of grain. In this case the rise in grain prices had nothing to do with the bountifulness of the harvest, but was driven by forces outside of the agricultural sector. The Bengal famine, Sen points out, was a "boom famine" caused by "powerful inflationary pressures" unleashed by a rapid expansion of public expenditures (pp. 66, 75).

The relevance of the entitlement approach to the interpretation of the economic demography of the early modern era does not depend on the source of the rise in grain prices that triggers the redistribution of entitlements. It is the similarity in the structural characteristics of traditional societies of the past and of low-income countries today that makes the entitlement approach pertinent (Flinn 1974; Post 1976; Appleby 1979a; Hufton 1983: Tilly 1983). At the root of these structural similarities is the highly unequal distribution of wealth and the overarching importance of land as a source of wealth. These twin characteristics lead directly to two other structural features. First, they cause the price elasticity of the total demand for grains to be quite low. Second, they drive a large wedge between the grain demand elasticities of the upper and the lower classes, with the elasticity of the lowest classes having a value that may be 10 or 20 times as large as the elasticity of the class of great land magnates.

It is these large class differences in demand elasticities (caused by social organization) rather than wide year-to-year swings in harvest yields (caused by variations in weather or other natural phenomena) that were the source of the periodic subsistence crises that afflicted late medieval and early modern England. Once the yield-to-seed ratio reached four and carryover inventories exceeded a month or two of the annual supply (which probably occurred in England before 1400), large weather-induced reductions in the normal national supply of foodgrains probably became exceedingly rare. The evidence at hand suggests that during the 331 years covered by the analysis of Wrigley et al. there were probably not more than 7 or 8 years (and there may have been as few as 3) during which the average food supply fell below its normal level by as much as 7%. One implication of the proposition that the national subsistence crises of the pre-industrial era were the

products of entitlement shifts (rather than natural disasters that cut deeply into the food supply) is that the impact of these crises on average national mortality rates was fairly limited.[35]

Equation (7) is a convenient starting point for the estimation of the relevant elasticities:

$$\bar{\epsilon}_i = (\theta_i - \beta_i)\psi_i - \epsilon_i \tag{7}$$

where

$\bar{\epsilon}_i$ = the income-compensated price elasticity of the demand for grain
ψ = the income elasticity of the demand for grain
ϵ_i = the income–fixed price elasticity of the demand for grain
β = the share of grain in total consumption expenditures
θ = the share of income arising from the ownership of grain
i = a subscript designating the ith class.

Equation (7) states that the income-compensated elasticity of demand for grains of a given class depends not only on ϵ_i (the income–fixed price elasticity, which is often referred to as the "substitution" elasticity) but the relative magnitude of θ_i and β_i. It follows from equation (7) that wealthy landlords would have a much more inelastic demand for grain (because the share of their income arising from ownership of grain-producing lands equaled or exceeded the share of their income that was spent on the consumption of grains—that is, because $\theta_i \geq \beta_i$) than landless laborers (for whom $\theta_i = 0$ and β_i is large).

Table 9.13 divides the English population at the middle of the Wrigley-Schofield-Lee period (ca. 1700) into four classes that correspond to the aristocracy and gentry, the yeomanry, artisans and shopkeepers, and common laborers (including the unemployed). Servants working in the households of the upper classes are included with these classes, since their masters provided the grains which they consumed. In other words, the population embraced by the landlords (class 1 in table 9.13) includes not only the landlords and their immediate families but all of their retainers, high and low. Table 9.13 also presents my estimates of share of the English population represented by each of the classes, the normal share of each class in the annual consumption of grain (ϕ_i), and of θ_i, β_i, ψ_i, and ϵ_i. The population shares are based on King's table (Laslett 1984).[36] The values of ϕ_i follow from the population shares and estimates of the per capita consumption of grains and calories in each class indicated by recent studies (including Drummond and Wilbraham 1939; Everitt 1967; Kerridge 1968; Appleby 1979b; Dyer 1983; Shammas 1983, 1984; Lindert 1985; Williams 1985). The values of ϕ_i shown in column 2 imply that landlords and yeomen consumed about 50% more grain per capita than the national average (much of it as ale and spirits), that shopkeepers and craftsmen consumed the national average, and

Table 9.13 **Estimates of the "Normal" Shares in Foodgrain Consumption and of the "Normal" Price Elasticities of the Demand for Foodgrains by Socioeconomic Class in England about 1700**

Class of Household Head	Share in Population (1)	Normal Share in Consumption of the Foodgrains (2) ϕ_i	Share of Grain in Total Consumption of a Class (3) β_i	Share of Grain in Income (4) θ_i	Income Elasticity (5) ψ_i	Income-Fixed Price Elasticity (6) ϵ_i	Income-Compensated Price Elasticity (7) $\bar{\epsilon}_i$
1. Landlords (including servants and retainers)	0.11	0.16	0.20	0.50	0.24	0.05	−0.02
2. Farmers and lesser landlords (including servants)	0.34	0.51	0.30	0.50	0.38	0.12	0.04
3. Shopkeepers, minor professionals, and craftsmen (including servants)	0.11	0.11	0.40	0.00	0.52	0.20	0.41
4. Laborers and the unemployed (not including servants covered in lines 1, 2, and 3)	0.44	0.22	0.70	0.00	0.96	0.50	1.17

Source: Fogel 1985.

that common laborers and paupers consumed about 50% of the national average. Allowing for waste and storage losses, these values of ϕ_i imply that the average caloric intake of the poor was at about the mean level of Afghanistan or Bangladesh today (World Bank 1984), while the landlords and yeomen were at the level of United States farmers about 1850 (Fogel and Engerman 1974). The values of θ, β_i, ψ_i, and ϵ_i are based on available evidence regarding the share of gross farm income originating in grains during the early modern era and cross-sectional studies of less developed countries today with income levels and agricultural sectors similar to those of early modern England (Fogel 1987).[37]

One important implication of table 9.13 is that although laborers were about 44% of the population, they accounted for only 22% of the normal consumption of foodgrains, and that landlords (who with their retainers and servants represented only 11% of the population) accounted for nearly as large a share of consumption. Another implication of table 9.13 is that both the magnitude and the direction of the effect of a rise in grain prices on elasticities was quite different for different classes (see col. 6 and 7). In the case of landlords the income–compensated price elasticity is positive, even though the income–fixed price elasticity is negative. This change in sign reflects the fact that landlords were not only consumers of grain but owners of large surpluses. In their case the rise in prices had two effects: the rise in grain prices increased their income as owners of surpluses, while it reduced their income as consumers. For landlords the producer's effect was so much stronger than the consumer's effect that rising grain prices would have led them to increase their consumption of grain. In the case of yeomen both effects were also present, but the income effect was not strong enough to have increased their grain consumption with rising prices; however, it did cut the income–compensated price elasticity to one-third of the income–fixed price elasticity. In the case of laborers only the consumption effect was present. Although the income–fixed price elasticity is already high, the income–compensated price elasticity is more than twice as high.

The values set forth in table 9.13 make it possible to estimate aggregate elasticity of the consumption demand for grains ($\bar{\epsilon}_c$), by making use of the relationship set forth in equation (8):

$$\bar{\epsilon}_c = \phi_1\bar{\epsilon}_1 + \phi_2\bar{\epsilon}_2 + \phi_3\bar{\epsilon}_3 + \phi_4\bar{\epsilon}_4. \tag{8}$$

Substituting the appropriate values of ϕ_i and $\bar{\epsilon}_i$ into equation (8) yields

$$\bar{\epsilon}_c = (0.16)(-0.02) + (0.51)(0.04) + (0.11)(0.41) + (0.22)(1.17) = 0.320. \tag{9}$$

Thus, the estimates of class elasticities in table 9.13 imply that the elasticity of the aggregate consumption demand was well below 0.5,

even though common laborers and paupers, who accounted for nearly half the population, had an elasticity in excess of one. However, as equation (8) indicates, it is shares in consumption rather than in population that determine the value of $\bar{\epsilon}_c$. If it were the population share that mattered, $\bar{\epsilon}_c$ would be nearly twice the indicated size and would fall between demand elasticities of artisans and laborers, who constituted the majority of the population. As it is, $\bar{\epsilon}_c$ falls below the demand elasticity of artisans because of the heavy weight given to the elasticities of landlords and yeomen who, although a minority of the population, accounted for two-thirds of consumption.

Although $\bar{\epsilon}_c$ is the price elasticity of the aggregate foodgrain demand, it is not the price elasticity of aggregate demand, which is given by equation (10):

$$\epsilon_t = \delta\epsilon_s + (1-\delta)\bar{\epsilon}_c, \tag{10}$$

where

ϵ_s = the price elasticity of demand for grains used as seed and feed
δ = the share of the total crop normally used as seed and feed.

Available evidence suggests that about 25% of grains were reserved for seed and feed and that the demand for this intermediate product was moderately inelastic (about 0.6) but not as inelastic as is often implicitly assumed.[38] Substituting these values into equation (9) results in

$$\epsilon_t = 0.25 \times 0.6 + 0.75 \times 0.32 = 0.39. \tag{11}$$

Equation (11) gives the "normal" value of ϵ_t—the value of ϵ_t when yields are close to the mean (within plus or minus one standard deviation). We have not yet considered the case in which the deviation from normality is large enough to trigger significant declines in exchange entitlements. Table 9.14 shows that such declines could be triggered with surprisingly small shortfalls in output. Even a shortfall in output as small as 8% triggers significant shifts in the shares of grain consumed by different classes. In the case of landlords, the rise in their share more than offsets the decline in output so that their per capita consumption rises slightly. In the case of laborers, however, the decline in their share reinforces the decline in output so that their per capita consumption is down by 23%. It is worth noting that although output declines by 8%, aggregate foodgrain consumption only declines by 6% because grain reserved for feed and seed declines by twice as much (13%) as foodgrain consumption. As a result the feed and seed share of the reduced crop declines from 25% to 22%. With a supply that is 15% of the normal level, the interclass distribution of per capita consumption is so greatly exacerbated that the per capita grain con-

Table 9.14 **Consequence of Shifting "Entitlement" Exchange Ratios on the Share of Each Class in the Reduced Crop and on the per Capita Consumption of Each Class**

Class of Household Head	Normal Share of Each Class in Foodgrain Crop $Q_d = Q_s = 1$ $p = 1$ $\epsilon_t = 0.39$ (1)	Case Where $Q_s = 0.92$		Case Where $Q_s = 0.85$	
		Share of Each Class in Reduced Output of Foodgrain at Market-Clearing Price (2)	Percentage Decline of Each Class from Normal per Capita Consumption of Foodgrains (minus signifies rise in consumption) (3)	Share of Each Class in Reduced Output of Foodgrain at Market-Clearing Price (4)	Percentage Decline of Each Class from Normal per Capita Consumption of Foodgrains (minus signifies rise in consumption) (5)
1. Landlords (including servants and retainers)	0.16	0.172	−0.5	0.186	−1.0
2. Farmers and lesser landlords (including servants)	0.51	0.540	0.9	0.570	2.0
3. Shopkeepers, minor professionals, and craftsmen (including servants)	0.11	0.107	8.8	0.100	18.6
4. Laborers and the unemployed (not including servants listed under 1, 2, and 3)	0.22	0.181	23.2	0.145	44.4

Source: Fogel 1985.

sumption of laborers and paupers falls to less than 60% of its normal level.

The sharp decline in consumption of the laboring class (when Q_s = 0.85) is due to the combination of its high elasticity of demand ($\bar{\epsilon}_4$ = 1.17) and the sharp rise in price (P goes to 1.65). It should be noted that more than a fifth of the indicated price rise is due not directly to a decline in Q_s from 1 to 0.85, but to the rise in the value of ϵ_t as the price increases. If ϵ_t had remained constant, the decline in Q_s would have led to a 52% increase in prices instead of a 65% increase. In other words, one of the effects of the shifting distribution of entitlement is to reduce both $\bar{\epsilon}_c$ and ϵ_t (to 0.23 and 0.30, respectively). It follows that an initial rise in prices tends to feed on itself, even in the absence of a speculative demand or irrational hoarding, by increasing the share of grain entitlements held by classes with a highly inelastic demand.

The estimates in table 9.14, together with the distribution of deviations in grain prices from their trend, make it possible to assess the extent of harvest failures in years of "exceptionally" high prices.[39] Perhaps the most important feature of the price distribution is that there are only 23 out of 198 observations (0.1162) between 1540 and 1737 in which the price index deviates positively from the trend by as much as 25%. Using 0.37 as an estimate of ϵ_t (the value implied by table 9.14 for a 25% rise in prices), this fact implies that the aggregate quantity of grains fell below the trend (or mean level) of quantity by as much as 0.0792 in only 11.62% of the years. With the deviations in quantity ("yields") normally distributed, [40] the preceding points imply equation (12):

$$1.20\sigma_g = 0.0792\bar{X}_g, \tag{12}$$

where

σ_g = the standard deviation (S.D.) of deviations from the "normal" level (or trend) of quantity

$\bar{X}_g$ = the "normal" level (trend level) of quantity, which is set equal to one.

Solving equation (12) for σ_g results in a value of 0.066.[41]

Thus, the standard deviation of the distribution of grain "yields" (deviations in the quantity of all grains) is far smaller than the standard deviation of the distribution of deviations in wheat prices, which over the same 198 years was 0.251.[42] This is an important finding for the assessment of investigations that have used the price of wheat as a proxy for the physical quantity of all grains. The large difference between the two standard deviations (of grain "yields" and of deviations in wheat prices) implies that the elasticity of grain "yields" with respect

to wheat prices was substantially below that of ϵ_f. This result is immediately apparent from equation (13):

$$\frac{\dot{Q}_g}{\dot{P}_w} = \epsilon_{gw} = \frac{\sigma_g}{\sigma_w} r_{gw}, \tag{13}$$

where

$\dot{Q}_g$ = percentage deviations in the quantity of grain
$\dot{P}_w$ = percentage deviations in the price of wheat
ϵ_{gw} = the elasticity of the quantity of grain with respect to the price of wheat
r_{gw} = the coefficient of correlation between deviations in grain quantities and deviations in wheat prices
σ_g = the S.D. of deviations in grain quantities
σ_w = the S.D. deviations in wheat prices.

Hence

$$\epsilon_{gw} = 0.263 r_{gw}. \tag{14}$$

Although it is often assumed that, except for the very rich, grains were virtually the equivalent of the food supply in early modern England, recent studies have revealed that meat, fish, game, dairy products, vegetables, fruits, and nuts formed a significant part of the diet of other classes, even of common laborers and paupers (Fussell 1949; Everitt 1967; Kerridge 1968; Richardson 1976; Dyer 1983; Oddy 1983; Shammas 1983, 1984; Skipp 1978; Drummond and Wilbraham 1958; cf. Webster 1845). The difficulty with the stylized approach to food supply is that it implicitly assumes that σ_g (the S.D. of deviations around the trend in the quantity of grain) is identical with σ_f (the S.D. of deviations around the trend in the total food supply). As it turns out this assumption is unwarranted. In the case of the all-agricultural-products price series, only five out of 198 observations (0.0253) between 1540 and 1737 deviate positively from the trend by as much as 25%. Hence, instead of equation (12) we have equation (15):

$$1.95\sigma_f = 0.0792\bar{X}_f, \tag{15}$$

which yields a value for σ_f of 0.041.[43] Now ϵ_{fw} (the elasticity of the quantity of food with respect to the price of wheat) follows from equation (16):

$$\epsilon_{fw} = \frac{0.041}{0.251} r_{fw} = 0.163 r_{fw}, \tag{16}$$

where r_{fw} is the coefficient of correlation between deviations in the quantity of food and deviations in wheat prices.

Equations (14) and (16) reveal the pitfall in the interpretation of studies that used wheat prices as a proxy for shortfalls in the supply of grain and of food. It was assumed that because wheat prices were highly correlated with other food prices, that large positive deviations of wheat prices implied not only large declines in the supply of wheat but in the supply of grains as a whole and in the total supply of food. Equation (14) shows that even if the correlation between grain "yields" and wheat prices were perfect, what has been termed an "exceptional" deviation in wheat prices (20% above trend) would only imply about a 5% decline in the total supply of grains and (taking account of the shift away from feed and seed) only about a 4% decline in the supply of foodgrains. Consequently, far from representing serious nutritional deprivation, most "exceptional" deviations in wheat prices fell within the range of "normal" consumption of grains (although not necessarily of wheat). Even among common laborers, who would have suffered from the adverse effect of a shift in entitlements, a positive deviation in wheat prices of 20% would imply a decline in per capita food consumption of about 9%—not a catastrophe, but not trivial either for a class suffering from the chronic malnutrition that accompanied a diet that probably averaged below 2,000 calories per capita. For the other three classes, the implied decline in per capita consumption is negligible.

The estimate of σ_g and σ_f developed from equations (12) and (15) makes it possible to assess the extent to which the English population suffered from generalized harvest failures during 1541–1871. A decline in grain "yields" of as much as 10% ($1.52\sigma_g$) was a relatively rare event, occurring less than 1 year in 15. It is unlikely that England suffered more than one 10% shortfall in the overall food supply ($2.44\sigma_f$) after 1540. The estimated value of ϵ_{fw} (assuming $r_{fw} = 1$) implies that even in 1556 when the positive deviation in wheat prices was at its highest post-1540 level (98%), the decline in the overall food supply was about 10.5%. All but seven of the other poor harvests imply grain shortfalls of less than 10%, foodgrain shortfalls of less than 8%, and overall food shortfalls of less than 7%.[44]

The information in column 3 of table 9.14 provides a basis for estimating the effect of post-1540 harvest failures on the mortality rate. That column shows that with total grain shortfalls of 8%, only the two lowest classes would have had nonnegligible reductions in their normal supply of grain. In the case of the artisan class, much of the 8.8% reduction in the apparent consumption of grains could have been offset by shifting from inefficient to efficient forms of the extraction of calories (from beer and ale, e.g., to gruels and porridges) and by reducing storage losses and waste (Fussell 1949; Drummond and Wilbraham 1958; Mathias 1959; Stern 1964; Walford 1970; Walter 1976; Appleby

1978). Consequently, the demographic effect of harvest failures would have been confined largely, if not exclusively, to the class of common laborers and paupers, many of whom were unrelieved by public or private charity (Lindert 1980; Lindert and Williamson 1983b). In this case, the gap between apparent and effective consumption was probably quite small because the lower classes could not indulge in inefficient forms of grain processing and consumption to the same extent as the other classes. Moreover, lacking the wherewithal to hold inventories of food, they were also spared the large inventory losses of the period.

On the other hand, part of the decline in the grain consumption of laborers would have been offset by an increased consumption of other foods, some normally considered inferior to grains, including nuts, rootcrops, garden vegetables, milk, and milk products (Fussell 1949; Drummond and Wilbraham 1958; Stern 1964). Equations (14) and (16) together imply that about 40% of the decline in grain consumption would have been offset by the increased consumption of other foods. Consequently, in order for the food consumption of laborers to have declined by 23%, the shortfall in the grain supply would have had to have been about 13% ($0.08 \div 0.615 = 0.130$), an event that would have occurred just one year in every 41. If we continue to assume an elasticity of the death rate with respect to nutritional status of -1.4 (although that figure may be too high for a single year's deprivation), then a shortfall in grains yields sufficient to reduce the food consumption of common laborers by 23% would have raised their mortality rate by 32%—more than enough to constitute a grave social crisis. Nevertheless, the effect of such a rise on the crude death rate (cdr) of the entire population (which is the statistic that has entered into studies which attempt to relate wheat prices to mortality rates) would be much smaller. During an age in which the trend value of cdr was about 28 per thousand, and assuming that the normal cdr for laborers was 10% above the average, the national cdr in a famine year would have risen by about 4.4 per thousand ($0.44 \times 1.4 \times 0.232 \times 0.028 \times 1.1 = 0.0044$), to a level which is about 16% more than the average cdr.

Thus, even if the elasticity of mortality rates with respect to nutritional status was as large in early modern England as the Trinidad data suggest, one would hardly expect to discover that the elasticity of wheat prices with respect to famine-induced deaths (as opposed to deaths due to regular and persistent malnutrition) was large since only one-fifth of the years identified as famine years using wheat prices would represent years in which there was actually a substantial increase in the national cdr due to a shortfall in food. The basic point, however, is not that regressions have been misspecified and the results misinterpreted. It is rather that after approximately 1400 or 1500, famines in England

were rare events, affecting a minority of the population, and accounting for a minuscule proportion of accumulated deaths. Over the entire 331 years covered by the Wrigley-Schofield-Lee analysis it is unlikely that *famine* deaths accounted for as much as 0.4% of the accumulated total deaths ($0.16 \times 0.024 = 0.0038$).[45] Given the exceedingly small proportion of deaths due to famines, the many factors (including nonfamine malnutrition) that affected annual death rates and the lack of controls for most of the relevant factors, the highly aggregated level of the time-series analysis, and the use of wheat prices as a proxy for food shortfalls, it is impressive that Lee was able to pick up any mortality effect at all, let alone one which appears to be quite close to the mark.[46]

The Wrigley-Schofield-Lee results seemed paradoxical only because it was widely assumed that the large swings in wheat prices represented correspondingly large swings in the aggregate supply of food.[47] When it is recognized that food shortfalls of 7% or more were quite rare, it becomes evident that their results neither contradict nor confirm the nutritional hypothesis. They do demonstrate that *famine* mortality was a minor factor in explaining both cumulated deaths and secular trends in the death rate during 1541–1871, but this finding does not eliminate *chronic* malnutrition as a significant component of pre-industrial mortality rates.[48] The "normal" distribution of shares in grain entitlements shown in table 9.13 and what is known about the course of agricultural production suggests that until the end of the Napoleonic Wars, English laborers were about as badly fed as the lower classes in such countries as India, Pakistan, and Bangladesh shortly after World War II. This conclusion is consistent with the currently available data on the heights of English laborers between 1760 and 1810. The impact of chronic malnutrition on English mortality rates during the preindustrial era remains an open issue.

9.4.4 Some Implications for the Standard of Living Controversies in Europe and America

The long and sustained upward movements in physical growth schedules in Europe and the United States that began late in the nineteenth century and continued through most of the twentieth century were a sharp break with the past. Before the last quarter of the nineteenth century, only the United States had experienced a long period of relatively rapid upward movement in growth schedules, with most of it occurring before 1710. Between cohorts born in 1710 and in 1780 final heights of native-born white American males increased at a rate of only 0.25 centimeters per decade.[49] During the next century the final heights of Americans oscillated in a narrow band or declined fairly sharply, losing about 3 centimeters in just two decades beginning with cohorts born about 1830. Rapid upward shifts in growth schedules probably

did not resume until the last decade of the nineteenth century. The new period of increase in final heights which lasted for about 60 years was more rapid than the increase experienced during the late seventeenth and early eighteenth centuries (Sokoloff and Villaflor 1982; Fogel et al. 1983; Margo and Steckel 1983).

The principal upward shift in English growth schedules before 1900 came later and was shorter than that experienced in the United States. The mean final height of English working-class males born about 1760 was about 64.5 inches (which was about 9 centimeters below that of United States whites) and remained more or less at that level for the next half century. The succession of cohorts born between approximately 1810 and 1840 appears to have experienced a fairly rapid upward shift in growth schedules, so that the 1840 cohort was about 3 centimeters taller at maturity than the 1810 cohort. Thereafter, the upward shift in growth schedules slowed down so that cohorts born half a century later were only about a centimeter taller at maturity than their 1840 counterparts (Floud and Wachter 1982, 1983; Sokoloff and Villaflor 1982; Floud 1983a, 1983b).

There were also relatively constant growth curves for France between approximately 1820 and 1900, and for Belgium between about 1830 and 1900. In both countries heights at age 20 remained below 65.5 inches down through the end of the nineteenth century (Floud 1983b). In the Netherlands and the Scandinavian countries the laboring classes appear to have experienced improved living conditions sometime during the third quarter of the nineteenth century. In the case of the Netherlands, for example, mean adult heights between 1865 and 1905 increased at about 1 centimeter per decade, which is about five times as large as the British rate of increase during the same period. Even so, the Dutch rate of increase during the late nineteenth century was only half as great as the rate of increase during the half-century following World War I (Van Wieringen 1978).[50]

Although the standard of living of the laboring classes in the United States was quite high early in the nineteenth century by European standards, it appears that the difference narrowed considerably over the course of the nineteenth century, partly because United States height declined for about half a century and partly because the heights of Europeans increased somewhat. At the end of the nineteenth century when the United States entered a new phase of rapid upward shift in growth curves, living standards for United States workers, as measured by final heights, were quite similar to those prevailing in northwestern Europe, but they were still significantly better than those of southern Europe. As late as 1900 the mean height of adult Italian males was below 65 inches (Terrenato and Ulizzi 1983).

In recent decades economists and economic historians have tended to assume that if the "real wage" (an index of nominal wages divided

by an index of prices) was rising, then the standard of living of workers was rising. Beginning with the late nineteenth century, but especially after 1930 when large investments were made in the gathering of wage and price data, older measures of the standard of living, such as weight, housing conditions, and mortality rates began to be abandoned in favor of the newer and presumably more comprehensive index. Criticisms have been made of the quality of the data from which long-term series of real wages were constructed (Von Tunzelmann 1979; Thirsk 1983), and far-reaching questions have been raised about what real wages actually measured, even when the indexes were ideally constructed (Taylor 1975). Nevertheless, the tendency has been to employ real wages not just as *a* measure (or even as the principal measure) of the standard of living but sometimes to convert it into a synonym for the standard of living.

The point is not that real wage measures should be discarded but that the interpretation that has been placed on them needs to be reconsidered. We may be able to obtain a deeper understanding of the changing standard of living of workers, develop a subtler appreciation of the manifold dimensions of the phenomenon, if the information embodied in both real wage indexes and measures of per capita income is reviewed in the light of the information contained in anthropometric measures, mortality and morbidity rates, and other measures of living conditions that are now becoming available.

What, for example, are we to make of a situation in which real wages were rising rapidly, as apparently occurred in England during the last half of the nineteenth century (Mitchell and Deane 1962; Matthews et al. 1982) while working-class heights remained at relatively low levels, showing little increase over half a century? How should we characterize conditions of workers in the United States between 1820 and 1860 if real wages were generally constant or rising, sometimes quite rapidly (Williamson 1976; David and Solar 1977), but heights and life expectation were decreasing? During an era in which from 50% to 75% of the income of workers was spent on food, is it plausible that the overall standard of living of workers was improving if their nutritional status and life expectations were declining? These are not questions that can easily be resolved, and I will not attempt to do so here. Rather, I want briefly to sketch some of the new issues about the course of the standard of living that are suggested by the anthropometric and demographic data. When real wages, per capita income, and other measures all move in the same direction there is little need to probe into their exact meaning. The interesting issues arise when the measures diverge, and it is on some of these issues that I wish to focus.

The evidence so far developed on height and mortality rates suggests that improvements in the living conditions of workers during the nineteenth century may have been more sporadic and uneven (both in time

and among subgroups of workers) than is suggested by indexes of real wages or the movements in per capita income. In England the period of rapid improvement in the nutritional status of workers seems to have been confined largely to the 3 decades following the end of the Napoleonic wars. In France there was little change from the end of the Napoleonic wars until the beginning of the twentieth century. In the United States, the nutritional status of the laboring classes, which was initially quite high by European standards, appears to have deteriorated during the middle decades of the nineteenth century.

Although the substantial declines in both heights and life expectation shown by figue 9.1 are too new and provisional to be accepted without substantial additional evidence, let us suppose for the moment that further investigation supports the provisional findings. Will these series then contradict prevailing estimates of fairly rapid increases in conventional measures of per capita income? Since Williamson and Lindert (1980) have provided evidence that the inequality in the income distribution increased during this period, the question does not have an obvious answer. It is now necessary to probe more deeply into the magnitude, nature, sources, and locus of both gains in income and the increase in inequality. Rising inequality provides one way, although not the only way, of resolving the apparent anomaly between current estimates of rapidly rising per capita income and of declining heights during the middle quarters of the nineteenth century. Steckel's (1983) analysis of the relationship between mean final height, per capita income, and the Gini coefficient (a measure of the inequality of income or wealth distributions) reveals that with respect to final heights an increase of 100% in per capita income would just offset an increase in the Gini ratio of 0.066. It follows that current estimates of the increase in per capita income and the estimated decline in mean heights of 1.5 inches together suggest that the Gini ratio increased by about 0.17, going from perhaps 0.30 in c.1830 (the current figure for Australia) to about 0.47, which is a plausible estimate of the Gini ratio for the United States near the turn of the twentieth century (Lebergott 1976; Sawyer 1976; Williamson and Lindert 1980).[51] Thus, the decline in final heights of native-born United States white males may add to the evidence recently developed by others which indicates that the middle quarters of the nineteenth century witnessed significant increases in the inequality of the American income distribution (Pessen 1973; Williamson and Lindert 1980).

It may seem odd to those unfamiliar with this body of evidence that changes in height should be used as a measure of changes in the inequality of the distributions of income and wealth. However, as Floud and Wachter (1982) have recently pointed out, there was a time when height was the most compelling and the most widely used index of

inequality. Moreover, because of the abundance of height data and their wide coverage of geographic regions and socioeconomic groups, it is possible to probe more deeply into vexing issues regarding variations in the conditions of the population of particular regions and occupations than has so far been possible with wage data which are skimpy in their coverage of particular categories. Two examples suggest the possibilities that now appear to be emerging.

The first is drawn from the work on British sources. Data recently collected by Floud on the British upper classes (1984) when combined with his data on the lower classes make it possible to estimate how much of the improvement in the average nutritional status in Great Britain over the past century and a half has been due to a closing of the gap between the upper and lower classes and how much was due to an upward shift in attainable average height. By "attainable" I mean not genetically attainable but, within genetic constraints, attainable under the most favorable prevailing socioeconomic circumstances. Floud's research indicates that about three-quarters of the increase in the mean final height of British males since about 1820 was due to the decrease in class differentials in height and the balance to an upward shift in the mean final heights of the upper class (which may be taken as a measure of the attainable mean height at any point in time).[52] In this connection it is worth noting that Sweden and Norway, which have two of the lowest after-tax Gini ratios, are the only countries in which height differentials by socioeconomic class have disappeared (Lindgren 1976; Sawyer 1976; Brundtland et al. 1980). The means of adult height in these nations now exceed those of high-Gini-ratio nations, such as the United States and Great Britain, by several centimeters (United States Bureau of the Census 1983).

The second example pertains to the effect of urbanization on both the level and distribution of real income. Previous research has revealed an association between the increase in the inequality of the American distributions of income and wealth during the nineteenth century and urbanization (Gallman 1969; Soltow 1971). Analysis of the height data in the samples drawn from the Union and the regular army rolls supports this finding but calls attention to the complexity and often roundabout nature of the influence of urbanization. Figure 9.6 shows the beginning of the long downward trend in the aggregate series on native-born whites (cf. fig. 9.1). It also shows that when this series is disaggregated into occupational and residential groups, the series for farmers exhibits a rising trend until around 1830 and then declines for the next two cohorts. The trend for urban laborers is basically flat at the beginning, rises slightly between 1825 and 1830, and then declines. These curves indicate that about 85% of the initial decline in the aggregate series was due to a decline in the mean heights of farmers and other rural

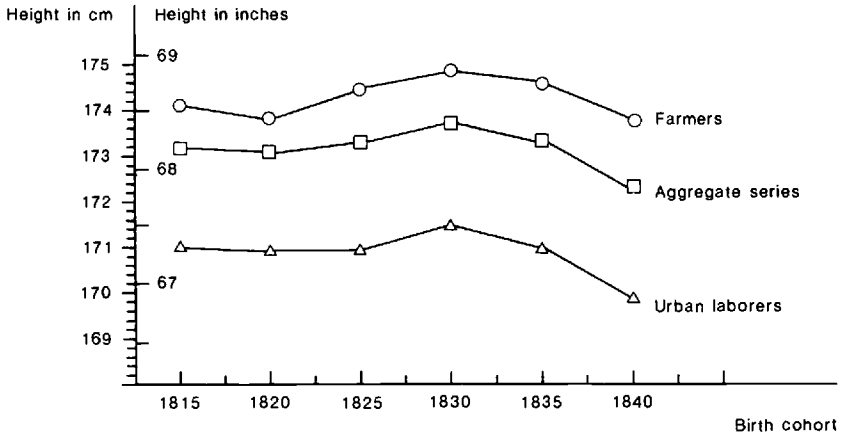

Fig. 9.6 A comparison of the aggregate time profile of final height for native-born white males with those for native-born white farmers and urban laborers. *Source:* unpublished data.

residents. The balance of the decline in the aggregate series was due to the increased proportions of the population experiencing the poor nutritional health conditions of the cities as well as to declining heights among urban residents (cf. Margo and Steckel 1983).

The deterioration in the mean final height of farmers apparent in the last two cohorts of figure 9.6 continued in subsequent decades. Native-born farmers who were born about 1860 were about 1.5 inches shorter than those who were born 3 decades earlier. Over the same period the final heights of urban laborers declined by about 0.8 inches. Thus, although deteriorating conditions in the cities and the shift of population from the countryside to the cities played a role, they explain only about one-fifth of the decline in the aggregate series shown in figure 9.1 for cohorts born between 1830 and 1860. About four-fifths of the decline was due to a deterioration of conditions affecting growth in the rural areas.

Current research is aimed at explaining this surprising decline in rural heights. One possibility is that an increasing proportion of the native-born rural males were children of foreign-born parents. It is likely that foreign-born mothers were relatively malnourished during their own developmental years and that foreign-born parents generally had lower incomes than native-born parents. Both factors would have made the children of foreign-born parents shorter than the children of native-born parents. Support for this hypothesis is found in a subsample of the Union Army recruits which has been linked to the manuscript schedules of the 1860 census. This subsample reveals that in the rural

areas native-born males of foreign parents were 0.4 inches shorter in final height than native-born males of native-born parents. The effect of parental ethnicity was even greater in the cities, with children of foreign-born parents averaging 1.2 inches less in final heights than children of native-born parents. It thus appears that the low incomes of foreign-born parents and the poor nutritional status and health of foreign-born mothers had an effect on children both in the cities and in the countryside, but that effect was greater in the cities than in the countryside. It is plausible that as much as half of the urban-rural differential in native-born heights was associated with parental ethnicity.[53]

The decline of heights in the rural areas is particularly puzzling. The ethnic effect could only have accounted for a small share of the rural decline, and the available evidence strongly suggests that the per capita production of food, especially in the Midwest, increased between 1840 and 1860. There is, of course, the possibility that rural food consumption declined, despite the increase in output. Steven B. Webb has recently suggested that improved transportation links between farms and the cities might have had two negative health effects in the rural areas:[54] "First, while railroads were lowering food prices in the cities and improving nutrition there, they may have raised farm gate prices and lowered nutrition there. Giving farmers the opportunity to trade corn for calico may raise their utility, or at least the parents' utility, but may also reduce caloric intake. Second, increased contact with the urban disease environment may have increased the spread of communicable diseases in the rural sector."

Both of these possibilities certainly ought to be pursued, and they may turn out to be right. Yet the evidence on the growth of per capita income in the Midwest between 1830 and 1860 is so compelling, and midwestern farmers as a class seem to have prospered so much during this era, that it is not easy to accept the hypothesis that their food consumption declined, let alone that it declined by enough to explain a decrease in height of more than 1.5 inches (Bidwell and Falconer 1925; Berry 1943; G. R. Taylor 1951; Easterlin 1975).[55]

Of course, not all rural residents were farmers, and not all farmers shared equally in the agrarian prosperity. As much as 40% of the rural labor force of the North was employed in manufacturing, construction, and trade (Yang 1984; Moen 1985). This was an age in which many sectors of manufacturing, including iron production, milling, textiles, and many handicrafts were located largely in rural areas, partly because access to water power and raw materials were significant considerations (Temin 1966). There is a good deal of evidence that the nonagricultural sector of rural labor force, particularly native-born craftsmen, may have suffered from the rise in the cost of living to which Webb refers (Fogel 1986). Moreover, the competition from foreign-born labor and a rise in

unemployment may have led a fair number of native-born craftsmen to shift into agriculture, either as laborers or at the low end of the distribution of farm owners and operators. Yet, even if half of these workers suffered income declines large enough to induce a 1.5-inch decline in the final heights of their children, the deprivation of this class would explain only about a fifth of the rural height decline.[56]

Perhaps Webb's second point holds the key. Recent studies have demonstrated a strong link between immigration rates and urban mortality rates both before and after the Civil War (Higgs 1979; Meckel 1985). Public health studies of the antebellum era demonstrated not only that mortality rates were much higher in the immigrant wards than in the wards in which the native-born were preponderant, but that epidemics often began first in the foreign-born wards and then spread outward, not only to other wards in the cities but to the rural areas as well.

The cholera epidemic of 1848–50 is the most dramatic case in point. This epidemic was brought to American shores in December of 1848 by two ships carrying German immigrants, one bound for New York, the other for New Orleans. Although New York-bound passengers who were sick with cholera when the ship arrived were kept in quarantine, others were allowed to enter the city. Within a few days cholera broke out in the immigrant districts of New York; then it spread to the predominantly native-born, lower-class districts nearby; and eventually to upper-class districts. In the case of the ship bound for New Orleans, public health officials were able not only to tie the spread of disease to New Orleans with the disembarkation of the immigrants there, but to follow the movement of cholera up the Mississippi and its tributaries. As immigrants from the infected ship boarded river steamers, cholera broke out aboard these ships and then in the cities at which the steamers called, including Memphis, Nashville, Louisville, Cincinnati, Wheeling (West Virginia), Pittsburgh, and Saint Louis (United States Surgeon-General's Office 1875). Soon after it reached these cities, cholera broke out in the surrounding countryside.

Cholera was the most dramatic disease of the antebellum era because it struck the nation suddenly, spread quickly, had a high case fatality rate, and its victims often succumbed within 24 hours after they became sick (Rosenberg 1962; *Encyclopaedia Britannica* 1961, 5: 615–18). But such diseases as malaria, typhoid, typhus, tuberculosis, smallpox, yellow fever, and dysentery took far more lives than cholera between 1800 and 1860. So severe was the increase in epidemic diseases during these decades that one historian of public health characterized it as a "period of great epidemics" (Smillie 1955). It seems likely, therefore, that increased exposure to disease was a major factor contributing to the decline in both the height and life expectation series shown in figure

9.1. The wide variations in the prevalence of particular diseases, by localities and over time, opens up the possibility of being able to measure the effect of variations in exposure to diseases on the variation in mortality rates with controls for height and a variety of socioeconomic variables.[57]

If cholera and other diseases that afflicted the United States during the nineteenth century were acts of God, unrelated to the functioning of the economic system, they would pose no special problem for the resolution of the standard-of-living controversy. However, economic growth, the spread of disease, and the concomitant increase in morbidity and mortality rates were intricately intertwined. Not only was internal migration responsible for as much as 50% of the increase in measured per capita income during the antebellum era (Fogel and Engerman 1971; Gallman 1972; Easterlin 1975; Fogel 1986), it was also a principal factor in the spread of cholera, typhoid, typhus, malaria, dysentery, and other major killer diseases of the era (Boyd 1941; Ackerknecht 1945, 1952; Smillie 1955). Increasing population density, another concomitant of economic growth, also increased the prevalence of various diseases, raising the level of malaria, enteric diseases, and diseases of the respiratory system (Ackerknecht 1945; Smillie 1955; May 1958; Kunitz 1983; New York State Board of Health 1867).

The increase in mortality between 1790 and 1860, therefore, calls for a downward adjustment in the measured growth of per capita income. Such an adjustment is necessary even if wage rates in high disease localities fully reflected the bribe which workers demanded for the increased risks of living in these areas, since national income accounting procedures treat the bribe as an increase in national income when it is merely a cost of production. Jeffrey Williamson's recent application of the bribery principle, using differences in wage rates between regions of high and low mortality to measure the disutility of English industrialization (Williamson 1981a, 1981b, 1982), represents an important advance in the assessment of both the short- and long-run costs and benefits of economic growth during the nineteenth century. The debate set off by his estimates (Pollard 1981; Floud 1984) involves such issues as whether workers had enough information to properly assess differences in risks, whether the measures of mortality used by Williamson were precise enough to gauge the differential risks that workers actually suffered in particular occupations and localities, and whether the various labor markets were all in equilibrium (or all out of equilibrium by the same degree). The resolution of these issues will no doubt add greatly to our knowledge about the costs and benefits of industrialization to the workers who experienced it.

There is an alternative approach to the computation of the mortality correction which, while not as comprehensive as the correction implied

by the bribery principle (Williamson 1984), is easier to measure. Equation (18) is derived from the theory of human capital:

$$w_n = (i + \delta_n)V_n \tag{18}$$

where

w_n = the wage rate (rental rate) at age n of a worker
i = the market rate of return on capital
δ_n = the annual rate of depreciation in the stock of human capital at age n (the probability of dying at age n)
V_n = the cost of producing a new worker aged n (the long-run equilibrium price of such a worker if he could be sold as a slave).

Differentiating equation (18) totally yields

$$\dot{w}_n = \phi\dot{i} + (1 - \phi)\,\dot{\delta}_n + \dot{V}_n, \tag{19}$$

where
$\phi = [i/(i + \delta_n)]$
$\dot{}$ = an asterisk over a variable indicates the rate of change in that variable.

Equations (18) and (19) indicate that increases in mortality rates will lead to spurious increases both in "real" wages (wages adjusted only for the price level) and in "real" per capita income. That is because conventional measures of "real" wages and per capita income fail to distinguish between rises in wages that are due to, say, technological change and those that are due to a more rapid consumption of human capital, treating both as if they represented net additions to human welfare.[58] Equations (18) and (19) indicate that increased mortality rates raise wages not only because they increase δ (the probability that someone in the labor force will die), but also because they increase V (the cost of producing a new entrant into the labor force). The higher the mortality rate, the greater the number of live births (and associated costs) needed to produce a new entrant into the labor force.[59] There is, of course, a corresponding increase in cost due to extra expenditures on nonsurvivors at all the other ages between birth and entry into the labor force.

The estimates of $\dot{\delta}$ and $\dot{V}$, which can be derived from the decline in life expectation shown in figure 9.1 and appendix A, indicate that rising mortality may have accounted for about two-fifths of the average annual increase in the conventional measure of "real" per capita income over the 70 years between 1790 and 1860.[60] In principle, this correction is a lower bound on the correction that one would obtain from the implementation of the bribery principle, since no account was taken of the psychic cost involved in the loss of loved ones, and since it was

implicitly assumed that workers were risk neutral with respect to their own fate.

The preceding estimate is merely meant to illustrate the new possibilities that are arising for refining our measures and conceptions of the changes in standards of living during the various stages of the industrialization process. The data on stature should provide additional information, beyond that conveyed by the mortality series, because they pertain to survivors, to individuals who lived to maturity (Floud 1984). Although the calculation based on equation (18) suggested the magnitude of the correction needed to net out the effect of rises in mortality rates on conventional measures of per capita income, it did not provide adjustments for the consequences of increased morbidity rates experienced by those who survived exposure to virulent diseases. Such corrections are needed to take account of medical expenditures and a variety of investments which merely offset the deterioration in the environment, and of diseases which degraded the quality of life and reduced the productivity of the labor of survivors. The data on stature promise to provide such adjustments, although we will need to learn more than we now know about how to separate out the effect of morbidity from other influences on stature before we can implement such an adjustment.

One issue which I have not yet probed in this discussion but which now leaps to the fore is the transfer of income and wealth between immigrants and native-born workers. Until now discussions of real economic growth by economic historians have tended to slight this issue, although it has been of great concern to many social, political, and labor historians (Hoagland 1913; Commons et al. 1918, I; Ernst 1949; Benson 1969; Handlin 1979; Pessen 1978; Hannon 1984). Whatever the long-run benefits of unrestricted immigration to native workers, it appears that in the short run it created severe hardships for them, not only by increasing the competition for jobs but also by increasing their exposure to disease and by reducing their life expectancy. The extent of the losses to native workers has been shrouded by the tendency to average over the experience of native and foreign-born workers. The available evidence suggests that the conditions of immigrants improved fairly rapidly after they arrived in the United States, not only because the disease environment was less virulent in America than in their native lands, but also because their wages rose fairly rapidly with time in the United States (Chiswick 1978, 1979; Lebergott 1984; Kearl and Pope 1986). Native workers, on the other hand, experienced declines in real income during the periods of most rapid antebellum immigration, even though the average level of their conventional income and their levels of health and longevity were higher than those of immigrants. This, then, is a case in which averaging over subgroups

gives quite misleading impressions of the fate of the different groups. Even if the average real wages of all United States workers rose between 1840 and 1860, as seems likely, such a highly aggregated index tends to underestimate the substantial gains of foreign-born workers and obscures the declines in the real income of native-born workers in the nonagricultural sectors during the intervening years (Fogel 1986).

9.5 Conclusion

The decline in mortality rates since 1700 is one of the greatest events of human history.[61] I was inclined to say "one of the greatest achievements of humankind," but the fact remains that we still do not know how much of that achievement was due to causes beyond human control. The paper published by McKeown and Brown in 1955 marked a turning point in the effort to provide a warranted explanation of the decline in mortality. Bridging the worlds of social scientists and of medical specialists, they brought into the discussion most of the range of issues that have been under debate for the past 3 decades. That debate not only defined the issues more clearly than previously, but also revealed that the critical differences were quantitative rather than qualitative. Nearly all the specialists agree on the range of factors that were responsible for the decline in mortality, but they have had quite different views about the relative importance of each of the factors.

The unresolved issue, therefore, is not really whether a particular factor was involved in the decline, but how much each of the various factors contributed to the decline. Resolution of the issue is essentially an accounting exercise of a particularly complicated nature, which involves measuring not only the direct effect of particular factors but also their indirect effects and their interactions with other factors. Our preliminary investigations indicate that the construction of data sets rich enough to permit such a complex accounting is critical to the successful outcome of the exercise. What is needed is a data set that can cope with the changes in the cause-of-death structure which, as Preston (1976) indicated, has varied significantly over time and place. To identify the locus of influences of each of the principal factors that contributed to the decline we need not only disease-specific but age-specific and generation-specific information, because the influence of both risk-increasing and risk-averting factors appears to vary markedly both over lifetimes and over generations.

The findings on the extent and the locus of the nutritional contribution presented in this paper are preliminary in two respects. First, we anticipate that more complete data will lead to revisions in the estimates we have presented. Second, nutritional status is only the first of numerous factors which contributed to the mortality decline in America

since 1700 that we hope to measure. Our preliminary results indicate that the contribution of improvements in nutritional status was neither inconsequential nor overwhelming; although it made a substantial contribution, the factors which contributed to the majority of the decline are still unmeasured. Moreover, although our preliminary estimates indicate that improvements in nutritional status may have accounted for about four-tenths of the mortality decline, this contribution was confined largely to the reduction in infant deaths, particularly to late fetal and neonatal deaths and to deaths during weaning. The concentration of the impact of improved nutrition in these age categories raises the possibility that increases in diarrhea and other diseases which diverted ingested nutrients from growth both before and after birth, rather than a decline in food intake, was the main cause of the decline in nutritional status and the rise in mortality during the middle decades of the nineteenth century.

Several important issues have been obfuscated by the confusion between diet (the gross intake of nutrients) and nutritional status (the balance between nutrient intake and the claims on that intake). The blurring of these concepts has diverted attention from the ingestion of harmful substances, which not only are devoid of nutritional value but prevent the body from assimilating nutrients at critical ages, especially in utero and in early childhood. Alcohol may have been the most devastating of these substances because it was long and widely consumed by pregnant women. But the administration of opiates to infants also appears to have been widespread for some stretches of time and may have been as widespread among the upper classes as it was among the lower ones. Even salt, in the quantities in which it was consumed prior to the development of refrigeration, was a toxic substance that may have taken a heavy toll at later ages. We are just beginning to become aware of the full range of ingested toxic substances and their role in the high mortality rates of the early modern and early industrial eras. More attention needs to be paid to the role of a variety of contaminants, including lead, arsenic, snakeroot, and mold poisoning (Ackerknecht 1952; Scrimshaw 1983; Matossian 1984).

Preoccupation with diet, especially the excessive focus on adult diets, has diverted attention from an array of intrauterine occurrences that undermined nutritional status and raised mortality rates during infancy and early childhood. Overwork of pregnant mothers and bacterial infections of minor consequence to mothers could have caused serious retardation of fetal development, especially when the insult occurred during the first trimester (Hurley 1980; Kielmann et al. 1983; Moore 1983; Steckel 1985). Intrauterine infections not only contributed to the large proportion of low-weight births, but increased the incidence of birth anomalies that severely affected the respiratory, circulatory, renal,

skeletal, immune, and neurological systems and thus undermined physical development throughout the first year, and often well into the second year and beyond (Fitzhardinge and Steven 1972a, 1972b; Bjerre 1975a, 1975b; McFarlane 1976; Shapiro et al. 1980; Christianson et al. 1981; McCormick 1985). Whether caused by a poor diet, by toxic substances, by overwork, or by intrauterine infections, low birthweight increased not only perinatal death rates but also late infant and early childhood death rates. Recent studies suggest that the incidence of arteriosclerotic diseases at middle and late ages may be promoted by adverse intrauterine and infant environments (Forsdahl 1977; Marmot et al. 1984).

The preliminary results indicate that the factors contributing to the unanticipated cycles in heights and mortality were concentrated at particular ages, and that the routes of influences might have been quite round about. These findings point to new issues in the standard-of-living controversy. It may turn out that the difficulties created by improved transportation and rapidly growing cities carried over into the rural regions surrounding the cities, so that urban disamenities imposed costs on the rural populations that have not yet been measured (Pollard 1981; Williamson 1981a, 1981b, 1982). In the American case it is difficult to believe that per capita food consumption was declining during the last two-thirds of the nineteenth century since there is so much evidence pointing in the opposite direction (Gallman 1960; Towne and Rasmussen 1960; Bennett and Pierce 1961). Yet there could have been more unequal distribution of food products, especially of meat, which adversely affected the nutritional status of the poor. This appears to have been the case with blacks, whose nutritional intake apparently declined and whose mortality increased, between 1860 and 1880 (Frissell and Bevier 1899; United States Department of Labor 1897; Atwater and Woods 1897; Fogel and Engerman 1974; Meeker 1976). A more subtle and possibly more pervasive effect on the living standards of laborers and their families, both in the cities and in the countryside, may have come from increased exposure to risks (not captured or only partially captured by current measures of real wages) that more than offset the rises in consumption. This possibility does not invalidate indexes of real wages which were designed to cope with a specific set of issues. Rather it raises new issues which require new measures, measures that will supplement the information obtained from the older ones.

The new findings suggest that much more attention needs to be given to the way that population pressures, urbanization, and other economic factors affected not just those of working age but the very young. It may well be that the main damage to the standard of living of workers occurred at exceedingly young ages, in ways that no one at the time fully appreciated, and in a manner that does not conform well to current scenarios regarding the factors and individuals responsible for the hard-

ships of working-class life during the nineteenth century. Nutritional and other health insults delivered in utero or early life appear not only to have affected adult health and longevity but to have reduced significantly the later productivity of those who recovered from early insults (cf. Fogel et al. 1983).

The search for data sources capable of dealing with both the new and the old issues on the interrelationship between demographic and socioeconomic variables has gained considerable force in recent years. Scholars have pushed in many different directions, and nearly all of the work has borne fruit. Careful examination of published data on disease-specific causes of death in United States cities have revealed that expenditures on sewers and waterworks had a relatively small effect on the decline in urban mortality before the beginning of the twentieth century (Condran and Crimmins-Gardner 1978), that the main diseases in which rural death rates were consistently lower than urban death rates in 1890 and 1900 were those which are nutritionally sensitive, and that the urban-rural differential was greater for infants and young children than for older persons (Condran and Crimmins 1980). These findings, although consistent with the nutritional hypothesis, raise questions about the role of exposure to disease, a variable that could not be measured in these studies. The weak relationship between public health expenditures and mortality rates could reflect the propensity of cities with the most virulent environments to make the heaviest expenditures. Similarly, urban-rural differences in disease-specific mortality rates might be more a matter of differences in exposure rates than in nutritional status. Such issues have led to a search for data sources that make it possible to measure exposure rates.

In this connection, there is much to be gleaned from a reexamination of published data in both state and local sources which can now be more effectively exploited than previously because of computers. Close examination of such published sources have revealed subtle aspects of the mortality structures (Preston 1976) and of influences upon them that were not adequately appreciated in the past. Condran and Cheney (1982), for example, have found that in Philadelphia during 1870–1930, medical intervention was effective, despite the absence of "high-tech" chemotherapy, because of the role of medical personnel in spreading knowledge about the environmental sources of diseases and in isolating carriers of diseases. Among the more suggestive findings of these recent studies of published data was the discovery by Higgs (1979) of marked cycles, around a declining trend, in the mortality rates of 18 large American cities between 1871 and 1900 that are strongly associated with variations in the rate of immigration.[62]

Work on the manuscript sources is still at an early stage, but as the studies of Wrigley and Schofield (1983), Preston and van de Walle (1978), Haines (1983), and Preston and Haines (1983) have already

indicated, these sources will permit us not only to push the empirical analysis of the causes of the decline in mortality further back in time but also to shed light on factors that are not apparent in published data. Linked micro data sets will make it possible to disentangle factors that are intricately convoluted in aggregate data. The ability to measure the separate and joint effects of nutritional status, disease exposure rates, medical practices, public sanitation, and intergenerational transmission of behavioral patterns will illuminate the past and directly contribute to a better understanding of important issues in current economic and social policies.

Appendix A
On the Construction of Figure 9.1

Table 9.A.1 gives the time series on height and on e_{10} used in the construction of figure 9.1.

The Height Series

The entries from 1710 to 1875 give the mean adult heights of native-born whites who were ages 25–49 at the time of measurement. Each observation is the mean height of a 5-year birth cohort centered on the indicated date. The various cohort averages in the time series were obtained from regressions on height with dummy variables for each 5-year birth interval. To prevent changes in the socioeconomic composition of recruits into the army from introducing spurious variations in the trend, a variety of control variables were introduced, including civilian occupation, state or region of birth, state or region of residence at time of enlistment, date of enlistment, number of previous enlistments, rural and city-size variables, and migration variables. The effects of alternative functional forms for the time trend and various transformations of the other variables were also investigated. The time trend proved to be robust to these alternative specifications. We chose to present the time trend generated by the dummy variables since this specification put the least constraint on that trend. The results of the regression analysis will be more fully reported at a later date. Some of the findings have been reported in Sokoloff and Villaflor (1982) and Margo and Steckel (1983).

There are three gaps in the time series on height (1760–75, 1800–1810, and 1880–1900). The data needed to close these gaps have been retrieved from the muster rolls but work on the processing of these data has not yet been completed. Consequently, the first two gaps were

Table 9.A.1 Data for Figure 9.1

$e°_0$		Height	
Years on Which Observation Is Centered	Entry	Year on Which Observation Is Centered	Entry (cm)
1720–24	51.8	1710	171.5
1725–29	52.7	1715	172.2
1730–34	52.0	1720	171.8
1735–39	51.2	1725	172.1
1740–44	52.9	1730	172.1
1745–49	52.3	1735	171.7
1750–54	52.5	1740	172.1
1755–59	52.9	1745	172.0
1760–64	53.9	1750	172.2
1765–69	53.7	1755	172.1
1770–74	54.8	1760	
1775–79	55.2	1765	
1780–84	56.4	1770	
1785–89	56.5	1775	
1790–94	56.7	1780	173.2
1795–99	55.4	1785	173.2
1800–1804	55.2	1790	172.9
1805–09	53.0	1795	172.8
1810–14	52.3	1800	
1815–19	51.9	1805	
1820–24	51.4	1810	
1825–29	51.1	1815	173.0
1830–34	51.0	1820	172.9
1835–39	50.2	1825	173.1
1840–44	48.7	1830	173.5
1845–49	48.2	1835	173.1
1850–54	47.9	1840	172.2
1855–59	47.8	1845	171.6
1860–64	49.2	1850	171.1
1865–69	51.4	1855	170.8
1870–74		1860	170.6
1875–79		1865	171.1
1880–84		1870	171.2
1885–89		1875	170.7
1890–94		1882.5	168.9
1901	50.6	1887.5	169.2
1910	51.3	1892.5	169.0
1920	54.1	1897.5	170.0
1930	55.0	1902.5	170.0
1940	57.0	1906.5	171.6
1950	59.0	1911	172.2
1960	59.6	1916	172.9
1970	59.8	1921	173.2
		1931	175.5

closed by linear interpolation. The third gap was closed by interpolation on the trend in the height of native-born recruits into the Ohio militia (Steckel 1982b).

The observations on the birth cohorts for 1905.5–1931 were obtained from Fogel et al. (1983). They pertain to all whites, foreign as well as native.

The Series of e_{10}

The first step in the construction of time series on e_{10} was the construction of period life tables for 5-year intervals extending from 1710–14 through 1875–79. The value of e_{10} for each of these life tables was then computed. The series was smoothed by taking a five-item moving average of the e_{10}'s. In other words, each observation of e_{10} listed in table 9.A.1 reflects mortality experience over a 25-year period centered on the midyear of a given interval.

Time series of e_0 and e_5 were also constructed. The secular trend in these series are quite similar to that generated by e_{10}. However, there appears to be a significant undercount of infant deaths among cohorts born before 1850 and a smaller undercount in ages 1–4. There are data in the genealogies that may permit us to correct the deficiency but the work on this problem is still in progress. Although the listing of deaths at ages 5–9 appears to be virtually complete, we chose to work with e_{10} until we completed our analysis of the problem of undercounting at younger ages.

The observations from 1901–70 give e_{10} for all whites, foreign as well as native. The life tables from which these statistics were computed are based on the states in the death registration system which expanded from 10 states in 1900 (with 26% of the national population) to the entire nation in 1933 (United States Bureau of the Census 1975, p. 44). The observations for 1901, 1910, 1920, 1930, 1940, and 1950 are each for 3 years centered on the designated year (United States National Office of Vital Statistics 1963). The observations for 1960 and 1970 are each based on data for a single year (United States National Center for Health Statistics 1963, 1974).

Appendix B
The Derivation of Equation (5)

Equation (5) may be derived from the identity:

$$S = \frac{D_i}{B} \cdot \frac{B}{P} + \frac{D_n}{P_n} \cdot \frac{P_n}{P} \tag{5.1}$$

which may be rewritten as

$$S = I\alpha + S_n\beta. \tag{5.2}$$

Total differention of (5.2) yields

$$\frac{dS}{S} = \frac{I\alpha}{S}\left(\frac{dI}{I} + \frac{d\alpha}{\alpha}\right) + \frac{S_n\beta}{S}\left(\frac{dS_n}{S_n} + \frac{d\beta}{\beta}\right). \tag{5.3}$$

Since

$$\frac{I\alpha}{S} = \frac{\frac{D_i}{B} \cdot \frac{B}{P}}{\frac{D_i + D_n}{P}} = \frac{D_i}{D_i + D_n} = \phi,$$

equation (5.3) may be rewritten as

$$\dot{S} = \phi\,(\dot{I} + \dot{\alpha}) + (1 - \phi)\,(\dot{S}_n + \dot{\beta}), \tag{5.4}$$

where

D_i = the number of infant deaths
D_n = the number of non-infant deaths
B = the number of births
P_n = the number of non-infants alive at midyear
P = the total number of individuals at all ages alive at midyear
S = the age-standardized death rate
I = the infant death rate
S_n = the age-standardized non-infant death rate
α = the crude birthrate (B/P)
β = the proportion of non-infants in the population (P_n/P)
$\dot{}$ = an asterisk over a variable indicates the rate of change in that variable
$\phi = [D_i/(D_i + D_n)]$ = the share of infant deaths in total deaths

It follows that if the birthrate and the age structure of the population are held constant, which is the assumption of the computation presented in the text, then equation (5.4) reduces to equation (5), since $\dot{\alpha} = \dot{\beta} = 0$.

The preceding derivation rests on two assumptions which require further consideration: First, the mortality schedule was treated as if it consisted of just two ages, without demonstrating that the reduction of the number of age intervals from z to 2 does not invalidate the results. Second, since the percentages employed in equation (6) are large, there is the question of the appropriateness of applying an equation derived from differential approximations to discrete changes.

The first question can be addressed by beginning with the definition of an age-standardized rate, which is given by equation (5.5):

$$S = \frac{P_0}{\Sigma_0 P_i} M_0 + \frac{P_1}{\Sigma_0 P_i} M_1 + \ldots + \frac{P_z}{\Sigma_0 P_i} M_z, \tag{5.5}$$

where

P_i = the number of individuals at age i
$\Sigma_0 P_i$ = the total population
M_i = the age-specific death rates.

Here, the $P_i/\Sigma_0 P_i$ are fixed from one time period to another; in other words, only the M_i change so that $S_0 \neq S_i$ is due only to temporal differences in the M_i. If we let $\alpha_i = (P_i/\Sigma_0 P_i)$, equation (5.5) becomes

$$S = \alpha_0 M_0 + \alpha_1 M_1 + \ldots + \alpha_z M_z. \tag{5.6}$$

Total differentation of equation (5.6) yields

$$\dot{S} = \frac{D_0}{\Sigma_0 D_i} (\dot{M}_0 + \dot{\alpha}_0) + \frac{D_1}{\Sigma_0 D_i} (\dot{M}_1 + \dot{\alpha}_1) + \ldots + \frac{D_z}{\Sigma_0 D_i} (\dot{M}_z + \dot{\alpha}_z). \tag{5.7}$$

However, since by definition $\dot{\alpha}_i = 0$, equation (5.7) reduces to

$$\dot{S} = \frac{D_0}{\Sigma_0 D_i} \dot{M}_0 + \frac{D_1}{\Sigma_0 D_i} \dot{M}_1 + \ldots + \frac{D_z}{\Sigma_0 D_i} \dot{M}_z = \frac{\Sigma_0 D_i \dot{M}_i}{\Sigma_0 D_i}. \tag{5.8}$$

Now the sum of all the terms on the right-hand side of equation (5.8), except for the first term, is

$$\frac{\Sigma_1^z D_i \dot{M}_i}{\Sigma_0^z D_i} = \dot{M}_x. \tag{5.9}$$

By induction, from equation (5.8), the average rate of change in the age-standardized rates above age 0 is

$$\dot{S}_n = \frac{\Sigma_1 D_i \dot{M}_i}{\Sigma_1 D_i}. \tag{5.10}$$

If we let $\Sigma_1 D_i = D_n$ and $\Sigma_0 D_i = D_0 + D_n$, then it follows from equations (5.9) and (5.10) that

$$(D_0 + D_n) \dot{M}_x = D_n \dot{S}_n, \text{ or } \frac{D_n}{D_0 + D_n} \dot{S}_n = \dot{M}_x. \tag{5.11}$$

Substituting equation (5.11) into equation (5.8) yields

$$\dot{S} = \frac{D_0}{D_0 + D_n}\dot{M}_0 + \frac{D_n}{D_0 + D_n}\dot{S}_n. \tag{5.12}$$

Then letting $\phi = \frac{D_0}{D_0 + D_n}$ and $\dot{I} = \dot{M}_0$, yields

$$\dot{S} = \phi\,\dot{I} + (1 - \phi)\dot{S}_n, \tag{5.13}$$

which is the same as equation (5).

The discrete analogue to equation (5.4) may be derived as follows:

$$S_1 = I_1\,\alpha_1 + S_{n1}\,\beta_1, \tag{5.14}$$

$$S_0 = I_0\,\alpha_0 + S_{n0}\,\beta_0, \tag{5.15}$$

$$\alpha_1 = \alpha_0 + \Delta\alpha \tag{5.16}$$

$$S_1 = S_0 + \Delta S \tag{5.17}$$

$$S_{n1} = S_{n0} + \Delta S_n \tag{5.18}$$

$$\beta_1 = \beta_0 + \Delta\beta. \tag{5.19}$$

$$I_1 = I_0 + \Delta I \tag{5.20}$$

Subtracting equation (5.15) from (5.14) and making the substitutions indicated in equations (5.16) – (5.20) yields

$$\Delta S = \alpha_0\Delta I + I_0\Delta\alpha + \Delta I\Delta\alpha + \beta_0\,\Delta S_n + S_{n0}\,\Delta\beta + \Delta S_n\,\Delta\beta. \tag{5.21}$$

Then, dividing both sides of (5.21) by S_0 and multiplying the first right-hand term by I_0/I_0, the second by α_0/α_0, etc., yields

$$\dot{S} = \phi(\dot{I} + \dot{\alpha} + \dot{I}\cdot\dot{\alpha}) + (1 - \phi)(\dot{S}_n + \dot{\beta} + \dot{S}_n\cdot\dot{\beta}), \tag{5.22}$$

where

$$\phi = \frac{I_0\alpha_0}{S_0} = \frac{D_{i0}}{D_{i0} + D_{n0}}.$$

Equation (5.22) is the discrete analogue to equation (5.4), and these two equations differ only by the two interaction terms $\dot{I}\cdot\dot{\alpha}$ and $\dot{S}_n\cdot\dot{\beta}$. Since by nature of an age-standardized death rate $\dot{\alpha}$ and $\dot{\beta}$ are equal to zero, equation (5.22) also reduces to equation (5).

Notes

1. Richard Hellie has called my attention to "the coincidence of the lowest life expectation [in table 9.2 above] with the Maunder Minimum (the Little Ice Age)," and notes that "the declining life expectations of the English peerage parallels the worsening

of the enserfment process in Russia, with the nadir of life expectations coinciding with the completion of the enserfment process." He suggests "that low yields and the generally unhealthier-than-usual climatic conditions" may have "played a role in driving both processes." From a letter to R. W. Fogel dated July 17, 1984.

2. From comments made at the Bellagio Conference on Hunger and History, June 1982.

3. The principal disadvantage of an emphasis on whole books is that a single aberrant book will have a large influence on the whole sample. Although such instances can be handled by reweighting, the aberrations reduce the efficiency of the sample and diminish its usefulness for some purposes. This problem will diminish as the sample size increases. The final sample will contain over a thousand books.

4. The mortality file is smaller than the family file partly because a requirement for entry into that file is that both the birth and the death dates of an individual are known. Nonbloodline spouses, who are at risk only after their marriages, have not yet been integrated into the mortality file. Their inclusion will increase the size of the mortality file by about 25%.

A family is defined by the existence of marriage, whether or not the family produces progeny. Families with multiple marriages have not yet been integrated into the family file, but they are a relatively small percentage of the families already in the file. Bloodline individuals who marry will appear in both their families of birth and the families formed by their marriages. The number of families suitable for the computations of various statistics varies because of the completeness of information. For example, it is possible to compute total births in completed families for about 77% of the families. In most of the other 23% of the families, date of publication of the genealogy preceded the end of the childbearing period of families at risk to have children. However, mother's age at last birth can be computed only for about 35% of the families since computation of this statistic not only requires that the date of publication of a genealogy follow the end of the childbearing period, but also requires information on the date of birth of both the mother and of the last child.

The small percentage of the individuals in the sample who have been linked to economic information reflects the recent start on this task. As of May 1984 we had searched for economic information on only 20% of the individuals in the sample. In other words, so far we have been able to obtain economic information on about 75% of the individuals for whom this information was sought.

5. The Mormon Church was not founded until 1830. The religious objective behind the compilation of family group sheets required the identification of ancestors who had not been Mormons. There are three parts to the collection of family group sheets. The *Main Section* consists of about 4.7 million sheets submitted mainly before 1962 and contains information on about 25 million individuals, only a small proportion of whom are Mormons. The *New Patrons Section* was started in 1962 when members of the Mormon Church were asked to submit sheets on the most recent four generations of their families. There are about 1 million sheets in this part of the collection, and a fairly large proportion of the individuals in these sheets are Mormons. The *Old Patrons Section* was launched in 1924 and consists of any genealogical records held by Church members that they desired to place on deposit in the Genealogical Library. There are about 4 million sheets in this part of the collection. The DAE/CPE pilot sample of group sheets is drawn from the *Main Section*. See Wimmer 1984.

6. About 43% of males age 20 and over had zero real wealth. The largest endowment in the linked genealogical sample thus far is about $200,000 which falls into the top 0.01% of the national distribution estimated by Soltow (1975) for 1850.

7. The first term was suggested by P. H. Lindert at the Williamsburg meeting.

8. In practice "health" is usually defined as "freedom from clinically ascertainable disease" (Great Britain Department of Health Social Service 1980). Measures of health include morbidity rates, mortality rates, number of GP consultations, days absent from school, disability days, days absent from work, parental assessments, weekly laboratory cultures, and weekly clinical diagnosis. Responses to key questions (e.g., "Do you have any physical difficulty with shopping?") are also used (Balinsky 1975; Blaxter 1976; McKeown 1976b; Sackett et al. 1977; Martorell 1980).

9. I do not mean to argue that average stature cannot be employed to assess factors affecting the general level of health in a population, but only to argue against the notion

that the health and stature are synonyms. The relationship between growth retardation and disease is complex, varying from one disease to another, and from one context to another. Consequently, while it may be possible to use data on stature to make valid statements about the probable level of exposure to certain diseases, a great deal more will have to be learned than is now known about complex interactions within a multivariate context. (The same caveat applies to attempts to infer diet from stature without controlling for exposure to disease and other relevant variables.) An oversimplified approach which assumes strict proportionality or some other simple relationship between stunting, or its absence, will miss or greatly underestimate nearly all respiratory diseases, most chronic diseases, many infectious fevers, and most nonspecific disorders—much of the range of diseases occurring among well-fed populations. Indeed, in one study anthropometric measures and the incidence of nonspecific health disorders were positively and significantly correlated (Butler 1974; Martorell et al. 1975; Condon-Paoloni et al. 1977; Briscoe 1979; Beisel 1977; Cole and Perkin 1977; Mata et al. 1977; Frisancho 1978; Martorell 1980; Baumgartner and Mueller 1984). On the other hand, I believe that there is enough evidence around to permit us to investigate and establish the robustness of these complex interrelationships. Given adequate patience, caution, and a good deal of ancillary information, we may be able to establish the likely combination of factors which affected growth profiles. In secs. 9.3 and 9.4 of this paper attempts are made to identify situations in which such inferences may be tenable.

10. Because the series of e_{10} is not yet controlled for the variables that were controlled in producing the height series, considerable caution needs to be exercised in interpreting leads and lags which are evident in the two series of fig. 9.1.

11. See appendix A for a discussion of the procedures employed in constructing the two time series displayed in fig. 9.1. Table 9.A.1 gives the values of each of the entries.

12. Levy's life tables begin with age 25. The e_{10} values shown for his data in figure 9.1 were extrapolated to age 10, using the model West tables of Coale and Demeny (1966).

13. Levy also has a life table for 1650–99 which was used to establish the location and slope of the line segment between 1715 and 1725 which shows the effect of a possible correction for the undercount of southern observations in the genealogical sample employed in fig. 9.1.

14. At the Williamsburg meetings Paul A. David and Warren C. Sanderson called our attention to a time series of e_{10} contained in a dissertation by Kent Kunze (1979) which overlaps with our series. Kunze covers the period from 1800 to 1880 in 14 life tables for males (and an additional 14 for females). His first table is based on a 15-year period (1800–1815), the second on a 10-year period (1815–25), and the remaining 12 are for quinquennia centered on years ending with zero or five. Kunze's data were obtained from samples of the family group sheets in the files of the Genealogical Society of the Church of Jesus Christ of Latter-day Saints. Only information on individuals who resided outside of Utah were included in his computations. Foreign-born as well as native-born white individuals were included.

Over the years in which they overlap, Kunze's series and the DAE/CPE series depict quite similar patterns. Both show a fairly steady decline after 1805. His series bottoms out in 1865 and then rises. The DAE/CPE series bottoms out 1857 and then rises. The decline of e_{10} in Kunze's series from 1807 to 1865 is 10%, which is the same decline as that shown in the DAE/CPE series between 1807 and 1857. However, between the peak of the DAE/CPE series (which occurred 1792, about 15 years before Kunze's first observation) and its trough, the decline in about 16%. (All dates are approximate.)

15. An improvement in the diet is not by itself a sufficient basis for inferring an increase in final heights, since a substantial secular deterioration in environmental conditions could have offset the potential improvement in nutritional status associated with a better diet. Based on current knowledge of the epidemiology of Massachusetts during 1675–1720, however, such a deterioration seems unlikely (Duffy 1953; Vinovskis 1972).

16. The elasticity of the mortality rate with respect to height in Trinidad was estimated from regressions (and the mean values of the variables in these regressions) reported in a memorandum from Meredith John to Robert W. Fogel dated November 30, 1983. I used four of John's logit regressions relating the probability of surviving to a series of variables including height. These regressions were for males $\geq$ age 15 (p. 36), females $\geq$ age 15 (p. 39), males $\leq$ age 15 (p. 42), females age $\leq$ 15 (p. 43). The elasticities for

these four groups (estimated over the arc between the average height of each group of Trinidadians and the current British height standard for the mean age of each group) were: $\epsilon_{m<15} = -2.00$, $\epsilon_{f<15} = -1.54$, $\epsilon_{m\geq 15} = -2.50$, $\epsilon_{f\geq 15} = 0$. The average of these four elasticities (weighted by the share of each group in the total population of Trinidad) was -1.57. If English population weights in 1801–5 are used (Wrigley and Schofield 1981, p. 529), the average elasticity is -1.44 (the sex ratio was assumed to be equal both above and below age 15, since Wrigley and Schofield do not give the sex ratio by age). United States population weights for 1980 (United States Bureau of Census 1983, p. 33) yield an elasticity of -1.33. Population weights for England and Wales for 1851 and 1961 (Mitchell 1975, p. 52) yield elasticities of -1.41 and -1.32.

17. The eight nations included in the Floud (1983b) study and the dates covered are Belgium (1880–1969), Denmark (1880–1975), France (1880–1960), Italy (1880–1952), Netherlands (1877–1970), Norway (1880–1960), Sweden (1880–1961), and Switzerland (1884–1957).

18. Total differentation of eq. (3) yields

$$\dot{I} = -15.9106\dot{H} - 0.3889\dot{Y} - 0.00837, \tag{3.1}$$

where an asterisk over a variable indicates the rate of change in that variable. Regressions of the log of each of the variables in equations (2) and (3) on time yielded the following estimates of average annual rates of change:

Variable	*Average Annual Rate of Change (%)*
$\dot{C}$	-1.05
$\dot{I}$	-2.51
$\dot{H}$	0.0610
$\dot{Y}$	1.75

It follows that height accounts for 39% ($15.9106 \times 0.0610 \div 2.51 = 0.39$), income for 27% ($0.3889 \times 1.75 \div 2.51 = 0.27$) and time for 33% ($0.837 \div 2.51 = 0.33$) of the average annual decline in the infant death rate.

19. The similarity between the results of eq. (2) and (3) and John's (1984) set of logit regressions on the probability of dying in Trinidad should not obscure the significant differences in the nature of the two sets of regressions. The Trinidad regressions related the *own* height of an individual to *his or her* probability of dying between two points in time. Equation (3), on the other hand, relates the average height of males (mainly in their early twenties) to a nation's infant death rate. When used in this way adult heights have only indirect bearing on the nutritional status of infants. Since the adult heights measure the nutritional status of males during the preceding 2–3 decades, and since the mean nutritional status of a nation exhibits high serial correlation, such a lagged measure of average nutritional status may be a fairly good predictor of a nation's current nutritional status especially during a period when the nutritional status of particular nations has been changing in a fairly steady way. Adult heights also have a bearing on current infant mortality rates to the extent that they indicate the nutritional circumstances of mothers during their developmental years. When comparing several nations, lagged measures may be a good predictor of differences in current nutritional status if the different nations had different starting levels and different rates of change in nutritional status.

20. The figure 64.5 inches is the estimated mean height of the pool of adult males from which recruits into the Royal Marines were drawn. Floud and Wachter have not yet completed their examination of possible sample selection and other biases in their military samples. Consequently, the use of 64.5 inches as an estimate of the mean final height of English males reaching maturity around 1800 must be considered tentative and is subject to revision. However, no currently plausible revision (the possibilities of change are pretty well bounded by $\pm$ 1.0 inches) would substantially alter the estimates of the effect of improved nutritional status on the decline in non-infant mortality rates.

21. Neonatal deaths are those which occur within the first 28 days of life. Perinatal deaths are late fetal deaths (generally of 20 or 28 weeks of gestational age) plus early neonatal deaths (generally deaths during the first 7 days after birth). In the United States

and Great Britain during the early 1960s the distribution of infant deaths was approximately as follows (Shapiro et al. 1968):

Days	*%*	*Cumulative Percentage*
1	40	40
2–7	25	65
8–28	8	73
29–365	27	100

Late fetal deaths in Britain and the United States have recently been approximately equal to neonatal deaths (United States Bureau of the Census 1983, p. 77). For countries experiencing death rates in the range of 100 per thousand and over, neonatal deaths range between 35% and 70% (Bouvier and Tak 1976; Mata 1978, table 2.16; Ashworth 1982).

Wrigley and Schofield (1981, p. 97) estimate neonatal deaths in England during the seventeenth and eighteenth centuries at about half of infant deaths. However, the practice of treating infant deaths during the 7–9 days as stillbirths, common in England and America before 1900 and still common in some high-mortality societies today, suggests that neonatal death rates may have been substantially underestimated. Although Wrigley and Schofield wrestled with this problem, the procedure they developed for correcting the bias may only have captured a part of the undercount in infant deaths. Their procedure rests on the assumption that the count of deaths during the last 11 months of infancy is virtually complete, and that there is a linear relationship between cumulated deaths (per thousand births) during the course of the first year and the cube of the log of days (plus one) elapsed since birth. Their procedure is described briefly in Wrigley and Schofield (1981, pp. 98–100), and at greater length in Wrigley (1977); see Knodel and Kintner (1977) and Hogan (1976), for discussions of the linearity assumption. Other reasons for a possible substantial undercount of mortality by Wrigley and Schofield, especially after the turn of the nineteenth century, are suggested by Lindert (1983). The small stature of the English workers prior to 1820 and the high correlation between the infant death rate and stature at age 3 and at maturity (see Floud 1985a; Steckel 1985) also indicate that infant mortality rates were substantially higher than suggested by Wrigley and Schofield. However, because of the widespread consumption of alcohol and other toxic substances by pregnant women, and the probable undercount of deaths during the first 7–9 days, 0.6 seems to be a more appropriate figure. See n. 34 and sec. 9.4.2 below.

22. However, in recent years new high technology introduced into maternity hospitals in the more developed nations has led to some downward shift in this schedule, especially for births of less than 1,500 grams that are of early gestational age but not otherwise impaired (Jones et al. 1979; *Lancet* 1980, p. 481; Pharoah and Alberman 1981).

23. The distribution of birthweights is not normal, mainly because there are too many observations in the left tail. The fat left tail may be treated as the result of adding together a distribution of the weights of underdeveloped babies (which I will call "pre-term") to a much larger distribution of fully developed babies (which I will call "full term"). Nevertheless, for the purposes to which they are put in fig. 9.5, normal approximations to the distribution of the United States nonwhite and Bombay lower classes yield satisfactory results. The mean and standard deviation in the normal approximation to the United States distribution differs from those of the actual distribution by less than 1% (Chase 1969). In the Bombay case the difference in the means is less than 1% (the standard deviation of the sample was not reported) (Jayant 1964).

24. In this case it was necessary to estimate the heaping of preterm births on the left tail of the distribution of birthweights. My procedure was based on the proposition set forth in fn. 23 that the observed distribution of birthweights may be viewed as the result of a convolution of a small distribution of weights of preterm babies which is heaped on the left tail of a much larger distribution of weights of full-term babies that is normally distributed. Under this assumption the underlying normal distribution can be recovered by truncating the left tail of the distribution at (say) 2,001 grams and then using the QBE procedure described in Wachter (1981) and Wachter and Trussell (1982) to estimate the

complete normal distribution of full-term babies. It follows that the difference between the number of observations below 2,001 grams in the reconstituted normal distribution and in the original distribution yields an estimate of the distribution of preterm babies that have been heaped on the left tail.

In estimating col. 4 of table 9.10, which is graphed in fig. 9.5, I assumed that the underlying full-term distribution was $N(2{,}300; 420)$. To this distribution I then added the estimated number of preterm births at weights below 2,001 grams, using ratios obtained from Guha et al. (1973) which provides information on birthweight by gestational age for a Delhi sample quite similar to the Bombay sample. Basically, the number of births under 2,001 grams in the original normal distribution was inflated by the ratio of all births to full-term births in the Delhi sample, but the additional births were distributed over two intervals: under 1501 and 1,501–2,000. This adjustment for preterm births produced a convoluted distribution with a mean of 2,276 grams (down 24 grams from the underlying normal) and a standard deviation of 399 grams (down 21 grams from the underlying normal).

Although the mean birthweight of the hypothetical distribution is quite low, it is consistent with the final height of English workers 1800–1810 and the high infant death rates indicated by current estimates (before adequate allowance is made for the undercount of deaths during the first 9 days after birth). In this connection, I would emphasize the likelihood that the mean birthweight in rural areas of countries such as India and Bangladesh is below those prevailing in the hospitals of their large cities. This inference is consistent with evidence indicating that incomes and final heights are generally lower (and the infant death rates are higher) in the rural areas of these countries than in the cities. In the villages covered by the Narangwal experiment, e.g., the mean birthweight was below 2,500 grams (Kielmann et al. 1983). Moreover, babies born to women of a given socioeconomic class in hospitals are liable to be higher by several hundred grams than those born to women of the same class at home because of the phenomenas discussed in fn. 25.

See Steckel (1985) for an alternative procedure that produces similar results.

25. My estimate of the mean birthweight of lower-class children is about 590 grams (about 1.3 pounds) less than mean weights of about 27,000 births at the Maternité de Port Royal in Paris delivered during the first decade of the nineteenth century (Tanner 1981, pp. 255–56). I suspect that this differential was due partly to late third-trimester weight gains associated with much improved diets received by mothers while at the Maternité. Until the late nineteenth or early twentieth century lying-in hospitals, often called prematernity homes, functioned more as charities aimed at rescuing destitute women and their often illegitimate children than as scientific institutions. The aim of these hospitals was to provide food, clothing, and shelter to "poor and desperate" women who were awaiting delivery, rather than to offer a surgical type of facility (Vogel 1980, pp. 12–13). Destitute expectant mothers were often kept in these hospitals for several weeks or longer on a nutritious diet before their deliveries. A study of birthweights at English prematernity homes shortly after the turn of the twentieth century revealed that the birthweight of children whose mothers worked up to the day of confinement averaged 280 grams less than those of mothers who were confined to the homes for 10 days (Ashby 1915).

Since 45% of the increase in fetal weight normally takes place during the last 8 weeks of pregnancy (Thomson et al. 1968; Birkbeck 1976; Southgate 1978), birthweights at the Maternité may well have exceeded the average of babies born to working-class women in England and France during the first decade of the nineteenth century by the 590 grams suggested by the birthweight distribution of fig. 9.5. The combination of an enforced sedentary life together with a substantial increase in the intake of nutrients for several weeks could have led to a substantially larger gain in the weight of fetuses than would otherwise have been the case. The exceedingly high death rates among deliveries in the maternity hospitals of the nineteenth century, as much as seven times as high as home deliveries (McKeown 1976a), is consistent with this possibility. One would expect an unusually high rate of stillbirths and neonatal deaths among fetuses that suffered first trimester insults, even though they had high levels of nutrition during the third trimester (Hurley 1980; Moore 1983).

In the case of food-supplemented pregnancies (when supplementation begins late in, or after, the first trimester), mean birth size may not be as good a predictor or perinatal

and neonatal mortality rates (Kielmann et al. 1983) as in unsupplemented feeding regimens. This discrepancy may be due to the diversity of responses to first trimester insults and later supplementation. Some pregnancies will abort early. Some fetuses that have suffered permanent impairment during the first trimester will continue to develop (at a retarded rate) and will be at high risk late in the third trimester and after birth. Still, others will fully recover from the first trimester trauma and, as a result of supplementation, will experience third trimester weight gains that are greater than they otherwise would have been. It seems likely, therefore, that a very poor first trimester diet (or other nutritional insult) combined with supplementation in the third trimester will increase the coefficient of variation in birthweights (with an uncertain effect on the mean weight, since supplementation may increase the proportion of small-for-dates fetuses that are born alive). This appears to have been the case in the Maternité. Its coefficient of variation is 26% higher than that of a sample of 43 populations with low mean birthweights (under 3,000 grams) reported in the WHO survey (1980).

Two recent studies, one on births between 1851 and 1905 at the University Lying-In Hospital in Montreal (Ward and Ward 1984), the other births between 1848 and 1865 at the Philadelphia Alms House (Goldin and Margo 1984), promise to increase our knowledge of nineteenth-century birth size and of its bearing on perinatal deaths during that century. The Wards have discovered a decline in mean birthweight of about 420 grams between the late 1860s and the beginning of the twentieth century. The Wards are also collecting birthweight data for the nineteenth and early twentieth centuries from a number of other lying-in hospitals in North America and Europe. Preliminary analysis of the Philadelphia data by Goldin and Margo not only indicates a decline in birthweights between the mid-1850s and the mid-1860s, but also reveals that the first-day death rate, even for live births in the range of 3,000–4,000 grams, was about twice as high as in the United States national sample in 1950. Goldin and Margo are also collecting data that will relate the duration of the stay in the Alms House prior to birth to birthweights and to perinatal mortality rates.

26. I have not distinguished birthweight and length by sex since it is a refinement not justified by the rough calculations which follow. At birth the mean weight of girls is less than that of boys, but the difference is only about 100 grams (about 3 ounces). The average difference in birth length between the sexes is about 0.6 centimeters (about a quarter of an inch) and shows less variation across nations than weight differences. Cf. Tanner et al. 1966; Hytten and Leitch 1971; Eveleth and Tanner 1976; Beal 1980; and the sources cited in WHO 1980.

I have used 0.6 rather than the 0.5 figure employed by Wrigley and Schofield as the share of infant deaths that occurred during the neonatal period, partly because of the widespread ingestion of toxic substances by pregnant women (cf. the discussion in nn.21, 33, 34, and in sec. 9.4.2 above).

The percentage reduction in the infant death rate due to a shift in the birthweight schedule is quite robust to the principal assumptions employed in table 9.10. If, for example, I had used assumptions which yielded an than indicated in table 9.10), the decline in the infant mortality rate due to the shift in the birthweight schedule would still be 76% [1 − (48.9 ÷ 200.0) = 0.76].

27. See app. B for the derivation of eq. (5).

28. Wrigley and Schofield (1981, p. 529) give the following values for 1801–5:

$$e_0 = 35.89$$

$$cdr = 27.08$$

$$cbr = 37.71.$$

The appropriate value of ${}_1Q_0$ (179.0 per thousand) for the indicated value of e_0 was obtained from their table A14.5 (p. 714) by interpolating between their levels 8 and 9. Then $37.71 \times 0.179 = 6.42$ is the number of infant deaths per 1,000 persons in the total population. Consequently, infant deaths were 23.7% of all deaths ($6.42 \div 27.08 = 0.237$).

29. The age-standardized death rate in Britain circa 1980 was 8.32 per thousand, using the Wrigley and Schofield (1981, p. 529) age distribution for 1801–5 and the 1978–80 life table for the United Kingdom to estimate the ${}_nm_x$ values (Great Britain Central Statistical Office 1983, p.43). Since the crude death rate for 1801–5 in Wrigley and

Schofield (1981, p. 529) was 27.08, the decline in the standardized mortality rate is 18.76. Non-infant deaths were 20.66 per thousand in 1801–5 (0.763 × 27.08 = 20.66). Then 20.66 × 0.1085 = 2.24 is the reduction in non-infant mortality due to improved nutrition. The last figure is 12% of the total decline in mortality (2.24 ÷ 18.76 = 0.119).

30. Some caveats about the foregoing estimates are in order. After maturity, height will not adequately measure nutritional status unless the relationship between nutritional status during the growing ages and after maturity is not only strong but of a simple form. Consequently, the computations presented in the text may miss part of the effects of improvements in nutritional status after maturity on the decline in adult mortality. The assumption that the elasticity between ${}_nQ_x$ and height is stable with respect to time, place, and circumstances (which is involved in the application of the Trinidad elasticity to the British case) requires confirmation and may have to be modified as additional evidence becomes available.

31. Roger Schofield has called my attention to evidence which suggests that diseases listed in the first column of table 9.11 might have accounted for the majority of deaths among the peerage well before 1750. I do not mean to suggest that the shift in the distribution of diseases is the main factor explaining the decline in the death rate of the peerage, but only that it might have played some role. The principal point of this paragraph is that improvements in nutritional status could not have played a significant role in reducing the death rates of either the peerage or of the lower classes as long as the main killer diseases were those whose outcome was unaffected by nutritional status. Just when this shift took place, or even whether it took place, is still an open issue. Some students of medieval demography believe that the outcome of epidemics of medieval plague, or at least some forms of it, may have been influenced by nutritional status. See, e.g., Hellie 1982.

32. From a letter to R. W. Fogel dated July 16, 1984.

33. A half-pound of hard-salted herring, bacon, or similarly cured meat per capita per day (and upper-class individuals may have consumed that much at breakfast alone—cf. n. 34) would have exceeded maximum "safe" levels of sodium consumption for an average individual by more than fivefold. Salt was also used much more heavily than today to cure and flavor butter (about 1.5 ounces of salt per pound of butter) and other dairy products, and in bread (five pounds of salt per sack of flour) and porridges. Sodium occurs naturally in significant quantities in various root vegetables, pulses, milk, beer, meats, and fish (Encyclopedia Britannica 1961, 2:888; Ashbrook 1955; Webster 1845, pp. 747, 773–833, 1135; Davidson et al. 1979; Meneely and Battarbee 1976; United States Senate 1977).

34. According to Dyer (1983, pp. 193–94) a gallon of ale was the standard ration for adult males in "lordly households" during the late medieval era, but superior members of the household consumed two or three pints of this amount as wine. At Northumberland the Lord and Lady split a quart of beer and a quart of wine at breakfast alone (along with two pieces of salt fish and six baked herrings), and even the children in the nursery consumed a quart of beer at breakfast. Children of all classes drank ale or beer, but usually of a weaker type than their parents (Pullar 1970; Wilson 1973). The per capita ration of ale for nuns at Syon was 7 gallons per week (Pullar 1970, p. 111). According to Thurgood (1984, p. 6) the accounts of the first Duke of Buckingham during the mid-fifteenth century indicate that daily consumption of wine among "upper members of the household" was about a half gallon per capita. If peeresses consumed two-thirds of a gallon of liquid, half as ale and half as wine, then their daily consumption of absolute alcohol would have been between 7.2 ounces (allowing 4% of absolute alcohol in ale and 11% in wine) and 9.1 ounces (if the percentage of absolute alcohol in ale is set at 8%). Even if peeresses drank only weak ale (4% absolute alcohol) their daily consumption of absolute alcohol would still have averaged about 3.4 ounces. Recent studies indicate that among women whose daily consumption of absolute alcohol during pregnancy equaled 2 or more ounces per day, about one out of five newborns suffered from symptoms of Fetal Alcohol Syndrome (Hanson et al. 1978; Abel 1982). Heavy salt intake further endangered fetuses by causing edema and by increasing the likelihood of kidney damage (United States Senate 1977).

35. The balance of this section presents a highly condensed version of the analysis set forth in Fogel (1987). See that paper for the derivation of the equations and the estimates of parameters that are merely reported in this section.

36. Estimates of the social distribution of the population based on revisions of King's table (Lindert 1980; Lindert and Williamson 1983b) would not significantly affect the analysis that follows.

37. The provisional nature of the estimates of these parameters and of those set forth in eq. (11) should be emphasized. Although consistent with the available evidence thus far developed for the early modern era, current research into estate and probate records and similar sources should permit improvements in these estimates. See Fogel (1987) for a discussion of the effects of plausible variations in these parameters on the estimates of entitlement shifts, foodgrain shortfalls, overall food shortfalls, and famine-induced mortality rates. Cf. the discussion in n. 40.

38. A yield/seed ratio of 8 implies that 12.5% of the crop was used for seed, and grains used as feed for work animals may have accounted for a like amount by the beginning of the eighteenth century. Hoskins (1964, 1968) and other economic historians have implicitly assumed that the seed elasticity of demand was close to zero. However, econometric estimates of agricultural production functions, as well as controlled experiments by plant breeders, suggest a fairly high elasticity of substitution between seeds and other inputs. That finding, together with the theory of demand for inputs, suggests that 0.6 is a reasonable estimate of ϵ_s (Fogel 1987).

39. The grain price series used here includes peas and beans, which were also used to make bread during the early modern era. The prices are from Bowden (1967, 1985). Missing entries in the peas and beans series were linearly interpolated. Peas and beans were then added to the Bowden grain series, using a weight of 0.165 each for peas and beans and 0.67 for Bowden's grain series.

40. The assumption that yields were lognormally distributed (there is a slightly better fit of grain prices to a lognormal than a normal distribution) would have little effect on the analysis that follows. Under the lognormal assumption the estimated value of σ_g would rise by only 4% (from 0.066 to 0.069). In this paper I have, therefore, employed the normal assumption in order to simplify the exposition. See Fogel (1987) for the results obtained when the analysis is based on a lognormal distribution of yields.

41. I believe that this estimate of σ_g is probably too high. Since I did not take account of a hoarding and speculative demand, my assumption that $\epsilon_t = 0.37$ probably biases the value of σ_g upward. If, for example, hoarding demand reduced ϵ_t to 0.25, σ_g would be just 0.045.

It is possible to estimate σ_g directly for the first 30 years (1884–1913) that output and yield-per-acre data are available in Great Britain (Mitchell and Deane 1962). Yields on wheat, barley, and oats were combined into an index of grain yields (using equal weights), and then detrended by regressing the index on time. The S.D. of the errors divided by the mean of the index, σ_g, was 0.051. However, σ_g is determined not purely by deviations in the per acre yields of these grains but by deviations from trend in the total supply of all grains. When total supply of all grains (broadly defined) was taken into account, the estimate of σ_g for the period 1884–1913 dropped to 0.035 (Great Britain House of Commons 1899, 1917; Great Britain Ministry Agricultural Fishery 1927). These two figures (0.051 and 0.035) may be taken as reasonable estimates of the bounds within which σ_g probably fell during 1540–1871 (cf. Fogel 1987).

If we now substitute 0.051 for σ_g in eq. (12), that equation becomes $1.2 \times 0.051 = 0.061\bar{X}_g$. Hence, we can estimate ϵ_t from $\log 0.939 = \epsilon_t \log 1.25$, which yields a value of 0.282 for ϵ_t. Similarly, if we use 0.035 for σ_g, the estimate of ϵ_t becomes 0.192. These computations suggest that failure to take account of the speculative demand may have biased the estimate of ϵ_t used in the text upward by between 31% and 93%. The correction of σ_g and ϵ_t would also reduce the estimates of ϵ_{gw}, σ_f, and ϵ_{fw} by similar proportions. Cf. n. 46 below.

42. The S.D. of deviations of wheat prices from a 25-year moving average was computed from the price series of Bowden (1967, 1985). Lee (Wrigley and Schofield 1981, p. 374), using a somewhat different series of wheat prices, found that the SD of the deviations around an 11-year moving average was 0.25 during 1540–1640 and 0.24 during 1641–1745.

43. The S.D. in the total food supply (σ_f) was estimated from the "all-agricultural-products" price series developed by Bowden (1967, 1985). The deviations in food prices were computed from a 25-year moving average of this series. The weight of food in Bowden's series varies between 87% and 97%. When the Phelps Brown and Hopkins

series (1956) of the price consumables (in which food has a weight of 80%) is used, σ_f is 0.043.

44. This estimate of the decline in the food supply is based on Bowden's (1967, 1985) all-agriculture price series. When the price series of consumables constructed by Phelps Brown and Hopkins (1956) is used, equation (16) becomes

$$\epsilon_{fw} = 0.172 r_{fw}. \tag{16.1}$$

This equation also implies that there were only 8 years with food shortfalls in excess of 7%. In setting up eq. (15), I assumed ϵ_f for all food was the same as for all grain, which probably biases the estimate of σ_f upward.

45. The first figure in the parentheses is the proportionate increase in the national cdr due to a 23% decline in food consumption by the class of common laborers. The second figure is the proportion of years ($1 \div 41$) in which such a decline occurred. For reasons explained in n. 41 and 46, the upper bound on famine deaths as a percentage of accumulated total deaths during 1541–1871 may be as low as 0.1

46. Lee (Wrigley and Schofield 1981, pp. 370–73) finds a sharp decline in fertility between 3 and 9 months after a rise in wheat prices, which he attributes to first and second trimester fetal mortality. Not all fetuses that suffered nutritional insults would have died. One would also expect an increase in neonatal death rates because impaired fetuses survived the entire gestation period but died shortly after birth. In other words, some part of the apparent fertility decline in year zero may be due to a rise in infant mortality that was unmeasured. If we add half of the zero-year elasticity of fertility decline to the zero-year elasticity of mortality rise, Lee's 5-year cumulated elasticity of mortality rates with respect to a rise in wheat prices becomes 0.163.

The last figure is not far from the elasticity implied by my computation, which is about 0.23. A 23% decrease in the food consumption of the class of common laborers requires (from eq. [14] and the accompanying text) a 70% increase in wheat prices and will lead to a 16% increase in the national cdr in such a year. Hence, the elasticity of the national cdr with respect to wheat prices is $0.16 \div 0.70 = 0.23$. For various reasons that I have suggested, this last figure is an upper bound. If, for example, I had made allowance for a speculative demand for wheat, the elasticity of the mortality rate with respect to wheat prices implied by my computations would have been between 0.163 and 0.094. Cf. n. 41.

47. This widely used assumption in the analysis of time series on grain prices was questioned by Landes in 1950, but his caveat has been largely unheeded.

48. A similar point was made by Flinn (1974, p. 315) in a different context. Citing the work of two French demographic historians (J. Meuvret and J. Lebrun), Flinn argued that after the beginning of the eighteenth century "improved social organization prevented the very poor from actually dying from starvation during a famine, but it did not rescue them from their more permanent state of undernourishment which left them vulnerable to both endemic and epidemic disease. This kind of social action, in other words, leveled the unevennesses of mortality without reducing it in aggregate."

49. Galenson (1981) has shown that the majority of English immigrants to North America were from the lower classes. If it is assumed that the mean height of adult male immigrants before 1650 was 64.5 inches (the approximate mean male adult height of the English laboring population for cohorts born ca. 1750), then the implied rate of increase in adult height between immigrants born ca. 1630 and native cohorts born ca. 1710 was about 1 centimeter per decade. There is no information currently available on the mean height of English cohorts born before 1750. However, the high mortality rates in England between 1625 and 1790 (Wrigley and Schofield 1981, pp. 528–29) suggest that there was little change in height schedules during this period. (Cf. Palliser [1982] on conditions during 1300–1640.)

50. Swedish and Norwegian adult heights appear to have increased fairly rapidly during the first third of the nineteenth century. However, the secular increase slowed during the middle third of the century and accelerated during the last third (Kiil 1939; Udjus 1964; Sandberg and Steckel 1980).

51. The estimate of the Gini ratio for the distribution of income ca. 1900 (0.47) was obtained from Steckel's regression, Gallman's income estimate for that year (converted in dollars of 1970), and the adult height estimate for ca. 1900 derived from the data in

fig. 9.1. Alternative specifications of Steckel's regression, with dummies for high-income countries raised the estimated Gini ratio for ca. 1900 to about 0.50. A Gini ratio can be computed from the income distribution for 1900 estimated by Lebergott (1976) from budget surveys. That computation yielded a Gini ratio of 0.56.

52. The equation used in this computation can be derived as follows:

$$H = (1 - \pi) H_u + \pi(H_u - D). \tag{17.1}$$

Hence

$$H = H_u - \pi D. \tag{17.2}$$

Differentiating (17.2) totally yields:

$$\dot{H} = \psi \dot{H}_u - (\psi - 1)(\dot{\pi} + \dot{D}) \tag{17.3}$$

where

H = the mean height of the population
H_u = the mean height of the upper class
D = the difference between the mean height of the upper and the lower class
π = the share of the lower class in the total population
ψ = $H_u \div H$
$\dot{}$ = an asterisk over a variable indicates the rate of change in that variable.

In eq. (17.3) both $\dot{D}$ and $\dot{\pi}$ only enter into the second right-hand term. Thus, we can measure the share of increase in the mean height of the English that is due to the growth of the upper class just by computing $\psi \dot{H}_u \div \dot{H}$. The values of the variables needed to compute this ratio and the sources of the estimates are as follows:

$H^0 = 64.7''$ (a weighted average of the mean height of the pool from which British soldiers aged 23–35 were recruited into the British armed forces in 1810 and of the implied final height of Sandhurst boys (1820), using a weight of 0.05 for the share of the gentry in the English population. The share is from Laslett (1984). The Sandhurst height data are from the unpublished files of Floud described in table 9.6).
$H^1 = 68.9''$ (Rona et al. 1978, table 3).
$H_u^0 = 68.9''$ (The Sandhurst sample, and the ratio of the final height to heights at age 15 computed from the United States Civil War sample).
$H_u^1 = 69.9''$ (Rona et al. 1978, table 3).

These data yield the following estimates (rates of change are in % per annum; the period of change was assumed to be 150 years):

$$\dot{H} = 0.042$$

$$\dot{H}_u = 0.010$$

$$\psi = 1.040\ [0.5(68.9 \div 64.7) + 0.5(69.9 \div 68.9)]$$

and

$$\frac{\psi \dot{H}_u}{\dot{H}} = 0.25.$$

53. It is likely that immigrants too poor to provide their growing children with adequate nutrition (which may be viewed as an intergenerational transfer of human capital) also were unable to provide children with adequate transfers of ordinary capital.

54. The quote is from a letter to R. W. Fogel dated November 30, 1984. Similar points were called to my attention by S. Peltzman and P. A. David.

55. It is, of course, possible that the increase in Midwestern per capita income was accompanied by a rise in the inequality of the income distribution sufficient to impoverish a substantial percentage of the region's population. Even if it is unlikely that the per-

centage so impoverished would have been large enough by itself to explain the observed height decline, it could have been a nontrivial contributing factor.

56. There is also the possibility that part of the decline is a statistical artifact. It might be argued that children of the farm families who enlisted during the peacetime years of the 1880s came, on average, from poorer families than those who enlisted during wartime years. On the other hand, the mean height of the native-born population as estimated from the recruits who joined the regular army during the peacetime years of 1850–55 was 68.1 inches, which is just one-tenth of an inch below the corresponding figure for the Union army (Sokoloff 1984).

57. Our investigation of available data sources indicated that there is enough quantifiable information on the disease environment of localities in the recruiting and station records of the United States Army, in the surveys of medical societies and public health officials, and in mortality records to be able to construct time series on the extent of exposure to particular diseases by counties (by wards within large cities) or at least by congressional districts. We have, therefore, initiated a new project aimed at collecting and analyzing these data.

58. Meade (1955) and Kuznets (1959) called attention to the fallacies that may arise in growth accounting when the effect of mortality rates on the measured growth in per capita income is neglected.

59. This point was made in another context by Butlin (1971).

60. For the period 1790–1860 the best current estimate of the average annual rate of increases in United States "real" per capita income appears to be about 0.71% (David 1967; Engerman and Gallman 1983). The mortality increase between 1790 and 1860 involved a shift from the life table at level 16 to that at level 10 in West Model Female (Coale and Demeny 1966). The values of δ computed the $\int_x$ schedules of these tables during the prime working ages (taken to be ages 20–50) was 1.10% for level 10 and 0.58% for level 16. Hence, the average value of $\bar{\delta}$ over the 70 years from 1790 to 1860 was 0.91%. The value of $(1 - \phi)$ was computed from

$$(1 - \phi) = \frac{0.5 \times 1.10 + 0.5 \times 0.58}{(0.5 \times 1.10 + 0.5 \times 0.58) + 6.0} = 0.123.$$

The values V for 1790 and 1860 were computed as follows. First, the number of live births required to produce one survivor at age 20 was computed from ${}_{20}l_0$, which turned out to 1.18 births at mortality level 16 and 1.45 birth at level 10. Then, V_{1860}/V_{1890} was computed from

$$\frac{V_{1860}}{V_{1790}} = \frac{1.45X_1 \int_0^{20} e^{(-\alpha_1 - \delta_1 + i_1)t} dt}{1.18X_0 \int_0^{20} e^{(-\alpha_0 - \delta_0 + i_0)t} dt},$$

where

X = the net expenditures on child rearing at birth

and

α = the average annual rate of decline in X.

It was assumed that $X_1 = X_0$, that $\alpha_1 = \alpha_0 = 0.08$, and that $i_1 = i_0 = 0.06$. The values of δ_1 and δ_0 were computed from ${}_{20}l_0$ for mortality levels 10 and 16, respectively, and turned out to be 0.0170 for δ_1 and 0.0075 for δ_0. Consequently, $\dot{V} = \left(\frac{20.4915}{18.1527}\right)^{\frac{1}{70}} - 1 = 0.0017$. The result is fairly robust to alternative plausible assumptions regarding the values of X, α, and i. It follows from eq. (19) that the average annual rate of increase in "real" wages due to the rise in mortality is 0.28% ($0.877 \times 0 + 0.123 \times 0.91 + 0.17 = 0.28$).

The relationship between the rate of change of "real" per capita income and the average "real" wage is given by

$$\dot{w} = \dot{\beta} + \dot{Y} - \dot{L} = \dot{\beta} + \dot{\bar{Y}} - \dot{\rho} \tag{19.1}$$

or

$$\dot{\bar{Y}} = \dot{w} - \dot{\beta} + \dot{\rho} \tag{19.2}$$

where

w = the "real" wage

$\bar{Y}$ = "real" per capita income

β = the labor share in income

Y = "real" national income

L = the number of workers

ρ = the labor force participation rate.

Hence, if $\dot{\beta}$ and $\dot{\rho}$ were unaffected by the rise in mortality (both appear to have been low during 1790–1860 [David 1967; Gallman 1972; Engerman and Gallman 1983]), then $\dot{\bar{Y}}$ will be equal to $\dot{w}$. It follows that the mortality adjustment reduces the rate of growth in "real" per capita income by about 39% (0.28 ÷ 0.71 = 0.39).

61. The decline in the mortality rates of low-income countries since 1950 is even more remarkable than the mortality decline in the industrialized nations between 1700 and 1980. The less developed nations have accomplished in 3 decades what took two centuries or more in the industrialized nations. A significant part of this acceleration is due to the transfer of medical and economic technology from the industrialized nations to the less developed nations (cf. Preston 1976, 1980, 1985).

62. That the debate launched in the mid-1950s still continues should not distract attention from the considerable advances in knowledge that have occurred because of the debate. Investigators have probed increasingly into aspects of issues that were obscure at the outset. The point is well illustrated by the evolution of research on the pathways of airborne diseases. McKeown (1976a) stressed direct exposure; Preston and van de Walle (1978) called attention to the risk-increasing effects of the lowering of resistance to airborne pathogens brought about by infections caused by waterborne pathogens. Thus, in the course of the debate the concept of nutritional status has been refined and the factors which affect it have been elaborated. Similarly, Condran and Cheney (1982) have provided evidence that medical intervention became increasingly effective before the dramatic chemical breakthroughs that became apparent during and after World War II. However, the extent of mortality reduction due to these less dramatic contributions has yet to be measured.

Comment

Peter H. Lindert

Robert Fogel's excellent paper, and the larger research effort it draws upon, promises to be a durable landmark in the history of mortality trends. If subsequent documentation and independent estimates confirm key results that have only been sketched here, we will be citing his mortality time series and debating his interpretations for a long time. My task is to voice lasting praise and lingering questions on three fronts Fogel has opened: (1) his pioneering estimates of United States mortality trends from early colonial times through the Civil War, (2)

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his emphasis on the role of changes in "nutritional status," and (3) his helpful suggestions on several puzzles about the effects of material living standards on life expectancy.

United States White Male Mortality since about 1700

Thanks to the research team headed by Fogel, and to an independent study by Kent Kunze (1979), we are beginning to get a fresh look at changes in the length of life before the Civil War. The new estimates come from genealogical data, including the genealogies of ancestors of Mormons. The nature of this source imposes some limitations. The ancestral population was nearly all native white, precluding any measurement of nonwhite or immigrant life expectancy. In addition, serious underregistration of infant and early childhood deaths forces one to measure life expectancy only from later childhood, in this case from the tenth birthday, instead of from birth.

The tentative new contours of American mortality history are sketched in figure C9.1A. According to Fogel, life expectancy improved rapidly until the middle of the eighteenth century. This improvement first became noticeable in New England and then spread southward. By the 1750s white American colonists apparently lived longer than any other national population, even longer than British peers.[1] For the nineteenth century Fogel unveils a puzzling surprise: life expectancy for native white male 10-year-olds dropped by more than 4 years in the first half of the century, both in his sample and in that of Kunze. By the 1850s Americans faced shorter life spans than the English. This worsening of mortality was foreseen by only a slight plurality of previous studies, which lacked a clear consensus about antebellum mortality trends.[2] The new estimates present us with a

1. It should be noted that the pilot sample being used by Fogel and his associates contains very few observations per decade for the seventeenth and eighteenth centuries. Yet his tentative conclusions for the seventeenth and eighteenth centuries are at least consistent with the results of previous local studies. See the studies of colonial demography cited by Vinovskis (1972, pp. 184–213) and Fogel et al. (1978, pp. 75–108).

2. Some studies suggested improvement in life expectancy in some areas (e.g., Boston and perhaps the South) with no clear trend in others (e.g., in Massachusetts outside Boston). Again see Vinovskis (1972) and Fogel et al. (1978) and the sources they cite. Other studies, by contrast, saw an increase in antebellum mortality: (1) Duffy's study of New York City found that infant mortality and overall crude death rates both rose considerably across the first half of the nineteenth century (see Duffy, pp. 575–79). (I am indebted to my colleague Alan L. Olmstead for this reference.) (2) Meindl and Swedlund (1977, pp. 389–414) found that mortality worsened in two villages of central Massachusetts between 1790 and 1840. (I am indebted to Robert W. Fogel for this reference.) (3) Yasukichi Yasuba (1962, p. 89) found crude death rates rising in three cities, though not in two others, between 1804 and 1860. (4) See also the discussion in Kunze (1979, pp. 118–24).

Future acceptance of the Fogel-Kunze finding that mortality worsened will further discredit any comparison of Wigglesworth's 1789 life table with later tables for Massa-

major challenge: to explain a worsening in American health that lasted as long as three generations.

The surprising decline in life expectancy in the early nineteenth century cannot be explained by shifts in population toward less healthy areas. Urbanization was one such shift, but would have shortened life for 10-year-old males only by 1.3 years or less even if they shifted from being completely rural in 1800 to the true urban-rural mix of 1860.[3] This falls far short of accounting for the declines estimated by Fogel (a decline of 4.6 years from 1800–1815 to the trough of 1848–52) or Kunze (5.2 years from 1800–1815 to the peacetime trough of 1858–62). Nor is it likely that interregional shifts would explain the observed decline: the dominant westward shift was not a shift toward less healthy areas.

The early nineteenth-century fall from grace also clashes with what we would expect from the trend in real wages and income per capita. Even for the unskilled, real wages at least doubled between 1800–1809 and 1850–59.[4] Fogel rightly stresses the importance of such anomalous cases: What indeed do we make of historical periods in which real wages or incomes moved one way but life expectancy moved in the opposite direction? Such experiences warn against any simple belief that real income variables dominated life expectancy so strongly as to force the latter to rise and fall with the former.

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chusetts. Even after Vinovskis's (1971) careful adjustments and warnings, Wigglesworth would imply a life expectancy of only 36.5 years (both sexes) back in 1789, whereas comparable figures from around 1830 and 1860 imply 47–48 years. This improvement cannot coexist with the Fogel-Kunze results unless one imagines radically different trends between Massachusetts and the national native white population.

3. The figure of a decline of 1.3 years or less due to urbanization is derived as follows: Let us overstate the rural-urban gaps in white male life expectancy (e_{10}) for 1860 by taking some extreme values from Vinovskis's study. Vinovskis estimates that $e_{10} = 52.7$ years for all rural areas. Extend this figure to towns smaller than 2500, even though the small towns had slightly lower e_{10}. Vinovskis further estimates that $e_{10} = 51.9$ for males in towns with population between 2500 and 4999 and $e_{10} = 52.6$ for towns 5000–9999. For towns over 10,000 let us use the pessimistic Boston rate, $e_{10} = 44.2$, instead of the true grand average rate ($e_{10} = 46.7$). Compute an average national rate by using the actual population shares for the United States in 1800 and 1860 (it is necessary to use shares for all races, all ages, and both sexes in place of the missing rural and urban shares for 10-year-old white males). The results are shown in the unnumbered table below.

Mixture of places	e_{10}
Entirely rural	52.7
Actual 1800	52.4
Actual 1860	51.4

The extreme decline, from entirely rural to actual 1860, is only 1.3 years, even under the exaggerating assumptions just mentioned.

4. Specifically, the real wage rate rose 134% for Vermont farmhands and by 101% in the David-Solar estimates for nonfarm unskilled laborers (see Adams 1944; David and Solar 1977).

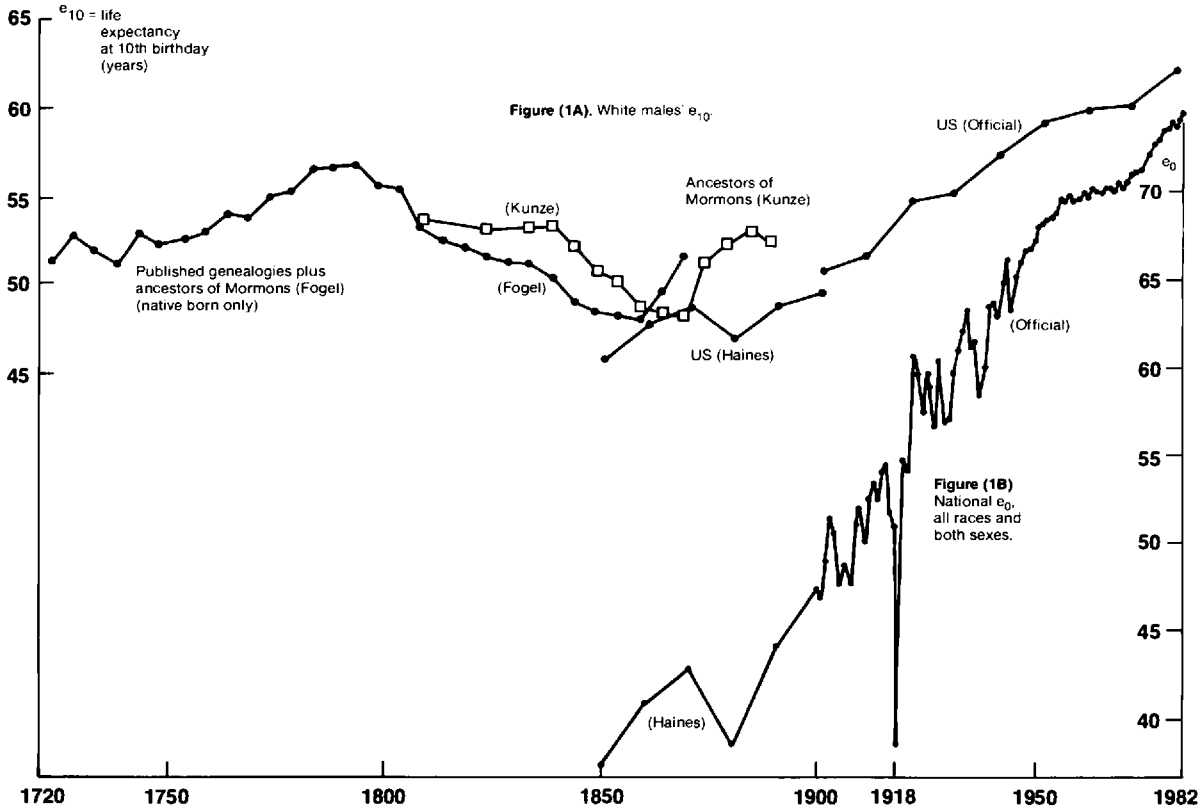

Fig. C9.1 Period measures of life expectancy, United States, 1720–1982. *Sources:* Fogel estimates are from his fig. 9.1 in this volume. Kunze estimates are from Kunze (1979, p. 113). Haines estimates are from Haines (1979, p. 307). United States official estimates are from United States Bureau of the Census (1976, 1: 55; 1983, pp. 73, 74); United States Public Health Service (1970); and (for male e_{10}, 1900–1960) Preston et al. (1972, pp. 2–11).

The new estimates of early American mortality also serve to dramatize the extent to which improvements have been concentrated in the period since the 1850s. Indeed, if fuller investigations bear out Fogel's chronology, American life expectancy showed improving trends only before 1790 and since 1860, and not during the intervening 70 years. The improvement since 1860 has been especially dramatic for infants, women, foreign born, and nonwhites. Figure C9.1B presents a series on life expectancy from birth to remind us of the extent of the improvement of these groups, and to pose two questions:

1. Could the rise in life expectancy before 1790 and the decline from 1790 to 1860 have been as dramatic as the rise after 1860? If e_0 and native-white-male e_{10} always followed the same life table relationship, sharing common tables from the same model, as they might have done after 1860, then yes, the earlier movements in e_0 were as dramatic as shown for years after 1860 in figure 9.C.2. We need more reliable estimates of e_0 before we can tell.

2. If the improvement in life expectancy since 1860 was indeed more dramatic than any swing that went before it, we need to renew past scholars' efforts to find out why.

The Role of "Nutritional Status"

Fogel goes beyond presenting new estimates and plunges into the difficult task of explaining his mortality results. His explanation features the concept of "nutritional status" so prominently that it dominates the title and substance of the paper. His careful conjectures about the possible role of nutritional status are a clear gain in knowledge. Yet he has chosen a concept that is sure to mislead at least some share of readers. To minimize that share, I must go beyond Fogel's own caveats and stress that "nutritional status" lacks most of the meaning that unwary readers are liable to assign it. Fogel defines nutritional status as "the balance between the intake of nutrients and the claims against it." What would enter into this balance? Almost every determinant of health and mortality would seem a candidate. To stress this vagueness, figure C9.2A's schematic portrayal simply refers to "other factors" as codeterminants, along with nutritional intake, of nutritional status. Furthermore, as shown in figure C9.2A, nutritional intake and the claims against it *jointly* determine this status, a jointness that complicates analysis. Fogel allows the search for determinants to be narrowed slightly by arguing at length that height is a good proxy for his concept of nutritional status (and a correlate of life expectancy). Some health and mortality factors are helpfully excluded thereby: childhood health factors that do not affect growth, plus influences impinging after the growth phase has ended. But this does not exclude much. We are left with a menu of possible influences on nutritional status that look

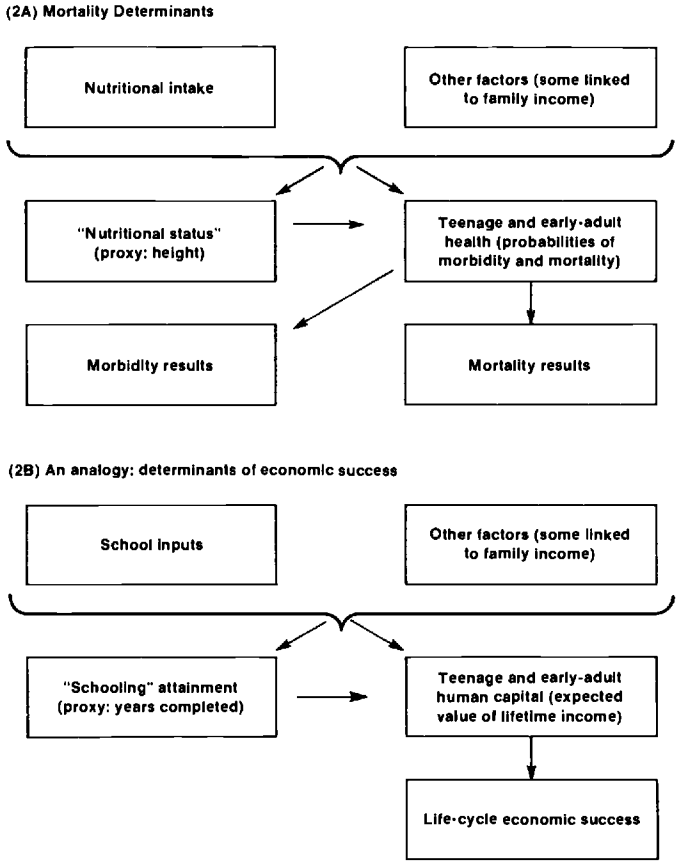

Fig. C9.2 Illustrative schematic diagrams of determinants of mortality and of economic success.

very similar to the influences on health itself, at least for the teen and early adult years. Little progress has been made toward weighing exogenous influences on mortality.

Employing a concept that is a near neighbor to teen and early adult health is not too dangerous by itself. But if the concept is labeled "nutritional status" it will be hard to keep readers from thinking that something significant has been said about the role of nutritional *intake*. That is not the case.

The dangers here can be underlined with an analogy from the familiar literature on the determinants of the lifetime economic success of individuals, as is diagrammed schematically in figure C9.2B. Just as one could call on height as a measure of nutritional status, one

could use years of schooling attainment as a measure of something called "schooling" and then proceed to argue that schooling is a good correlate of economic success. But little has been gained in our attempt to analyze the manipulable sources of economic success, since almost everything that might influence teen and early adult earning potential (human capital) and, through it, lifetime economic success, could influence the quality and years of one's schooling experience. Furthermore, emphasis on the importance of "schooling," like Fogel's emphasis on "nutritional status," invites misjudgment with its semantic link to school inputs. Precisely this confusion has led public debate to slip from the schooling-success correlation to overstate the case for extra investment in school inputs, overlooking the role of other schooling (and success) determinants such as family inputs into child development.

Living Standards and Life Expectancy

Fogel's paper offers some helpful suggestions and some provocative questions on the broader issue of how general purchasing power affects mortality.

One clear contribution is his short section on the surprisingly small effect of early modern English *harvest failures* on death rates (the "Wrigley-Schofield-Lee paradox"). With a minimum of fuss, he inverts our view of the wide swings in grain prices and real wages in the wake of harvest fluctuations. The percentage fluctuations in the harvest or in available supplies must have been smaller than those in grain prices, since price elasticities of demand for such staples are notoriously low. Thus a 20% jump in grain prices could have resulted from only a 5% grain harvest deficiency, in a low-income early modern England with only imperfect international trade in grains. This may be a key reason death rates were not more sensitive to grain price movements. It serves Fogel's argument by keeping his emphasis on the importance of nutrition from suffering criticism based on the low response of English mortality to grain prices: if the harvest failures involved small percentage shifts in grain supply, a small mortality response is still consistent with great unit impact of nutritional intake.

To this point well put, one should add only the other key point about early modern English experience with food and death: a given percentage of national harvest failure had less and less mortality effect with the passage from the sixteenth century to the early nineteenth. Prices themselves varied less and less from year to year and from region to region as storage and transportation improved. In addition, government intervened to give special food entitlements to the poorest, es-

pecially with the spread of poor relief specifically tied to the price of food after 1795 (Schofield 1983).[5]

A second living-standards area explored by Fogel is "the peerage paradox": why didn't peers live longer than the rest of the population before about 1750 despite their much higher incomes, and why did their life expectancy improve so much faster thereafter? To some this looks like evidence against a nutrition-centered explanation of mortality (Livi-Bacci 1982). Fogel resourcefully counters with the possibility that perhaps the nutritional intake of infants in peerage families was as bad as that for common babes until 1750, and then somehow improved both absolutely and relatively. He presents good prima facie evidence for deleterious dietary habits in peerage families in early modern times. When and why these habits were reformed remains uncertain. The peerage paradox remains high on the research agenda of British demographic history.

The final section of this paper links mortality to overall living standards rather than to nutrition. Here he stresses a point that is familiar yet seriously underemphasized: *inequality* of income and wealth *causes extra deaths* for any given average income per capita. It can do so because the dependence of life expectancy (or average height) on income is very nonlinear: the same extra income extends life much more near the margin of malnutrition than it does at higher incomes.[6]

In one respect, Fogel may be pushing this point too far. Regarding the antebellum United States, he argues for strong causal links among rising economic inequality, declining heights, and declining life expectancy, drawing on Steckel's regression results showing a strong impact of Gini coefficients on height (and, by extension, mortality) for any given income per capita. The inequality-health link is so strong, he suggests, that we can use heights and other data to predict unobserved antebellum Gini coefficients, and we should ask whether living conditions really improved before 1860: During an era in which 50%–75% of the income of workers was spent on food, is it plausible that the workers' overall standard of living was improving if their nutritional status and life expectation were declining?

Readers should take care not to infer that the antebellum decline in life expectancy and health would outweigh gains in more conventional measures of economic resources, leaving us with a net decline in "living

5. Thus early modern England is not the best context for conjecturing that social food entitlements, à la Sen, may have tended to break down.

6. See, in addition to the studies by Floud and Steckel cited in Fogel's paper, Preston (1976) and World Bank (1980). The relationship is nonlinear whether the dependent variable is life expectancy, death rates, or heights. While the most accessible evidence relates to international cross-sections, the results for cross-sections of individuals look similar.)

conditions." Any such judgment must await quantification of the net change in the value of disamenities, including those associated with the threat of morbidity and mortality. I think it likely that such quantification will show that the antebellum gain in real annual incomes outweighed the shortening of life plus the psychological cost of ill health and death risks, even for low-income groups.

A starting point for weighing changes in the value of health against conventional real income gains is to combine lifetime consumption plus final savings with the disamenities associated with the average living situation to derive a measure of adult material well-being. Drawing on work by Usher (1973), Williamson (1984), and others, we can employ the following measures of average material well-being (W) in two cohorts ($_0$ and $_1$):

$$W_0 = C_0 L_0(1 - d_0),\ W_1 = C_1 L_1(1 - d_1),$$

where C = average consumption and bequests, averaged over all persons in the relevant group and all years of the life cycle;
L = expectation of life, but with each extra year's survival chance discounted back to the present (here, to age 10) at a rate of time preference, as is done in Usher and Williamson;
d = the percentage disamenity discount associated with the living conditions of that cohort, as revealed in studies of adult's choices of jobs and places of residence.

To judge the impact of the antebellum changes in mortality Fogel has documented, let us compare a synthetic cohort based on conditions in 1800–1809 with one based on conditions in 1850–59. The ratio of the later to the earlier well-being is

$$(W_1/W_0) = (C_1/C_0)(L_1/L_0)(1 - d_1)/(1 - d_0).$$

If consumption plus bequests was in fixed proportion to the real wage rates for unskilled workers from 1800–1809 to 1850–59, then $(C_1/C_0) \geq 2$, as noted in n. 5. If life expectancy was proportional to the new e_{10} estimates developed by Fogel, then over the same period (L_1/L_0) slightly exceeded 0.866, a ratio based on comparing the low figure for 1855–59 with the high figure for 1800–1804. So far we have

$$(W_1/W_0) \geq 1.732\ (1 - d_1)/(1 - d_0).$$

Material well-being clearly advanced across the antebellum period *unless* psychological disamenities became much more severe ($d_1 >> d_0$).

If the antebellum (nonslave) population found urban life more unpleasant than rural life in its nonconsumption dimensions, then we could make some headway toward judging disamenity values by break-

ing the departure of $(1 - d_1)/(1 - d_0)$ from unit into three changes: the change in the quality of urban life, the change in the quality of rural life, and the change in the quality of life implied by the migration of a fraction of the population from rural to urban life. We can put an upper bound on the third change. American cities in the 1850s cannot have been as much worse than the American countryside of 1800 as appalling Manchester was worse than bucolic rural Norfolk in the late 1830s. In this extreme English contrast, Manchester employers offered unskilled workers a 65% annual wage premium over the annual pay given to comparable workers in low-wage Norfolk (Lindert and Williamson 1983). In other words, the disamenity discount for the worst urban-industrial life was less than or equal to $1 - (1/1.65)$, or 39.4%. Suppose that conditions of urban American life in the 1850s were that much worse than conditions for America as a whole in the 1800s. Then, combining the 1860 urban share of 19.8% with the 65% extreme disamenity, and assuming that city life was no worse than country life in 1800–1809, we get

$$(1 - d_1)/(1 - d_0) = 1 - (0.198)(1 - 0.394) = 0.880,$$

$$(W_1/W_0) \geq (2)(0.866)(0.880) = 1.524 .$$

The changes in real wages, length of life, and the disamenity of antebellum urbanization together imply that average well-being improved by more than 52%.

The most important omissions from these calculations are the net changes in the quality of life *within* the cities and *within* the countryside between 1800–1809 and 1850–59. If these can be shown to have deteriorated enough to pull (W_1/W_0) below unity, then Fogel will have led us to one of the most important findings in the history of modern economic well-being: a long era when things got demonstrably worse for the average American (or the average unskilled American worker) despite a large increase in annual purchasing power. Indeed, Fogel does help pave the way for such a conclusion by showing that heights (and, by presumption, life expectancy) dropped in both urban and rural settings before the Civil War. But the 52% improvement shown here is the mountain that must be removed if we are to believe that the best antebellum clues to overall material well-being were the mortality and health series he has unveiled.

Yet Fogel's key point about inequality and health remains. It carries an important policy suggestion: for countries whose poor are close to the malnutrition margin, poor relief (welfare payments) financed by the rich could lengthen life and increase the size of the labor force. In such settings, the life-extending effect of fiscal (or philanthropic) redistribution from rich to poor could even repay the taxed rich, partly or

completely or even more, through its tendency to expand the labor supply and enhance the pretax returns to property and skills.[7] The result depends on several aspects of our economy, in particular the elasticities of demand and supply for common labor, the elasticity of labor supply with respect to disposable income, the elasticity of supply of property (land, capital, skills), the level of taxation, and the share of unskilled earnings in national income.

Preindustrial England might have been a case in which greater poor relief would have taken Fogel's point to this paradoxical extreme. A large share of the population was malnourished, the levels of taxation and philanthropy were low, and a large share of national income accrued to the owners of relatively inelastically supplied land. In such a setting poor relief could have brought a net gain to the taxed propertied and skilled classes, thanks to its tendency to raise the supply of common labor (even without any Malthusian effect of poor relief on fertility). If so, it would be paradoxical that England was so much more tight-fisted in its opposition to poor relief before the revolutionary fears of 1795 and before the modern welfare state. Perhaps English history, like today's global cross-section of nations, finds poor relief least generous when (or where) it was most clearly a matter of life and death. This is another of the issues illuminated by Fogel's important preliminary findings.

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7. This argument assumes, of course, that the poor relief expands the population of labor-force age enough to outweigh any work disincentive from the fact that income maintenance reduces the postbenefit wage rate. In a very poor country, it might.

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