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NUTRITION AND THE DECLINE IN MORTALITY SINCE 1700: SOME ADDITIONAL PRELIMINARY FINDINGS

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ABSTRACT

This paper is an extensive revision and expansion of Working Paper No. 1402. It centers on a new time series of life expectations in the U.S. since 1720, which has been constructed from the NBER/CPE pilot sample of genealogies. Native-born Americans achieved remarkably long life expectations toward the end of the eighteenth century but then experienced a 70-year decline. A new rise began late in the 1850s but it was not until 1930 that the Americans again achieved the level of life expectation that was attained c.1790. Second, time series on average adult stature of national populations in North America and Europe are used as indexes of nutritional status (not diet alone but diet net of prior claims). These series are shown to be highly correlated with the series on e_{10} and other measures of mortality. It is estimated that improvements in nutritional status may have accounted for as much as four-tenths of the secular decline in mortality rates, but nearly all of this effect was concentrated in the reduction of infant mortality. Additional results include an assessment of the effect of toxic substances on the mortality rates of the English peerage; an estimate of the distribution of shortfalls in English supplies of food between 1540 and 1871, which reveals that famines were due primarily to social misallocations of food rather than to large declines in supply; and adjustments of conventional estimates of U.S. per capita income for the increase in mortality, which reduce the rate of economic growth between 1790 and 1860 by nearly 40 percent.

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1. The Issues

Between c.1700 and 1980 there was a decline of about 35 points in the standardized American death rate (see Table 1).* Between the same years, the British rate declined by about 21 points. About 70 percent of the American decline and about 50 percent of the British decline took place before 1911.

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 three 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 advances 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 birth weight 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.

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

Table 1

THE PROBABLE DECLINE IN STANDARDIZED DEATH RATES BETWEEN 1700 AND 1980 IN THE UNITED STATES AND GREAT BRITAIN

Part A. Standardized Death Rates (per thousand)

Approximate Date	United States	Great Britain
1. 1700	40	28
2. 1850	23	24
3. 1910	15	17
4. 1980	5	7

Part B. Percentage of the Total Decline Which Occurred Between c.1700 and the Specified Date

Approximate Date	United States	Great Britain
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 New England and Chesapeake rates weighted by the New England and Southern populations for 1700 as given in U.S. Bur. Cen. 1975, p. 1168. Line 2. Unpublished mortality tables for whites in 1850, cited in Haines, 1979. Line 3. Preston, Keyfitz, and Schoen, 1972, pp. 728, 730. Line 4. U.S. Nat. Cent. Health Stat. 1983, p. 12. Great Britain: The age distribution is standardized on the weights given in Wrigley and Schofield 1981, p. 529 for 1701-1705; male and female death rates were equally weighted. Line 1. Ibid. Lines 2 and 3. Case 1963, pp. 41, 53, 65, 76. Line 4. G.B. Cent. Stat. Off. 1983, p. 43.

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 c.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 percent of the decline was associated with airborne diseases, 28 percent with water- and food-borne diseases, and 18 percent 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 water-borne 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; McKeown 1978, p. 540). McKeown argues that improvements in

personal or public hygiene would not have reduced deaths from airborne 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 percent of its 1848-54 level by 1900 and to just 10 percent 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 for 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 to 67 percent of the deaths of children under age 5 in Latin America (1976a, p. 136).

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 to 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 between 30 and 40 percent. 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).

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 (Lindert 1983; Easterlin 1977; Vinovskis 1972).

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 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 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

Table 2

Cohort Life Expectation (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	
	II	55.3	41.5
	III	58.6	44.6
	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.

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.

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 clean up 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.

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 the circumstances of an individual. 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 or not 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 not only requires measures of food consumption but 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.

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 and 1980; Schuurman 1980; Lindert 1980; Lindert and Williamson 1983a; B. 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 (Demos 1970; Lockridge 1966; Greven 1970; Carr and Walsh 1980; Smith 1972; McCusker 1970; Menard 1975; Walsh and Menard 1974; Rutman and Rutman 1979; Fischer and Dobson 1979; Kulikoff 1976; McMahon 1980; Galenson 1981; Main 1982; Rothenberg 1984; Jones 1980; R. Gallman 1982; M. Gallman 1980; 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 U.S. 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 U.S.

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.

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 1,000,000 individuals in 200,000 families that will be linked intergenerationally for up to ten generations (see Table 3).

Table 3

Tentative Estimates of the Temporal Distribution of Observations in the Completed Genealogical Sample

Period	Families established during the period		Persons born or entering U.S. during the period	
	1 Number	2 Percent	3 Number	4 Percent
Before 1700	1,000	1	8,000	1
1700-1750	3,000	2	26,000	3
1751-1800	6,000	3	51,000	5
1801-1850	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 Appendix B, ibid., 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 column 3 does not count such individuals twice, the ratio of column 3 to column 1 for a generation is not equal to the average size of completed families during the period covered by that row.

During the past five 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 20,000,000 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 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 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 percent 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 10,000,000 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 samples 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 characterisitics. 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:

(1) $D_j = g_j(X_{ij}, B_{ij})$, 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 intervales, 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 4 compares their results with the means reported by Soltow (1975) for his random sample from the 1850 census schedules. Table 4 shows that although the means in the sample of Adams and Kasakoff 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 trace his lineage to the patriarch. Only then can he come forward in time to construct a complete

Table 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.	1,401	n.a.
Non-farmers	276	1,037	2803	n.a.	805	n.a.

Source: Adams and Kasakoff 1983; n.a. = not available

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 entirely 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 non-contraceptive 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 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 naitonal 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 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 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

Table 5

A COMPARISON OF A PERIOD LIFE TABLE FOR U.S. MALES DERIVED FROM THE GENEALOGICAL SAMPLE WITH TWO OTHER PERIOD LIFE TABLES

	A			B			C		
	Native-born whites, 1850-1860, derived from the genealogical sample			All whites, 1850-1860, average of Haine's tables for 1850 and 1860			All whites, 1900, registration states		
Age	$1000Q_x$	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 footnote 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, Keyfitz and Schoen, 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.

the ten 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 (U.S. Bur. Cen., 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 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 three 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 part 3 of this paper 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.

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 three, and then falls more slowly throughout the remaining pre-adolescent 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 two 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 U.S. 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 U.S. population and of the principal populations from which the U.S. 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 U.S. 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 birth weight. 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; Keilmann 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 upon environmental conditions. Consequently, mean height corresponds quite well to the type of measure of nutritional status called for in Section 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. Mosley 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 was 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 (Hytten and Leitch 1971; Waterlow et al. 1977; Habicht et al. 1979; Meredith 1970; Naeye 1981; Raman 1981; Thomson and Billewicz 1976; Goldstein 1976). 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 both reduces 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 (C.E. Taylor 1982; Kielmann et al. 1982; Kielmann et al. 1983). 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 percent 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 one and three 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 centile 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 three (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 (J. Interdiscip. Hist. 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 data base 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 3.2, 4.2, 4.3, and 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 Bureau project called "Secular Trends in Nutrition, Labor Welfare and

Table 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 (1-4)		
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 (5-10)		
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, 1807-1862	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)

Table 6 (continued)

Title of Samples	Number of Observations Originally Planned	Number of Observations Currently on Tape	Main Categories of Information Included	References
8. Colonial muster rolls, 1750-1783	20,000	14,000	Same as 1, except no mortality information	Sokoloff & Villaflor (1982)
9. Philadelphia Alms House, 1847-1877	4,500	3,000	Birthweight and birthlength, gestational age, birth order, characteristic of labor, mortality of child and mother during hospital stay, sex of child, race, characteristics of mother [age, ethnicity, residence, marital status, health status (eg. veneral, drunkeness)]	Goldin and Margo (1984)
10. Cost of living surveys, 1934-1877	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, 1813-1834	30,000	25,000	Height, age, color, births, deaths, and various other socio-economic variables for all ages and both sexes	Friedman (1982)
		British Samples (12-14)		
12. Marine Society,		50,000	Height, age, and various socio-economic characteristics including occupations of fathers and sons, literacy and vaccination (or inoculation); ages mainly 13-17	Floud and Wachter (1982)

Table 6 (continued)

Title of Samples	Number of Observations Originally Planned	Number of Observations Currently on Tape	Main Categories of Information Included	References
13. Sandhurst boys,	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 occu-	Floud (1983a)
Other European Samples (15-16)				
15. Swedish conscipt 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)

Labor Productivity," which is also co-sponsored by the Center for Population Economics.

The nutrition project currently involves a set of sixteen samples (see Table 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-aged 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 birth weights 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 percent 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 of which relate

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 techniques developed for coping with these problems (Trussell and Bloom 1979; Wachter 1981; Wachter and Trussell 1982; Fogel et al. 1982; Floud 1983a; Fogel et al. 1983; 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.

3. Some Preliminary Findings on the Relationship between Improvements in Nutritional Status and the Decline in Mortality

At the present time 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.

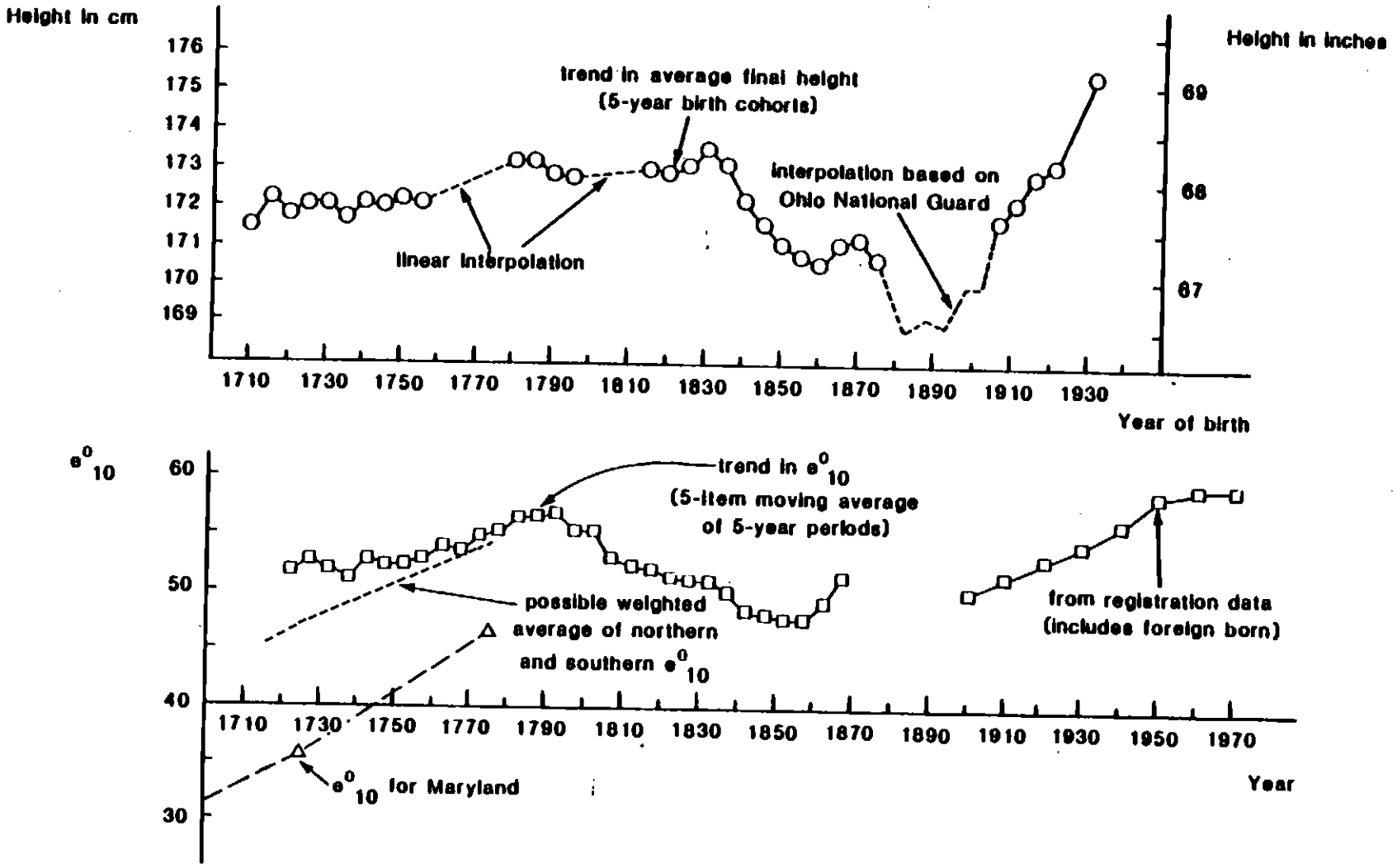
3.1 The Secular Trends in the Height and in the Life Expectation of U.S. White Males, 1700-1930

Figure 1 compares the time series that we have developed so far in both the height and the life expectation of U.S. 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-expectation series is not, but merely gives the mean life expectation 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-expectation 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 c.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 expectation of Maryland legislators. The value of e_{10} estimated from his data for 1700-1749 and 1750-1799 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 differential 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

Figure 1

A Comparison Between the Trend in the Mean Final Height of Native-Born White Males and the Trend in Their Life Expectation at Age 10 (e^{0}_{10})
(height by birth cohort; e^{0}_{10} by period)



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 expectation 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 c.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 c.1710 and c.1750 and the tall stature compared with the English in c.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 (1980). 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 c.1675 and c.1750 the average allotment increased from approximately 80 to approximately 168 pounds per annum: about half the increase took place by c.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 1980; Holmes 1907; Fogel 1986).[15]

Table 7
A Comparison Between the Cohort Life Expectations for Native-Born U.S. White Males, British Peers, and the English Population: 1700-1925

Century and Quarter		1 England and Wales (both sexes) e_0		2 British Peerage (males) e_0	3 British Peerage (males) e_{10}	4 U.S. 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 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 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 U.S. registration data in the sources listed in Appendix A and from U.S. Nat. Cent. Health Stat. 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 1968-1978 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 column 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 U.S. during the first quarter of the twentieth century.

Figure 1 and Table 7 reveal that Americans not only achieved modern heights by the middle of the eighteenth century, but that they reached levels of life expectation 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 c.1725 to about 47 years and the estimate for c.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 expectation 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 c.1725 and c.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 uncontrolled 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.

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 twenty 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 dispositon 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 8, which presents the heights of surviving and non-surviving males under age 26. The extent of the difference is more apparent in a regression format. Table 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 8 not only shows that non-survivors were shorter than survivors, but that even the survivors were exceedingly short by modern standards. Figure 2 indicates how bad their nutritional status was. In this diagram

Table 8
Mean Heights by Age and Mortality, 1813-1815, Trinidad-Born Males

	Survivors			Non-Survivors		
Age	Height	SD	N	Height	SD	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. Due to a transcription error the standard deviation of non-survivors were misreported in the original source. Those shown here, supplied by Friedman, are the correct ones.

3.3 Evidence From Regressions Between Height and Mortality

Table 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 rate with respect to height averaged about -1.4.[16] The effect of height on the death rate appears to have been greater among young children than adults, and on males than on females. There are reasons 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):

$$(2)\quad \hat{C} = 30.7877 - 5.3851\hat{H} - 0.0363\hat{Y} - 0.006647T$$

$$(5.292)(-4.534)\quad (-0.382)\quad (-4.040)$$

$$\bar{R}^2 = 0.85;\ N = 64$$

$$(3)\quad \hat{I} = 88.9781 - 15.9106\hat{H} - 0.3889\hat{Y} - 0.00837T$$

$$(12.327)\ (-10.797)\quad (-3.294)\quad (-4.213)$$

$$\bar{R}^2 = 0.96;\ N = 64$$

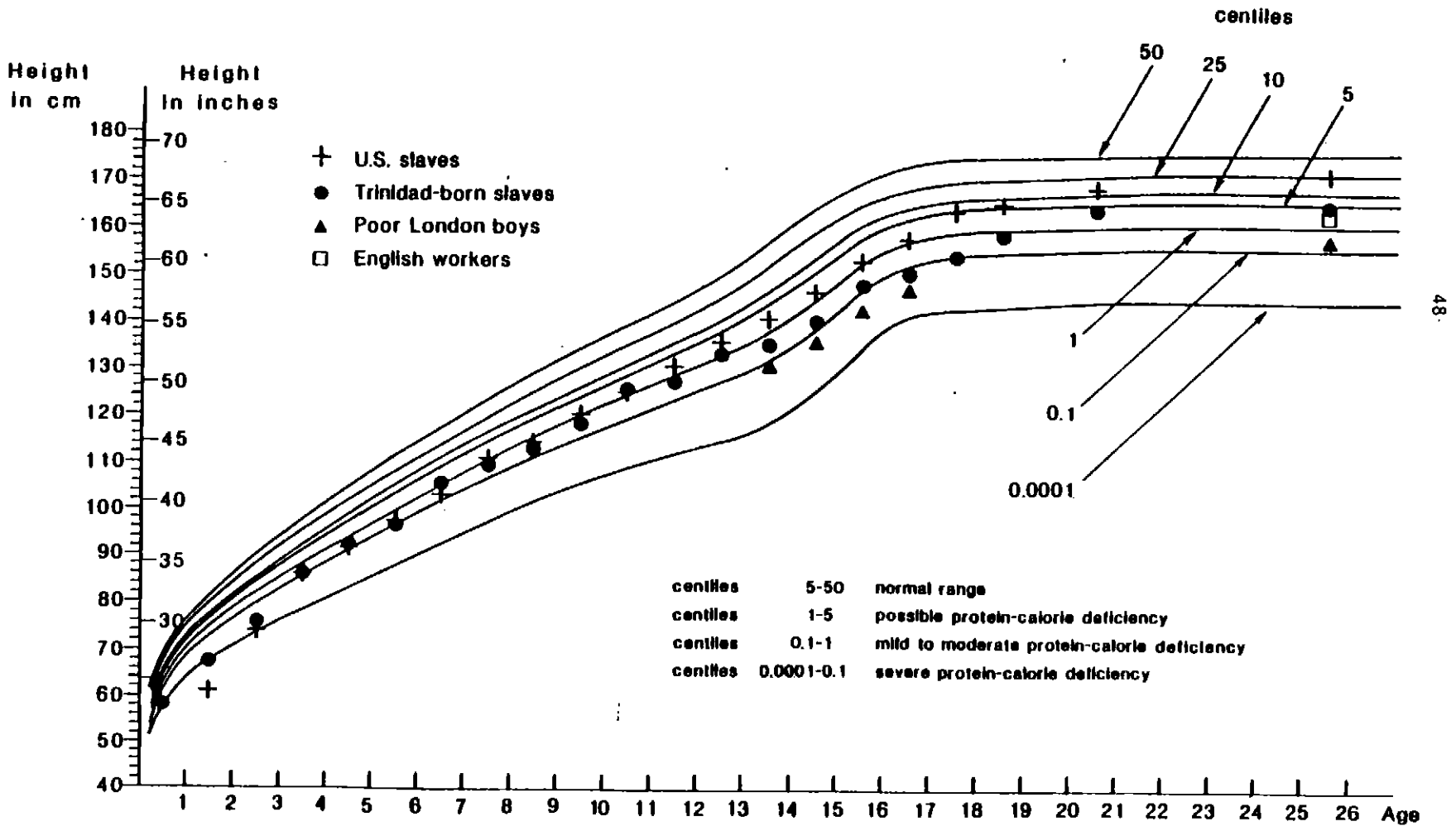
where

C = the crude death rate per thousand

I = the infant mortality rate per thousand

Figure 2

The Extent to Which Mean Heights of Male Slaves, Poor London Boys, and English Workers Deviated from the Modern Height Standard

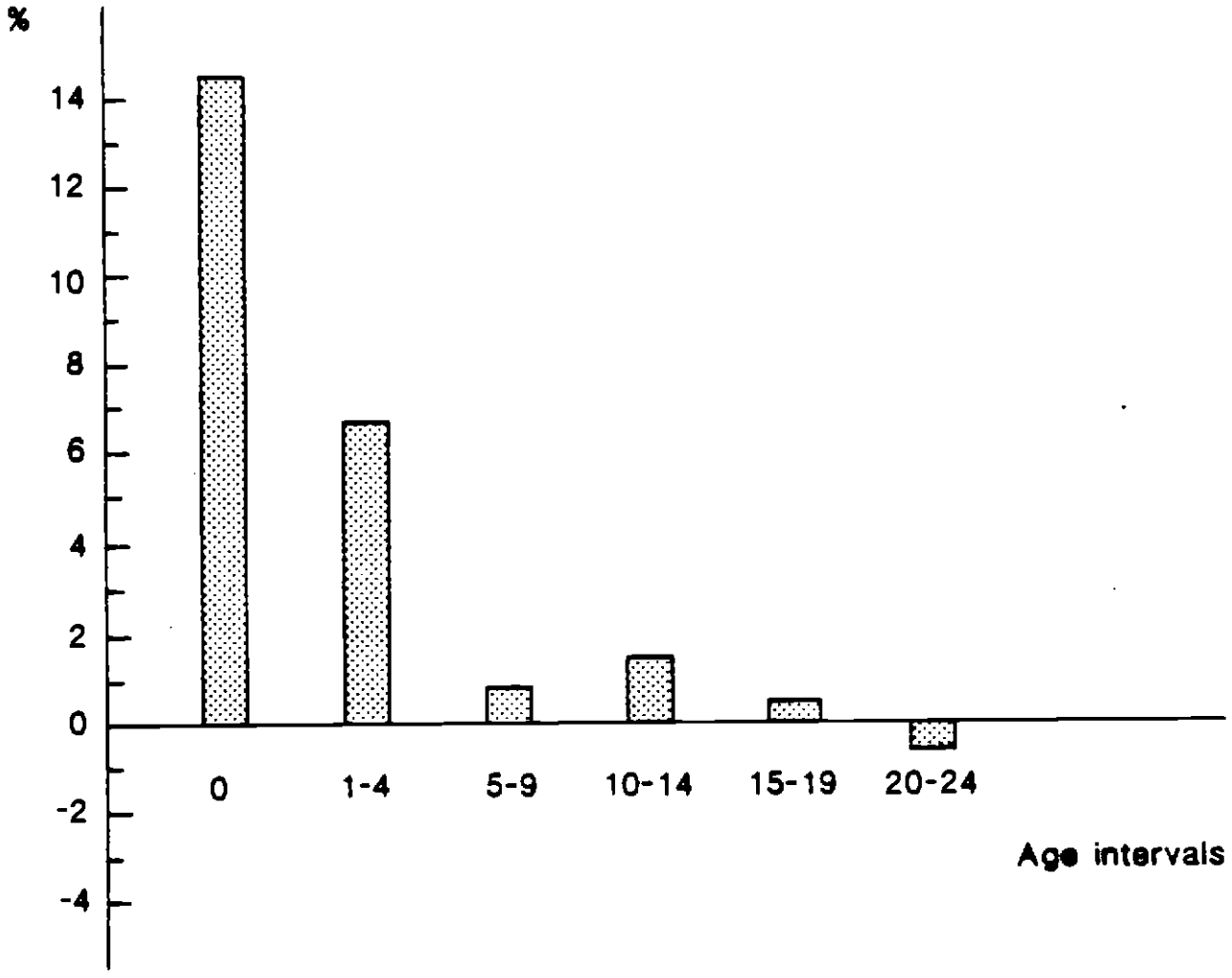


the heights of Trinidad-born male slaves, at ages from infancy to maturity, are superimposed on a set of curves which describe the current British standard for assessing the adequacy of physical development. The curve marked "50th centile" gives the average height at each age among generally well-nourished persons in Great Britain today. Also shown on the diagram are the heights of U.S. male slaves (which come from documents designed to prevent smuggling of slaves into the U.S.), 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 c.1800.

Figure 2 shows that during early childhood slaves in both Trinidad and the U.S. 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 centile. 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 U.S. and Trinidad slaves diverged, with the heights of U.S. 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, U.S. slaves were well into the normal range and Trinidad-born slaves were borderline normal. Thus, it appears that the diet that U.S. 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

Figure 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.

appears to have been inadequate to permit the same degree of catch-up, given the character of the physical environment.

Figure 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 U.S. slaves. This possibility is supported by available data on the death rates. Figure 3 indicates that it was excess death rates of slave children under 5 that accounted for the difference between the overall death rates of U.S. slaves and U.S. whites during the late antebellum era. Moreover, the fact that U.S. slaves and whites had similar life expectations after age 20 suggests that it was not the general virulence of the disease environment but conditions specific to young children. J. 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) 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 a 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 eight-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 U.S. the exceedingly small stature of slaves under 3 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 U.S. 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 U.S. slaves; the nutrient value of their diet would not permit it (Sheridan 1985). But in combination, the claims of work and disease and the disfunctions caused by alcohol appear to have left Trinidad slaves with a lower net nutrition to sustain an adolescent growth spurt than U.S. 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 one to two inches less than those of Trinidad-born slaves of the same ages. Nor was this gap made up during the late years of adolescence. Tanner (1982) has estimated that the height of the poor London boys at maturity was just 62 inches, about three 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 poor English boys between 1750 and 1800. When Tanner assessed this evidence (1981, p. 158) he said that the causes of such short stature, which persisted into adulthood without an acute retardation of the teenage growth spurt,

> have to be sought in early childhood and even the fetal period.... 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 he pointed out were the ingredients of popular patent medicines for children and which 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. When these "elixirs" and "cordials" were administered from birth they often led to a permanent stillness (Pinchbeck and Hewett 1969; Berridge and Edwards 1981).

Conditions of life were far more deleterious for the poor London boys than the generality of English workers. These boys, who represented the poorest of the English lower classes, were drawn largely from that fifth of English families that were unemployed or at best partially employed. They lived in the most crowded and virulent slums of London, many were homeless (Floud and Wachter 1982), and as Tanner has suggested, many of them were probably victims of toxic substances that were ingested while they were in utero or early in infancy. Evidence that the nutritional status of most English workers was superior to that of the poor London boys is also presented in Figure 2, which shows the mean final height of the pool of men from which the recruits into the Royal Marines were drawn c.1800. Although this pool included Londoners, the majority 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, which is just a shade below the final height of Trinidad slaves, but about 2.5 inches greater than the final height of the poor London boys.

3.3 Evidence From Regressions Between Height and Mortality

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$$(12.327)\ (-10.797)\quad (-3.294)\quad (-4.213)$$

$$\bar{R}^2 = 0.96;\ N = 64$$

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 U.S. dollars of 1970

T = time (year 1 = 1880)

^ = a hat over a variable indicates the natural logarithm of that variable

From these equations it can be seen that a one percent 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 percent of the decline in the infant mortality rate and per capita income accounted for another 27 percent, leaving only about 33 percent 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 one percent change in the Gini ratio (holding the level of income constant) had about four times as large an effect on mean heights as a one percent 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 expectation 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 very young children are more sensitive to nutritional status than the mortality rates of adolescents and adults.[19]

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.

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 non-infants. Because of the absence of data a more indirect approach is required for infants. This illustrative calculation will be applied to the British case.

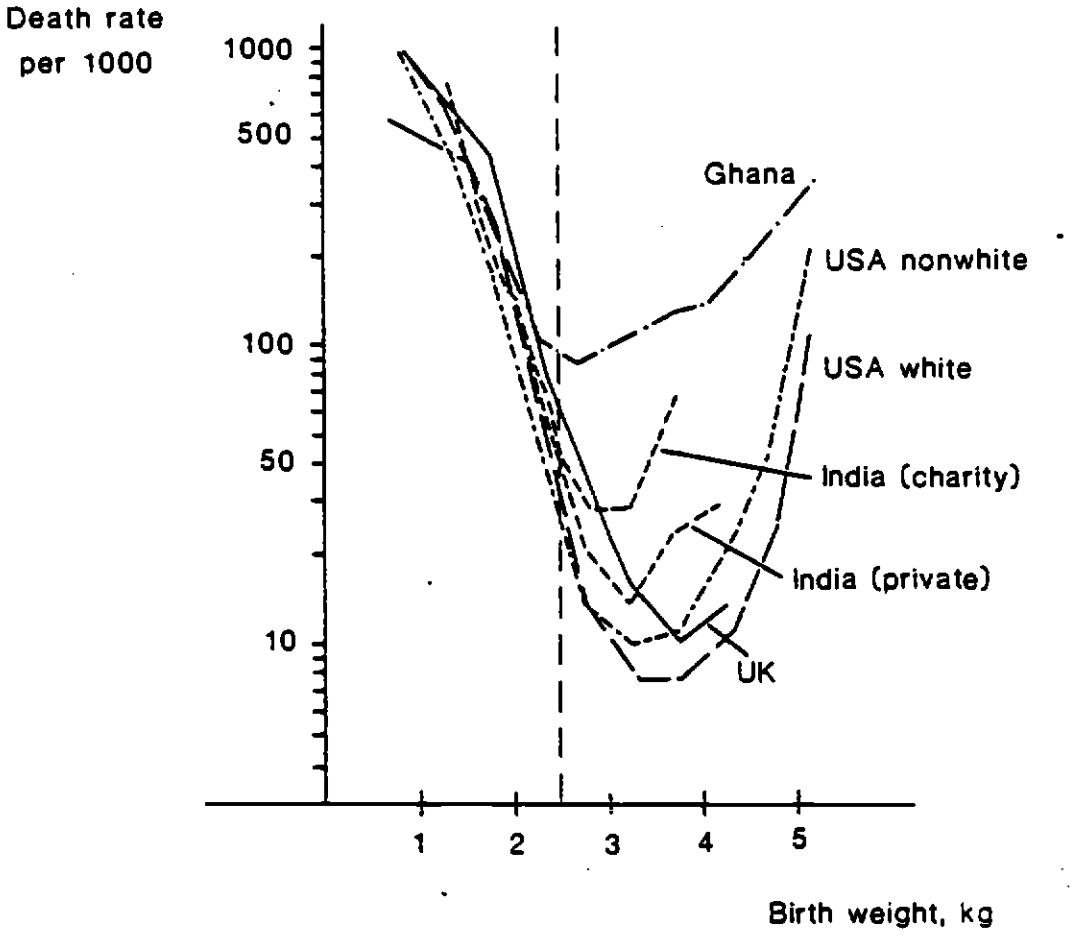
Let us begin by supposing that the nutrition of working class English males c.1800 had been improved to the point that they were able to achieve modern stature. Then their mean final height would have increased from 64.5 to 69.5 inches, which is an increase of 7.75 percent.[20] How much of an impact 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 percent increase in the final height of males would have reduced the non-infant death rate by about 11 percent (-1.4 x 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 neonatal death rates to birth weight.[21] The probability of dying at given birth weights is very high at weights below 2,501 grams (5.5 pounds). The schedule which relates the probability of dying to birthweight is stable below 2,501 grams. It varies little from one socioeconomic group to another within a nation or even across nations. This stability is evident in Figure 4.[22]

Mean birth weights vary greatly with the nutritional status of populations (Eveleth and Tanner 1976; WHO 1980). This point is illustrated

Figure 4

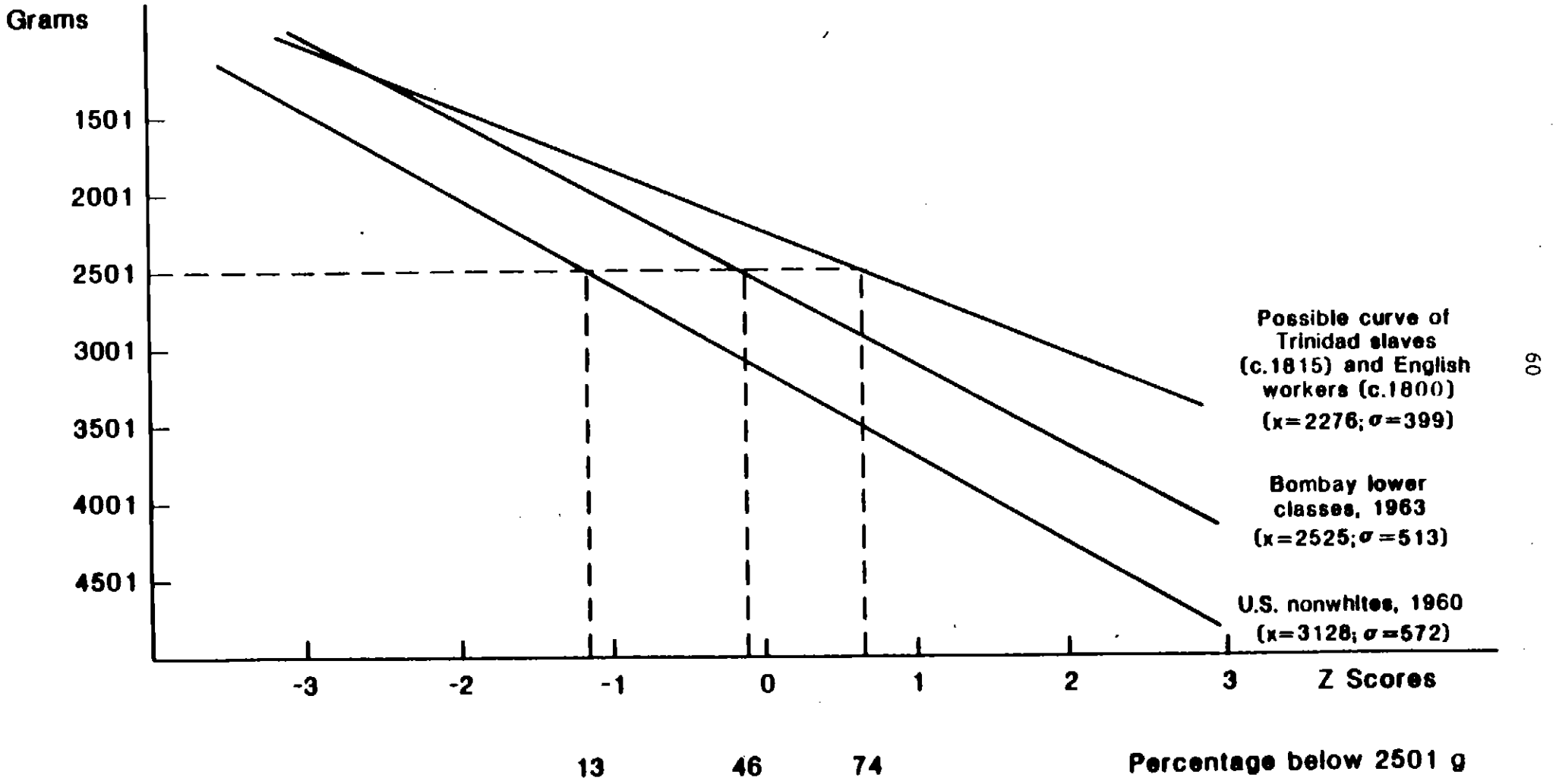
Perinatal Mortality by Birth Weight in Ghana, India, U.K., and U.S.A.



Source: Hytten and Leitch, Physiology of Human Pregnancy (2nd ed.) p. 324.

Figure 5

The Percentage of Male Births with Weights Below 2501 Grams in Two Modern Populations and the Possible Percentage Among Trinidad Slaves and English Workers During the Early Nineteenth Century



in Figure 5. The lines on this graph are normal approximations of the frequency distributions of birth weights.[23] Birth weight 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 deviation). Hence, the cumulative frequency distribution is represented by a straight line. The lowest line represents the distribution of U.S. non-whites in 1960. They had a mean birth weight of 3,128 grams and, as indicated by Figure 5, about 13 percent of the neonates weighed less than 2,501 grams at birth. The second line is the distribution of birth weights for lower class women in Bombay (Jayant 1964). Figure 5 indicates the mean birth weight in this population was just 2,525 grams. In this case nearly half (46 percent) 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 birth weights of the children of English workers c.1800.[24] In deriving this distribution I employed established correlations between height and birth weight 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 birth weights in this class c.1800 had a mean of 2,276 grams, which is about 249 grams (about a half pound) below the average in the deliveries of the lower class women in Bombay. It follows that about 79 percent of the births among English workers of c.1800 were at weights below 2,501 grams.[25]

The implication of this distribution of birth weights is revealed by Table 10. Column 2 represents the actual schedule of neonatal death rates by weight for non-white U.S. males in 1950 and Column 3 gives the actual

Table 10

THE EFFECT OF A SHIFT IN THE DISTRIBUTION OF BIRTH WEIGHTS ON THE NEONATAL DEATH RATE, HOLDING THE SCHEDULE OF DEATH RATES (BY WEIGHT) CONSTANT

(1) Weight (grams)	(2) Neonatal death rate of single nonwhite U.S. males in 1950 (per 1000)	(3) Distribution of birth weights of single nonwhite U.S. males in 1950 ($\bar{x}$=3,128 g.; σ=572 g.)	(4) Distribution of birth weights in a population with $\bar{x}$ = 2,276 g. σ = 399 g.
1500 or less	686.7	0.0117	0.1339
1501-2000	221.3	0.0136	0.2421
2001-2500	62.1	0.0505	0.3653
2501-3000	19.7	0.1811	0.2198
3001-3500	10.7	0.3510	0.0372
3501-4000	12.1	0.2599	0.0017
4001-4500	13.0	0.0865	———
4501 or more	23.2	0.0456	———
Implied neonatal death rate (per 1000)		26.8	173.0
Possible infant death rate (per 1000)		48.9	288.3

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

Sources: Columns 2 and 3: U.S. Nat. Off. Vital Stat., 1954; Column 4: See footnotes 21, 23, 24, and 26.

distribution of their birth weights. The product of 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 U.S. population had had the distribution of the birth weights of the English workers c.1800 which I have estimated, their neonatal death rate would have been 173.0 per thousand (see Column 3). The implication of Table 10 is that improvements in nutrition sufficient to have shifted the mean birth weight from 2,276 grams to 3,128 grams would have reduced the infant death rate by 83 percent [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 c.1800 and c.1980.[27]

(5) $\overset{*}{S} = \phi\overset{*}{I} + (1-\phi)\overset{*}{S}_n$

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

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

$\overset{*}{S}_n$ = 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 c.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 percent, equation (6) implies that improvements in nutritional status accounted for about 41 percent 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 non-infant deaths that may be attributed to nutrition account for just 12 percent 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 nutritonal 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]

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

Table 11

Nutritional Influence on Outcomes[a] of Infections

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

[a]Morbidity or mortality

Source: JIH, 1983

different diseases among different classes, and within different geographical areas, will obviously affect the impact of nutritional factors on fluctuations in mortality rates. Table 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), Hatcher (1977), Helleiner (1967), McNeill (1976), 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 it. 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. The fact 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 insure 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 percent of the increase in the life expectation between the cohorts of 1700-24 and of 1900-24 was due to the decline in deaths under age 10. Indeed, if the peerage had continued to suffer the ${}_{10}q_0$ value of 1700-24 in 1900-24, 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 1900-24 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, due 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 which bares both on 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; Dyer 1983; Heckscher 1954). 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 three 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 were 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 for 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 1720-1750 (Trevelyan 1942; Warner and Rosett 1975). One of the byproducts 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; Sussman 1977; Trumbach 1978; Stone 1977; 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.

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

Table 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			
	$\varepsilon = 1$	$\varepsilon = 0.5$	$\varepsilon = 0.25$	$\varepsilon = 0.1$
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

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 percent or more above trend) would imply shortfalls in grain yields that were only between 2 and 5 percent below the trend (see Table 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.

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 percent, 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 (Tilly 1983; Hufton 1983; Appleby 1979a; Post 1976; Flinn 1974). 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 1500), 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, Schofield, and Lee there were probably not more than seven or eight years (and there may have been as few as three) during

which the average food supply fell below its normal level by as much as 7 percent. 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.

$$(7) \quad \bar{\varepsilon}_i = (\theta_i - \beta_i)\psi_i - \varepsilon_i$$

where

$\bar{\varepsilon}_i$ = 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--i.e. because $\theta_i \geq \beta_i$) than landless laborers (for whom $\theta_i = 0$ and β_i is large).

Table 13 divides the English population at the middle of the Wrigley-Schofield-Lee period (c.1700) into four classes that correspond to the

Table 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 c.1700

Class of Household Head	1 Share in population	2 Normal share in consumption of the foodgrains ϕ_i	3 Share of grain in total consumption of a class β_i	4 Share of grain in income θ_i	5 Income elasticity ψ_i	6 Income-fixed price elasticity ε_i	7 Income-compensate price elasticity $\bar{\varepsilon}_i$
1. Landlords (including servants & retainers)	0.11	0.16	0.20	0.50	0.24	0.05	-0.02
2. Farmers & 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, & 3)	0.44	0.22	0.70	0.00	0.96	0.50	1.17

Source: Fogel 1985

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 13) includes not only the landlords and their immediate families but all of their retainers, high and low. Table 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 Everitt 1967; Kerridge 1968; Appleby 1979b; Dyer 1983; Shamas 1983; Shamas 1984; Lindert 1985; Drummond and Wilbraham 1958; Williams 1985). The values of ϕ_i shown in column 2 imply that landlords and yeomen consumed about 50 percent more grain per capita than the national average (much of it as ale and spirits), that shopkeepers and craftsmen consumed the national average, and that common laborers and paupers consumed about 50 percent 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 U.S. farmers c.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 1985).[37]

One important implication of Table 13 is that although laborers were about 44 percent of the population, they only accounted for 22 percent of the normal consumption of foodgrains and that landlords (who with their retainers and servants represented only 11 percent of the population) accounted for nearly as large a share of consumption. Another implication of Table 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 columns 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: as owners of surpluses the rise in grain prices increased their income, while as consumers it reduced their income. 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 elasticity to one-third of the income-fixed elasticity. In the case of laborers only the consumption effect was present. Although the income-fixed elasticity is already high, the income-compensated elasticity is more than twice as high.

The values set forth in Table 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):

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

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

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

Thus, the estimates of class elasticities in Table 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 $\bar{\varepsilon}_c$. If it were the population share that mattered, $\bar{\varepsilon}_c$ 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, $\bar{\varepsilon}_c$ 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 $\bar{\varepsilon}_c$ is the price elasticity of the aggregate foodgrain demand, it is not the price elasticity of aggregate demand, which is given by equation (10):

$$(10) \qquad \varepsilon_t = \delta\varepsilon_s + (1-\delta)\bar{\varepsilon}_c$$

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 percent 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:

(11) $\varepsilon_t = 0.25 \times 0.6 + 0.75 \times 0.32 = 0.39$

Equation (11) gives the "normal" value of ε_t--the value of ε_t when yields are close to the mean (say 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 14 shows that such declines could be triggered with surprisingly small shortfalls in output. Even a shortfall in output as small as 8 percent, 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 percent. It is worth noting that although output declines by 8 percent, aggregate foodgrain consumption only declines by 6 percent because grain reserved for feed and seed declines by twice as much (13 percent) as foodgrain consumption. As a result the feed and seed share of the reduced crop declines from 25 to 22 percent. With a supply that is 15 percent below the normal level, the interclass distribution of per capita consumption is so greatly exacerbated that the per capita grain consumption of laborers and paupers falls to less than 60 percent 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 ($\bar{\varepsilon}_4 = 1.17$) and the sharp rise in price (P goes to 1.65). It should be noted that more than

Table 14

The 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

	Normal share of each class in foodgrain crop $Q_d = Q_s = 1$ $P = 1$ $\varepsilon_t = 0.39$	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	Percentage decline of each class from normal per capita consumption of foodgrains (minus signifies rise in consumption)	Share of each class in reduced output of foodgrain at market-clearing price	Percentage decline of each class from normal per capita consumption of foodgrains (minus signifies rise in consumption)
	(1)	(2)	(3)	(4)	(5)
1. Landlords (including servants & retainers	0.16	0.172	-0.5	0.186	-1.0
2. Farmers & lesser landlords (including servants)	0.51	0.540	0.9	0.570	2.0
3. Shopkeepers, minor professionals, & craftsmen (including servants)	0.11	0.107	8.8	0.100	18.6
4. Laborers & the unemployed (not including servants listed under 1, 2, & 3)	0.22	0.181	23.2	0.145	44.4

Source: Fogel 1985

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 percent increase in prices instead of a 65 percent increase. In other words, one of the effects of the shifting distribution of entitlement is to reduce both $\bar{\varepsilon}_c$ 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 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 percent. Using 0.37 as an estimate of ε_t (the value implied by Table 14 for a 25 percent 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 percent of the years. With the deviations in quantity ("yields") normally distributed,[40] the preceding points imply equation (12):

$$(12) \qquad 1.20\sigma_g = 0.0792\bar{X}_g$$

where

σ_g = the standard deviation (SD) 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_t. This result is immediately apparent from equation (13):

$$(13) \qquad \frac{\overset{*}{Q}_g}{\overset{*}{P}_w} = \epsilon_{gw} = \frac{\sigma_g}{\sigma_w} r_{gw}$$

where

$\overset{*}{Q}_g$ = percentage deviations in the quantity of grain

$\overset{*}{P}_w$ = 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 SD of deviations in grain quantities

σ_w = the SD deviations in wheat prices

Hence

(14) $\quad \varepsilon_{gw} = 0.263 r_{gw}.$

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 (Shamas 1984; Dyer 1983; Oddy 1983; Shamas 1983; Skipp 1978; Richardson 1976; Kerridge 1968; Everitt 1967; Fussell 1949; 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 SD of deviations around the trend in the quantity of grain) is identical with σ_f (the SD 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 percent. Hence, instead of equation (12) we have equation (15):

(15) $\quad 1.95\sigma_f = 0.0792\bar{X}_f,$

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):

(16) $\quad \varepsilon_f = \frac{0.041}{0.251} r_{fw} = 0.163 r_{fw}$

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 percent above trend) would only imply about a 5 percent decline in the total supply of grains and (taking account of the shift away from feed and seed) only about a 4 percent 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 percent would imply a decline in per capita food consumption of about 9 percent--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 percent ($1.52\sigma_g$) was a relatively rare event,

occurring less than one year in fifteen. It is unlikely that England suffered more than one 10 percent 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 percent), the decline in the overall food supply was about 10.5 percent. All but seven of the other poor harvests imply grain shortfalls of less than 10 percent, foodgrain shortfalls of less than 8 percent, and overall food shortfalls of less than 7 percent.[44]

The information in column 3 of Table 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 percent, only the two lowest classes would have had non-negligible reductions in their normal supply of grain. In the case of the artisan class, much of the 8.8 percent 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, for example, to gruels and porridges) and by reducing storage losses and waste (Walford 1970; Walter 1976; Stern 1964; Mathias 1959; Fussell 1949; Drummond and Wilbraham 1958; Appleby 1978; McCloskey 1984). 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 and Williamson 1983b; Lindert 1980). 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 (Drummond and Wilbraham 1958; Stern 1964; Fussell 1949; Ashley 1928). Equations (14) and (16) together imply that about 40 percent 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 percent, the shortfall in the grain supply would have had to have been about 13 percent (0.08 ÷ 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 percent would have raised their mortality rate by 32 percent--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 percent above the average, the national cdr in a famine year would have risen by about 4.4 per thousand (0.44 x 1.4 x 0.232 x 0.028 x 1.1 = 0.0044), to a level which is about 16 percent 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 c.1400 or c.1500, famines in England were rare events, affecting a minority of the population, and accounting for a miniscule 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 percent of the accumulated total deaths (0.16 x 0.024 = 0.0038).[45] Given the exceedingly small proportion of deaths due to famines, the many factors (including non-famine 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 percent 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 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 pre-industrial era remains an open issue.

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 three 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; Margo and Steckel 1983; Fogel et al. 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 c.1760 was about 64.5

inches (which was about 9 centimeters below those of U.S. whites) and remained more or less at that level for the next half century. The succession of cohorts born between c.1810 and c.1840 appears to have experienced a fairly rapid upward shift in growth schedules, so that the c.1840 cohort was about three centimeters taller at maturity than the c.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 c.1840 counterparts (Sokoloff and Villaflor 1982; Floud and Wachter 1982; Floud and Wachter 1983; Floud 1983a; Floud 1983b).

There were also relatively constant growth curves for France between c.1820 and c.1900, and for Belgium between c.1830 and c.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 U.S. 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 U.S. 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 (A.J. 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 rising quite rapidly (Williamson 1976; David and Solar 1977), but heights and life expectation were decreasing? During an era in which from 50 to 75 percent 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 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 conditons 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 three 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 Figure 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 percent 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 U.S. near the turn of the twentieth century (Sawyer 1976; Lebergott 1976; Williamson and Lindert 1980).[51] Thus, the decline in final heights of native-born U.S. 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 (Williamson and Lindert 1980; Pessen 1973).

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.

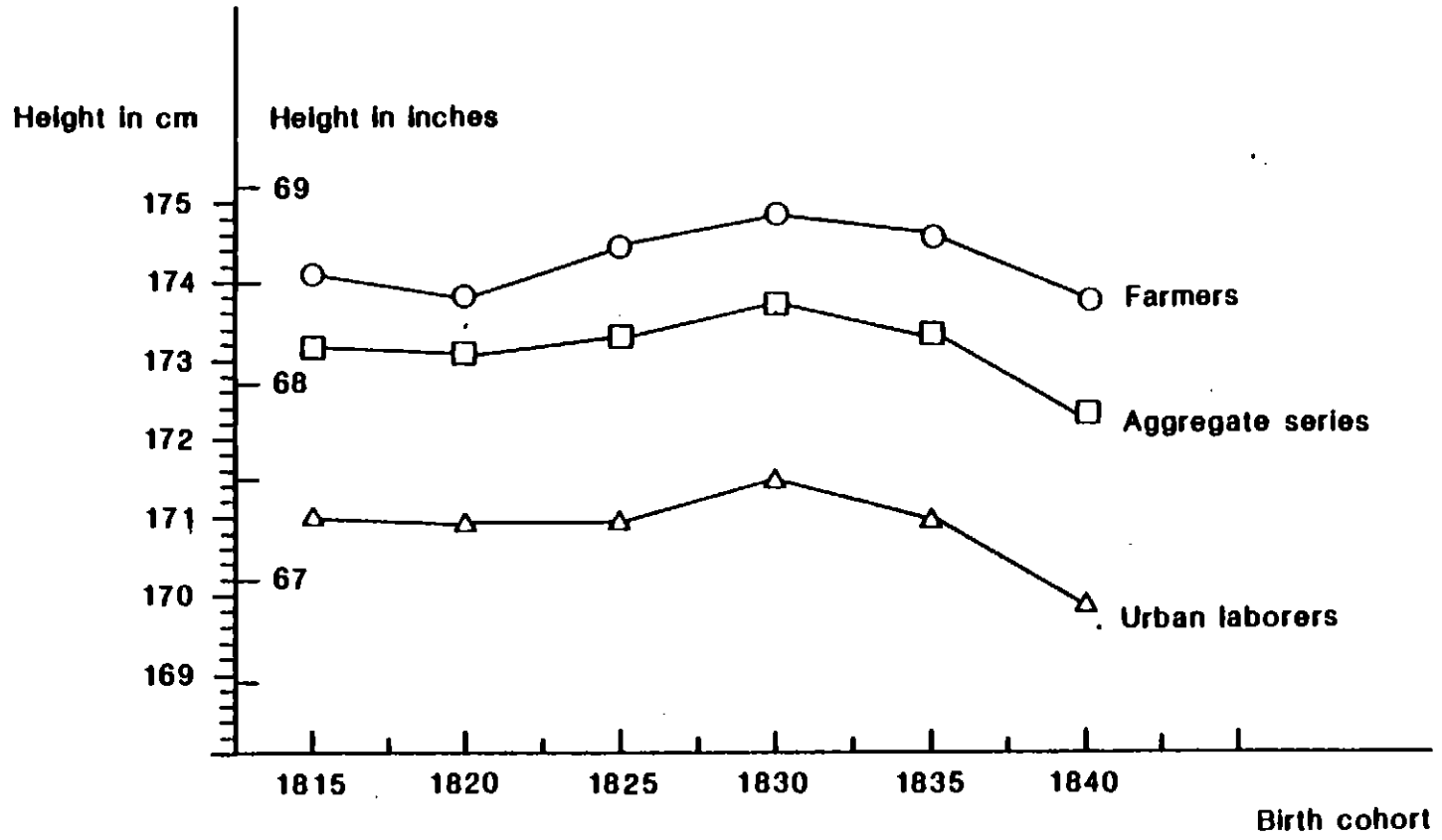
The first is drawn from the work on British sources. Data recently collected by Floud on the British upper classes (1984) when combined with his data on the lower classes make it possible to estimate how much of the improvement in the average nutritional status in Great Britain over the past century 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 c.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 (Sawyer 1976; Lindgren 1976; Brundtland et al. 1980). The means of adult height in these nations now exceed those of high-Gini-ratio nations, such as the U.S. and Great Britain, by several centimeters (U.S. Bur. Cen. 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 (Soltow 1971; R. Gallman 1969). 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 6 shows the beginning of the long downward trend in the aggregate series on native-born whites (cf. Figure 1). It also shows that when this series is disaggregated into occupational and residential groups, the series for farmers exhibits a rising trend until c.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

Figure 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.

1830 and then declines. These curves indicate that about 85 percent of the initial decline in the aggregate series was due to a decline in the mean heights of farmers and other rural 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 6 continued in subsequent decades. Native-born farmers who were born c.1860 were about 1.5 inches shorter than those who were born three 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 1 for cohorts born between c.1830 and c.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 that 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 percent 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 non-agricultural 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 1848-1850 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 not only able 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 (W. VA), Pittsburgh, and St. Louis (U.S. 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; Ency. Brit. 1961, 5, pp. 615-618). 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 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 percent 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; Ackerknecht 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; N.Y. State Bd. of Health 1867).

The increase in mortality between c.1790 and c.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, b, and 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 gage 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.

$$(18) \qquad w_n = (i + \delta_n)V_n$$

where

w_n = the wage rate (rental rate) at age $\underline{n}$ 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,

$$(19) \qquad \overset{*}{w}_n = \phi\overset{*}{i} + (1-\phi)\overset{*}{\delta}_n + \overset{*}{V}_n$$

where

$$\phi = \frac{i}{i + \delta_n}$$

$*$ = 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 in both "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 non-survivors at all the other ages between birth and entry into the labor force.

The estimates of $\overset{*}{\delta}$ and $\overset{*}{V}$, which can be derived from the decline in life expectation shown in Figure 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 (Commons et al. 1918, I; Benson 1969; Handlin 1979; Ernst 1949; Hoagland 1913; 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 expectations. 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 U.S., 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 U.S. (Lebergott 1984; Chiswick 1978 and 1979; 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 U.S. 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 non-agricultural sectors during the intervening years (Fogel 1986).

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 three 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 not only measuring 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 appear 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 other 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 are not only devoid of nutritional value but which 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 so 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 late 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; Matrossian 1984).

Preoccupation with diet, especially the excessive focus on adult diets, has diverted attention from an array of intrauterine occurances 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 (Moore 1983; Kielmann et al. 1983; Hurley 1980; 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, b; Bjerre 1975a, b; 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 birth weight not only increased 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, Shipley, and Rose 1984).

The preliminary results not only indicate that the factors contributing to the unanticipated cycles in heights and mortality were concentrated at particular ages but 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 (cf. Williamson 1981a; Williamson 1981b; Williamson 1982; Pollard 1981). 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 (Towne and Rasmussen 1960; Gallman 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 (Meeker 1976; Fogel and Engerman 1974; Atwater and Woods 1897; Frissell and Bevier 1899; U.S. Dept. Lab., 1897). A more subtle and possibly more pervasive effect on the living standards of laborers and their families, both in the cities and 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 hardships of working-class life during the nineteenth century. Nutritional and other health insults delivered in utero or early life not only appear to have affected adult health and longevity, but significantly reduced 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 U.S. 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 by Wrigley and Schofield (1983), Preston and van de Walle (1978),

Haines (1983), and Preston and Haines (1983) have already indicated, these sources will not only permit us 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 not only illuminate the past but will directly contribute to a better understanding of important issues in current economic and social policies.

6. Footnotes

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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, 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 benefitted 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, T. McKeown, 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. Srikantia, 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.

[1]Richard Hellie has called my attention to "the coincidence of the lowest life expectation [in Table 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 date and the death of an individual is known. Non-bloodline 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 percent.

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 percent of the families. In most of the other 23 percent 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 percent 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 percent of the individuals in the sample. In other words, so far we have been able to obtain economic information on about 75 percent 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 4,700,000 sheets submitted mainly before 1962 and contains information on about 25,000,000 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 1,000,000 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 4,000,000 sheets in this part of the collection. The DAE/CPE pilot sample of group sheets is drawn from the Main Section. Cf. Wimmer 1984.

[6]About 43 percent 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 percent 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" (Gt. Brit. Dept. Health Soc. Ser. 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; Sackett et al. 1977; Blaxter 1976; McKeown 1976b; 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 non-specific disorders--much of the range of diseases occurring among well-fed populations. Indeed, in one study anthropometric measures and the incidence of non-specific health disorders were positively and significantly correlated (Butler 1974; Condon-Paoloni et al. 1977; Briscoe 1979; Martorell et al. 1975; Beisel 1977; Cole and Perkin 1977; Martorell 1980; Mata et al. 1977; Frisancho 1978;

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 parts III and IV 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 Figure 1.

[11]See Appendix A for a discussion of the procedures employed in constructing the two time series displayed in Figure 1. Table 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 1 were extrapolated to age 10, using the model West tables of Coale and Demeny (1966).

[13]Levy also has a life table for 1650-1699 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 Figure 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 fifteen-year period (1800-1815), the second on a ten-year period (1815-1825), 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 c.1805. His series bottoms out in c.1865 and then rises. The DAE/CPE series bottoms out c.1857 and then rises. The decline of e_{10} in his series from c.1807 to c.1865 is 10 percent, which is the same decline as that shown in the DAE/CPE series between c.1807 and c.1857. However, between the peak of the DAE/CPE series (which occurred c.1792, about 15 years before Kunze's first observation) and its trough, the decline in about 16 percent.

[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:

$$\varepsilon_{m<15} = -2.00$$

$$\varepsilon_{f<15} = -1.54$$

$$\varepsilon_{m\geq15} = -2.50$$

$$\varepsilon_{f\geq15} = 0$$

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 1801-05 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). U.S. population weights for 1980 (U.S. Bur. Cen. 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 equation (3) yields:

$$(3.1) \qquad \overset{*}{I} = -15.9106\overset{*}{H} - 0.3889\overset{*}{Y} - 0.00837,$$

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 (in percent)
C^*	-1.05
I^*	-2.51
H^*	0.0610
Y^*	1.75

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

[19]The similarity between the results of equations (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 two or three 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 c.1800 must be considered tentative and is subject to revision. However, no currently plausible revision (the possibilities of change are pretty well bounded by ± 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 seven days after birth). In the U.S. and Great Britain during the early 1960s the distribution of infant deaths was approximately as follows (Shapiro et al. 1968):

days	percent	cumulative percentage
1	40	40
2-7	25	65
8-28	8	73
29-365	27	100

Late fetal deaths in Britain and the U.S. have recently been approximately equal to neonatal deaths (U.S. Bur. Cen. 1983, p. 77). For countries experiencing death rates in the range of 100 per thousand and over, neonatal deaths range between 35 and 70 percent (Mata 1978, Table 2.16; Ashworth 1982; Bouvier and Tak 1976).

Wrigley and Schofield (1981, p. 97) estimate neonatal deaths in England during the 17th and 18th centuries at about half of infant deaths. However, the practice of treating infant deaths during the first week or nine 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 Steckel 1985; Floud 1985a) 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 section 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 (Pharoah and Alberman 1981; Jones et al. 1979; Lancet 1980, p. 481).

[23]The distribution of birth weights 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 Figure 5, normal approximations to the distribution of the U.S. nonwhite and Bombay lower classes yield satisfactory results. The mean and standard deviation in the normal approximation to the U.S. distribution differs from those of the actual distribution by less than one percent (Chase 1969). In the Bombay case the difference in the means is less than one percent (the standard deviation of the sample was not reported) (Jayant 1964).

[24]In this case it was necessary to estimate the heaping of pre-term births on the left tail of the distribution of birth weights. My procedure was based on the proposition set forth in footnote 23 that the observed distribution of birth weights may be viewed as the result of a convolution of a small distribution of weights of pre-term 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 pre-term babies that have been heaped on the left tail.

In estimating column 4 of Table 10, which is graphed in Figure 5, I assumed that the underlying full-term distribution was N(2,300; 420). To this distribution I then added the estimated number of pre-term births at weights below 2,001 grams, using ratios obtained from Guha et al. (1973) which provides information on birth weight 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 1,501 and 1,501-2,000. This adjustment for pre-term 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 birth weight of the hypothetical distribution is quite low, it is consistent with the final height of English workers c.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 nine days after birth). In this connection, I would emphasize the likelihood that the mean birth weight 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, for example, the mean birth weight 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 footnote 25.

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

[25]My estimate of the mean birth weight 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 pre-maternity 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 birth weights at English pre-maternity homes shortly after the turn of the twentieth century revealed that the birth weight 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 ten days (Ashby 1915).

Since 45 percent of the increase in fetal weight normally takes place during the last eight weeks of pregnancy (Thomson, Billewicz, and Hytten 1968; Birkbeck 1976; Southgate 1978), birth weights 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 birth-weight distribution of Figure 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 percent higher than that of a sample of 43 populations with low mean birth weights (under 3,000 g.) 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 birth weight of about 420 grams between the late 1860s and the beginning of the twentieth century. The Wards are also collecting birth-weight 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 birth weights 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 and 4,000 grams, was about twice as high as in the U.S. 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 birth weights and to perinatal mortality rates.

[26]I have not distinguished birth weight 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. Beal 1980; Tanner et al. 1966; Eveleth and Tanner 1976; Hytten and Leitch 1971; 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 n.21, n.33, n.34, and in section 4.2 below).

The percentage reduction in the infant death rate due to a shift in the birth weight schedule is quite robust to the principal assumptions employed in Table 10. If, for example, I had used assumptions which yielded an infant death rate of 200 per thousand in 1800 (a more conventional figure than indicated in Table 10), the decline in the infant mortality rate due to the shift in the birth weight schedule would still be 76 percent [1-(48.9 ÷ 200.0) = 0.76].

[27]See Appendix B for the derivation of equation (5).

[28]Wrigley and Schofield (1981, p. 529) give the following values for 1801-05:

$$e_0 = 35.89$$

$$cdr = 27.08$$

$$cbr = 37.71$$

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 x 0.179 = 6.42 is the number of infant deaths per 1,000 persons in the total population. Consequently, infant deaths were 23.7 percent of all deaths (6.42 ÷ 27.08 = 0.237).

[29]The age-standardized death rate in Britain c.1980 was 8.32 per thousand, using the Wrigley and Schofield (1981, p. 529) age distribution for 1801-05 and the 1978-80 life table for the United Kingdom to estimate the ${}_nm_x$ values (G.B. Cent. Stat. Off. 1983, p.43). Since the crude death rate for 1801-05 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 1801-05 (0.763 x 27.08 = 20.66). Then 20.66 x 0.1085 = 2.24 is the reduction in non-infant mortality due to improved nutrition. The last figure is 12 percent 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 betweeen 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 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, for example, 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 five fold. 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 (Encyc. Brit. 1961, 2, p. 888; Ashbrook 1955; Webster 1845, pp. 747, 773-833, 1135; Davidson et al. 1979; Meneely and Battarbee 1976; U.S. Senate 1977).

[34]According to Dyer (1983, pp. 193-194) 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 seven 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 percent of absolute alcohol in ale and 11 percent in wine) and 9.1 ounces (if the percentage of absolute alcohol in ale is set at 8 percent). Even if peeresses drank only weak ale (4 percent 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 two or more ounces per day, about one out of five newborns suffered from symptoms of Fetal Alcohol Syndrome (Hanson, Streissguth, and Smith 1978; Abel 1982). The heavy salt intake of pregnant women further endangered fetuses by causing edema and by increasing the likelihood of kidney damage (U.S. Senate 1977).

[35]The balance of this section presents a highly condensed version of the analysis set forth in Fogel (1985). 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 equation (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 (1985) 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-to-seed ratio of eight implies that 12.5 percent 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 1985).

[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 and 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 percent (from 0.066 to 0.069). In this paper I have, therefore, employed the normal assumption in order to simplify the exposition. See Fogel (1985) 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 $\varepsilon_t = 0.37$ 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 SD 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 (G.B. House Com. 1899 and 1917; G.B. Ministry Agr. Fish. 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 1985).

If we now substitute 0.051 for σ_g in equation (12), that equation becomes $1.2 \times 0.051 = 0.061\bar{X}_g$. Hence, we can estimate ε_t from $\log 0.939 = \varepsilon_t \log 1.25$, 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 percent. 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.

[42]The SD of deviations of wheat prices from a 25-year moving average was computed from the price series of Bowden (1967 and 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 SD deviations in the total food supply (σ_f) was estimated from the "all-agricultural-products" price series developed by Bowden (1967 and 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 percent. When the Phelps Brown and Hopkins series (1956) of the price consumables (in which food has a weight of 80 percent) is used, σ_f is 0.043.

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

$$(16.1) \qquad \varepsilon_{fw} = 0.172 r_{fw}.$$

This equation also implies that there were only 8 years with food shortfalls in excess of 7 percent. In setting up equation (15), I assumed ε_t for all

food was the same as for all grain, which probably biases the estimate of σ_f upwards.

[45]The first figure in the parentheses is the proportionate increase in the national cdr due to a 23 percent decline in food consumption by the class of common laborers. The second figure is the proportion of years (1 + 41) in which such a decline occurred. For reasons explained in notes 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. 370-373) finds a sharp decline in fertility between three and nine 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 percent decrease in the food consumption of the class of common laborers requires (from equation 14 and the discussion on p. 86) a 70 percent increase in wheat prices and will lead to a 16 percent increase in the national cdr in such a year. Hence, the elasticity of the national cdr with respect to wheat prices is 0.16 ÷ 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 c. 1750), then the implied rate of increase in adult height between immigrants born c.1630 and native cohorts born c.1710 was about one centimeter per decade. There is no information currently available on the mean height of English cohorts born before c.1750. However, the high mortality rates in England between c.1625 and c.1790 (Wrigley and Schofield 1981, pp. 528-529) 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 c.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 c.1900 derived from the data in Figure 1. Alternative specifications of Steckel's regression, with dummies for high income countries raised the estimated Gini ratio for c.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:

$$(17.1) \qquad H = (1-\pi)H_u + \pi(H_u - D).$$

Hence

$$(17.2) \qquad H = H_u - \pi D.$$

Differentiating (17.2) totally yields:

$$(17.3) \qquad \overset{*}{H} = \psi\overset{*}{H}_u - (\psi-1)(\overset{*}{\pi} + \overset{*}{D})$$

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

$\psi = H_u + H$

$*$ = an asterisk over a variable indicates the rate of change in that variable.

In equation (17.3) both $\overset{*}{D}$ and $\overset{*}{\pi}$ 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 $\psi\overset{*}{H}_u + \overset{*}{H}$. The values of the variables needed to compute this ratio and the sources of the estimates are as follows:

$H^o = 64.7"$ (a weighted average of the mean height of the pool from which British soldiers aged 23-35 were recruited into the British armed forces c.1810 and of the implied final height of Sandhurst boys (c.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 6).

$H^1 = 68.9"$ (Rona et al. 1978, Table 3).

$H_u^o = 68.9"$ (The Sandhurst sample, and the ratio of the final height to heights at age 15 computed from the U.S. 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 percent per annum; the period of change was assumed to be 150 years):

$$\overset{*}{H} = 0.042$$

$$\overset{*}{H}_u = 0.010$$

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

and

$$\frac{\psi \overset{*}{H}_u}{\overset{*}{H}} = 0.25.$$

[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 percentage so impoverished would have been large enough by itself to explain the observed height decline, it could have been a non-trivial 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 1850-1855 was 68.1 inches, which is just a 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 U.S. 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 are 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 U.S. "real" per capita income appears to be about 0.71 percent (Engerman and Gallman 1983; cf. David 1967). The mortality increase between c.1790 and c.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 ℓ_x schedules of these tables during the prime working ages (taken to be ages 20-50) was 1.10 percent for level 10 and 0.58 percent for level 16. Hence, the average value of $\overset{*}{\delta}$ over the 70 years from c.1790 to c.1860 was 0.91 percent. 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 ${}_{20}\ell_o$, 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

$$\frac{V_{1860}}{V_{1790}} = \frac{1.45X_1 \int_0^{20} e^{(-\alpha_1 - \delta_1 + i_1)t} dt}{1.18X_o \int_0^{20} e^{(-\alpha_o - \delta_o + i_o)t} dt}$$

where

X = the net expenditures on childrearing at birth

α = the average annual rate of decline in X

It was assumed that $X_1 = X_o$, that $\alpha_1 = \alpha_o = 0.08$, and that $i_1 = i_o = 0.06$. The values of δ_1 and δ_o were computed from ${}_{20}\ell_o$ for mortality levels 10 and 16, respectively, and turned out to be 0.0170 for δ_1 and 0.0075 for δ_o. Consequently, $\overset{*}{V} = \left(\frac{20.4915}{18.1527}\right)^{\frac{1}{70}} - 1 = 0.0017$. The result is fairly robust to alternative plausible assumptions regarding the values of X, α, and i. It follows from equation (19) that the average annual rate of increase in "real" wages due to the rise in mortality is 0.28 percent (0.877 x 0 + 0.123 x 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

$$(19.1) \qquad \overset{*}{w} = \overset{*}{\beta} + \overset{*}{Y} - \overset{*}{L} = \overset{*}{\beta} + \overset{*}{\bar{Y}} - \overset{*}{\rho}$$

or

$$(19.2) \qquad \overset{*}{\bar{Y}} = \overset{*}{w} - \overset{*}{\beta} + \overset{*}{\rho}$$

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 $\overset{*}{\beta}$ and $\overset{*}{\rho}$ 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 $\overset{*}{\bar{Y}}$ will be equal to $\overset{*}{w}$. It follows that the mortality adjustment reduces the rate of growth in "real" per capita income by about 39 percent (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 three 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, and 1985).

[62]The fact 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 water-born 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.

7. Appendix A

On the Construction of Figure 1

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

The Height Series

The entries from 1710-1875 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 five-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 five-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 (1760-1775, 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 closed by

Table A.1

DATA FOR FIGURE 1

e^{o}_{10}				Height			
Years on which observation is centered	Entry	Years on which observation is centered	Entry	Year on which observation is centered	Entry (cm)	Year on which observation is centered	Entry (cm)
1720-24	51.8	1855-59	47.8	1710	171.5	1825	173.1
1725-29	52.7	1860-64	49.2	1715	172.2	1830	173.5
1730-34	52.0	1865-69	51.4	1720	171.8	1835	173.1
1735-39	51.2	1870-74		1725	172.1	1840	172.2
1740-44	52.9	1875-79		1730	172.1	1845	171.6
1745-49	52.3	1880-84		1735	171.7	1850	171.1
1750-54	52.5	1885-89		1740	172.1	1855	170.8
1755-59	52.9	1890-94		1745	172.0	1860	170.6
1760-64	53.9	1901	50.6	1750	172.2	1865	171.1
1765-69	53.7	1910	51.3	1755	172.1	1870	171.2
1770-74	54.8	1920	54.1	1760		1875	170.7
1775-79	55.2	1930	55.0	1765			
1780-84	56.4	1940	57.0	1770		1882.5	168.9
1785-89	56.5	1950	59.0	1775		1887.5	169.2
1790-94	56.7	1960	59.6	1780	173.2	1892.5	169.0
1795-99	55.4	1970	59.8	1785	173.2	1897.5	170.0
1800-04	55.2			1790	172.9	1902.5	170.0
1805-09	53.0			1795	172.8		
1810-14	52.3			1800		1906.5	171.6
1815-19	51.9			1805		1911	172.2
1820-24	51.4			1810		1916	172.9
1825-29	51.1			1815	173.0	1921	173.2
1830-34	51.0			1820	172.9	1931	175.5
1835-39	50.2						
1840-44	48.7						
1845-49	48.2						
1850-54	47.9						

linear interpolation. The third gap was closed by interpolation on the trend in the height of native-born recruits onto 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 five-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 A.1 reflects mortality experience over a 25-year period centered on the mid-year of a given interval.

Time series of e_o 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 1901-1970 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 ten states in 1900 (with 26 percent of the national population) to the entire nation in 1933 (U.S. Bureau of the Census 1975, p. 44). The

observations for 1901, 1910, 1920, 1930, 1940, and 1950 are each for three years centered on the designated year (U.S. Nat. Off. Vital Stat. 1963). The observations for 1960 and 1970 are each based on data for a single year (U.S. Nat. Cent. Health Stat. 1963; U.S. Nat. Cent. Health Stat. 1974).

8. Appendix B

The Derivation of Equation (5)

Equation (5) may be derived from the identity:

$$(5.1) \qquad S = \frac{D_i}{B} \cdot \frac{B}{P} + \frac{D_n}{P_n} \cdot \frac{P_n}{P}$$

which may be rewritten as

$$(5.2) \qquad S = I\alpha + S_n\beta.$$

Total differention of (5.2) yields

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

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

$$(5.4) \qquad \overset{*}{S} = \phi\,(\overset{*}{I} + \overset{*}{\alpha}) + (1-\phi)\,(\overset{*}{S}_n + \overset{*}{\beta}).$$

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 birth rate (B/P)

β = the proportion of non-infants in the population (P_n/P)

$*$ = an asterisk over a variable indicates the rate of change in that variable

$\phi = \frac{D_i}{D_i + D_n}$ = the share of infant deaths in total deaths

It follows that if the birth rate 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 $\overset{*}{\alpha}=\overset{*}{\beta} = 0$.

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):

$$(5.5) \qquad S = \frac{P_0}{\sum_0 P_i} M_0 + \frac{P_1}{\sum_0 P_i} M_1 + \ldots + \frac{P_z}{\sum_0 P_i} M_z$$

where

P_i = the number of individuals at age i

$\sum_0 P_i$ = the total population

M_i = the age-specific death rates

Here, the $P_i/\sum_0 P_i$ are fixed from one time period to another; in other words, only the M_i change so that $S_o = S_i$ is due only to temporal differences in the M_i. If we let $\alpha_i = (P_i/\sum_0 P_i)$, equation (5.5) becomes:

$$(5.6) \qquad S = \alpha_o M_o + \alpha_1 M_1 + \ldots + \alpha_z M_z.$$

Total differentation of equation (5.6) yields:

$$(5.7) \qquad \overset{*}{S} = \frac{D_o}{\sum_0 D_i}(\overset{*}{M}_o + \overset{*}{\alpha}_o) + \frac{D_1}{\sum_0 D_i}(\overset{*}{M}_1 + \overset{*}{\alpha}_1) + \ldots + \frac{D_z}{\sum_0 D_i}(\overset{*}{M}_z + \overset{*}{\alpha}_z).$$

However, since by definition $\overset{*}{\alpha}_i = o$, equation (5.7) reduces to

$$(5.8) \qquad \overset{*}{S} = \frac{D_o}{\sum_0 D_i}\overset{*}{M}_o + \frac{D_1}{\sum_0 D_i}\overset{*}{M}_1 + \ldots + \frac{D_z}{\sum_0 D_i}\overset{*}{M}_z = \frac{\sum_0 D_i \overset{*}{M}_i}{\sum_0 D_i}.$$

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

$$(5.9) \qquad \frac{\sum_1^z D_i \overset{*}{M}_i}{\sum_0^z D_i} = \overset{*}{M}_x.$$

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

$$(5.10) \qquad \overset{*}{S}_n = \frac{\sum_1 D_i \overset{*}{M}_i}{\sum_1 D_i}.$$

If we let $\sum_1 D_i = D_n$ and $\sum_0 D_i = D_o + D_n$, then it follows from equation (5.9) and (5.10) that:

$$(5.11) \qquad (D_o + D_n)\overset{*}{M}_x = D_n \overset{*}{S}_n, \text{ or } \frac{D_n}{D_o + D_n}\overset{*}{S}_n = \overset{*}{M}_x.$$

Substituting equation (5.11) into equation (5.8) yields

$$(5.12) \quad \overset{*}{S} = \frac{D_o}{D_o + D_n} \overset{*}{M}_o + \frac{D_n}{D_o + D_n} \overset{*}{S}_n .$$

Then letting $\phi = \frac{D_o}{D_o + D_n}$ and $\overset{*}{I} = \overset{*}{M}_o$, yields

$$(5.13) \quad \overset{*}{S} = \phi \overset{*}{I} + (1 - \phi)\overset{*}{S}_n,$$

which is the same as equation (5).

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

$$(5.14) \quad S_1 = I_1 \alpha_1 + S_{n1} \beta_1,$$

$$(5.15) \quad S_o = I_o \alpha_o + S_{no} \beta_o,$$

$$(5.16) \quad \alpha_1 = \alpha_o + \Delta\alpha$$

$$(5.17) \quad S_1 = S_o + \Delta S$$

$$(5.18) \quad S_{n1} = S_{no} + \Delta S_n$$

$$(5.19) \quad \beta_1 = \beta_o + \Delta\beta$$

Subtracting equation (5.15) from (5.14) and making the substitutions indicated in equations (5.16) - (5.19) yields:

$$(5.20) \quad \Delta S = \alpha_o \Delta I + I_o \Delta\alpha + \Delta I \Delta\alpha + \beta_o \Delta S_n + S_{no} \Delta\beta + \Delta S_n \Delta\beta$$

Then, dividing both sides of (5.20) by S_o and multiplying the first right-hand term by I_o/I_o, the second by α_o/α_o, etc., yields

$$(5.21) \quad \overset{*}{S} = \phi(\overset{*}{I} + \overset{*}{\alpha} + \overset{*}{I}\cdot\overset{*}{\alpha}) + (1-\phi)(\overset{*}{S}_n + \overset{*}{\beta} + \overset{*}{S}_n\cdot\overset{*}{\beta}),$$

where

$$\phi = \frac{I_o \alpha_o}{S_o} = \frac{D_{io}}{D_{io} + D_{no}}$$

Equation (5.21) is the discrete analogue to equation (5.4), and these two equations differ only by the two interaction terms $\overset{*}{I}\cdot\overset{*}{\alpha}$ and $\overset{*}{S}_n\cdot\overset{*}{\beta}$. Since

by nature of an age-standardized death rate $\overset{*}{\alpha}$ and $\overset{*}{\beta}$ are equal to zero, equation (5.21) also reduces to equation (5).

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