NBER WORKING PAPER SERIES

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

Robert W. Fogel

Working Paper No. 1402

NATIONAL BUREAU OF ECONOMIC RESEARCH
1050 Massachusetts Avenue
Cambridge, MA 02138
July 1984

This paper is a progress report on two projects jointly sponsored by the NBER's program in the Devleopment of the American Economy and by the Center for Population Economics of the University of Chicago. The findings presented in this paper are tentative and subject to change. The research reported here is part of the NBER's research programs in Development of the American Economy and Health Economics. Any opinions expressed are those of the author and not those of the National Bureau of Economic Research or any of the other cooperating institutions or funding agencies. See pp. 87-88 for the acknowledgements.

NBER Working Paper #1402
July 1984

Nutrition and the Decline in Mortality Since 1700: Some Preliminary Findings

ABSTRACT

This paper uses the data in the NBER/CPE pilot sample of genealogies to create a new time series on life expectation in the U.S. since 1720. After attaining remarkably high levels toward the end of the eighteenth century, life expectation as measured by e^{o}_{10} began a decline that lasted about 80 years before beginning the new rise with which we have long been familiar. Second, time series on the average adult stature of national populations in North America and Europe are used as a measure of nutritional status. The properties of this measure in the analysis of labor welfare and an explanation for the high correlation between stature and the Gini ratio are discussed. The time series on stature is strongly correlated with the series on e^{o}_{10} and other measures of mortality. Third, these correlations are used to estimate the contribution of improvements in nutritional status (not diet alone but diet net of prior claims) to the decline in mortality in Europe and America since 1800. Improvements in nutritional status may have accounted for as much as four tenths of the decline in mortality rates, but nearly all of this effect was concentrated in the reduction of infant mortality. The new findings are used to resolve several paradoxes and the implication of the findings for the standard-of-living controversy are considered.

Robert W. Fogel
Center for Population Economics
University of Chicago
1101 East 58th Street
Chicago, Il 60637
(312) 962-7709

Contents of

Nutrition and the Decline in Mortality Since 1700: Some Preliminary Findings

List of Tables

List of Figures

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.[1]

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. A pediatrician, McKeown gained prominence for his studies of the relationship between birth weight and perinatal mortality rates in Birmingham after World War II (McKeown and Gibson, 1950, 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 (1976) 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 through a residual argument in which he rejects the other principal explanations. The alternatives to nutrition are advances in medical

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. 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.

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 1976, 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, 1976, 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, 1975, 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 may be 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 (1975, 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 and 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. This judgment has recently been reinforced by Easterlin who, primarily on the basis of the trend in per capita income, estimated that U.S. life expectation increased by 6.1 years between 1800 and 1840 and by 5.8 years between 1840 and 1880 (Easterlin, 1977, p. 138). 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 eighteenth century and the beginning of the second quarter of the nineteenth century, the mortality rates of the aristocracy were about as high as those of the general population. Both the high mortality rates of the nobility before 1725 and the rapid fall in these rates thereafter, although there was no apparent change in the diet of the peerage, predisposed Razzell "to look at the food supply hypothesis very critically."

Table 2

Cohort Life Expection (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.

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 discontent 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 causal 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 indicates that factors which McKeown diminished had a substantial impact 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 system 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 varies 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.[1] 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.

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 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, 1983; 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 micro-economic 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 quite 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).

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

Table 3

Tentative Estimates of the Temporal Distribution of Observations in 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
1851 or after	144,000	77	608,000	65
1801-50	34,000	18	246,000	26
1751-1800	6,000	3	51,000	5
1700-1750	3,000	2	26,000	3
Before 1700	1,000	1	8,000	1
Totals	188,000	100	939,000	100

Sources: 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.

a sample of the family histories into machine-readable form. At the present time the sample of published family histories consists of about 60,000 individuals drawn from about 270 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.[2]

Of the 72,000 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.[3]

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.[4] 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.[4]

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

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

(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 age 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 portion of the X_{ij} variables that we intend to consider have been brought into analysis.. Still missing are the main economic variables, which we are now in the process of linking to the demographic variables.

Nevertheless, the initial runs on age-specific risk of death (for each sex, on each of the seven age intervals, for each of four birth cohorts) are rather promising. Birth order is statistically significant and has a relatively large impact on the probability of dying in most of the age intervals, with first and last births having a higher probability of dying than intermediate births in families with at least four live births. Place of birth has a significant impact on the probability of dying and the high risk regions change over time.

The bias variables (B_{ij}) indicate that practices by the compilers of genealogies had a small but statistically significant effect on the measured level of risk. So far these biases do not appear to have had much effect on the coefficients of the X_{ij}, generally changing the values only of the second or third significant digit. Much remains to be done, however, on investigating alternative ways in which the bias variables may be introduced into the regressions. But so far the impact of the various biases identified on <u>a priori</u> ground appear 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

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

shows that although the means in the sample of Adams and Kasakoff are biased upward as one would expect, the differences are not very large and clearly indicate the genealogies cover virtually the whole range of wealth holders. 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 partriarch 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 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.

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 national population to which the final sample will pertain. Although the individuals at risk during the 1850s come from all of the major regions, the Northeast is overrepresented and the South and Midwest are underrepresented. The rural areas are also overrepresented and urban areas are underrepresented. In principle the deficiencies in the sample could be remedied by reweighting each of the cells in an appropriate manner. I have not engaged in such an exercise for two reasons. First, the current sample is too small; on average there are only about 13 observations for each of the 70 cells that need to be reweighted. Second, the life tables which are available for comparison suffer from similar 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 the ten original death

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 Native-born whites, 1850-1860, derived from the genealogical sample			B All whites, 1850-1860, average of Haines's tables for 1850 and 1860			C 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.

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 populatons 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 anthropometic measures as measures of nutrition rests on a well-defined patterns 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. 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, 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). 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 final 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" (Mosley and Chen, 1983).[5] 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 problem? 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, 1974; Meredith, 1970; Naeye, 1981; Raman, 1981; Thomson and Bellewicz, 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 characterizations of the empirical relationships we seek to uncover.

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. 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 depravation 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.I.H., 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 these lacunae in growth have no affect 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 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 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

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 & Steckel (1983)
2. Union army, blacks	5,000	10,000	Same as 1, plus complexion,	Margo & 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	Fishman & Walker (1984)
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 & Steckel (1982)

(continued)

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 & birthlenght, 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 & Margo (1984)
10. Cost of living surveys, 1934-1937	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 socioeconomic variables for all ages and both sexes	Friedman (1982)
		British Samples (12-14)		
12. Marine Society, boys, 1760-1870		50,000	Height, age, and various socioeconomic characteristics including occupations of fathers and sons, literacy and vaccination (or innoculation); ages mainly 13-17	Floud & Wachter (1982)

(Continued)

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, 1808-1893	11,000	11,000	Height by age, date of recruitment, fees paid	
14. Military recruitment records, 1750-1910	130,000	130,000	Same as 11, except for father's occupation; mainly for males aged 16-45	Floud (1983a)
Other European Samples (15-16)				
15. Swedish conscript rolls, 1750-1910	30,000	30,000	Height by age, years of service, and various 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)

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 late 1983, information had been collected on about 400,000 individuals, which is about 75 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 findings which have arisen out of these data sets which are highly suggestive, and which appear to be sufficiently robust to warrant their presentation.

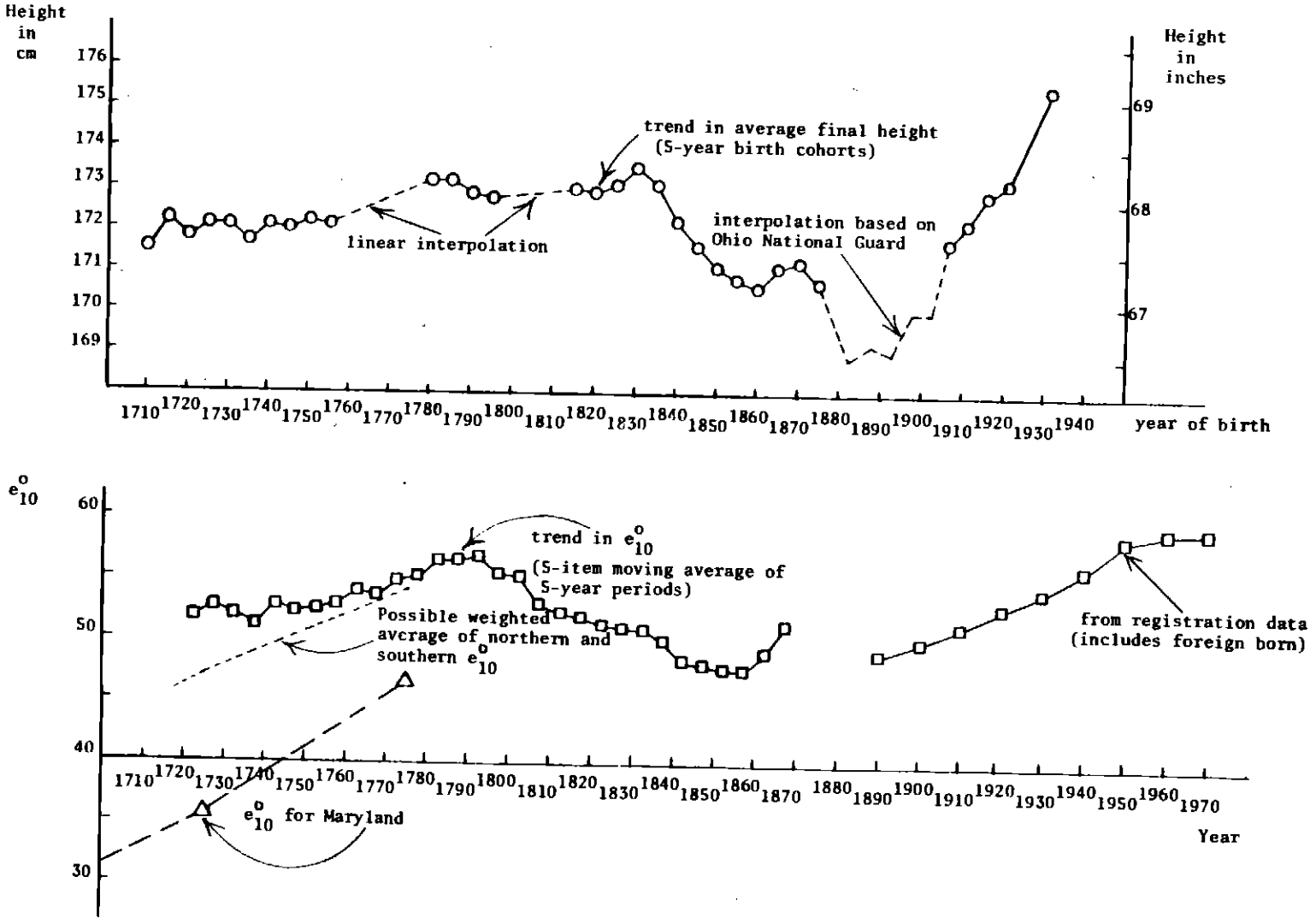
3.1 The Secular Trends in the Height and in the Life Expectation of U.S. White Males, 1700-1930

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.[6] Second, southerners are underrepresented in both the height and 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.[7]

It is possible to estimate tentatively the effect of the 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 1700-49 and 1750-99 are shown in the lower portion of the diagram.[8] 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.[9] 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 1750. The beginning of the peak may be shifted by one or two decades and the level of the peak may be lowered slightly.

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^o_{10})
(height by birth cohort; e^o_{10} by period)



These corrections will not change the striking 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. 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.

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, 1985).

Figure 1 and Table 7 reveal that Americans not only achieved modern heights by the middle of the seventeenth 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

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}			
19th	I		(males)	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: Hollingworth 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 notes to Table 3 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 during the first quarter of the twentieth century.

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 and Poor London Boys

The second piece of evidence linking mortality and nutritional status comes from data on slaves and on the London poor. 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 disposition of all the slaves who were registered in 1813. Friedman (1982) was the first to investigate the differences between the height of the slaves who died and those who survived. The difference is evident in Table 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.

Table 8

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

Age	Survivors Height	SD	N	Non-Survivors Height	SD	N
			Males			
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 deviations of non-survivors were misreported in the original source. Those shown here, supplied by Friedman, are the correct ones.

Table 9

Height Regression, Slaves Under Age 26, Trinidad-Born

Dependent Variable: Slave's Height.

	Males			Females		
Variable	Mean	Coefficient	T-ratio	Mean	Coefficient	T-ratio
Intercept		9.2	4.64		15.8	7.77
Age	6.7374	5.0927	27.05	6.8528	4.9525	26.05
Age Squared	77.4127	-0.4148	-11.71	81.0743	-0.3533	-9.93
Age Cubed	1155.665	0.0227	9.50	1243.841	0.0177	7.47
Age to the fourth	20048.90	-0.0005	-8.90	22013.99	-0.0004	-7.13
Number of Slaves on the Unit	75.6142	0.0054	0.95	76.8831	0.0006	0.10
Number of Slaves Squared	9109.826	0.000016	0.63	9349.246	0.000034	1.34
Sugar Unit	0.6975	-0.1437	-0.42	0.6722	0.1958	0.56
Sugar times Number of Slaves	63.9059	-0.0032	-0.78	62.8478	-0.0031	-0.80
Cotton Unit	0.04796	-0.0918	-0.22	0.0577	0.4115	1.02
Light Child of Dark Mother	0.0496	0.9285	2.41	0.0432	-0.5840	-1.38
Creole Mother	0.2436	2.9238	0.74	0.2803	-8.5058	-2.33
Creole Mother Times Her Height	15.0205	-0.0561	-0.87	0.2803	-8.5058	-2.33
Crude Death Rate on Unit	0.0399	-4.5974	-1.85	0.0373	-3.1587	-1.12
Mother's Height	60.8135	0.1983	6.13	60.7987	0.0830	2.49
Died 1813 to 1815	0.0652	-1.1687	-3.33	0.0812	-0.8538	-2.61
Mean of Dependent Variable	40.3			40.4		
R-squared			0.92			0.91
F-ratio			1358.35			1296.09
Degrees of Freedom			1749			1857

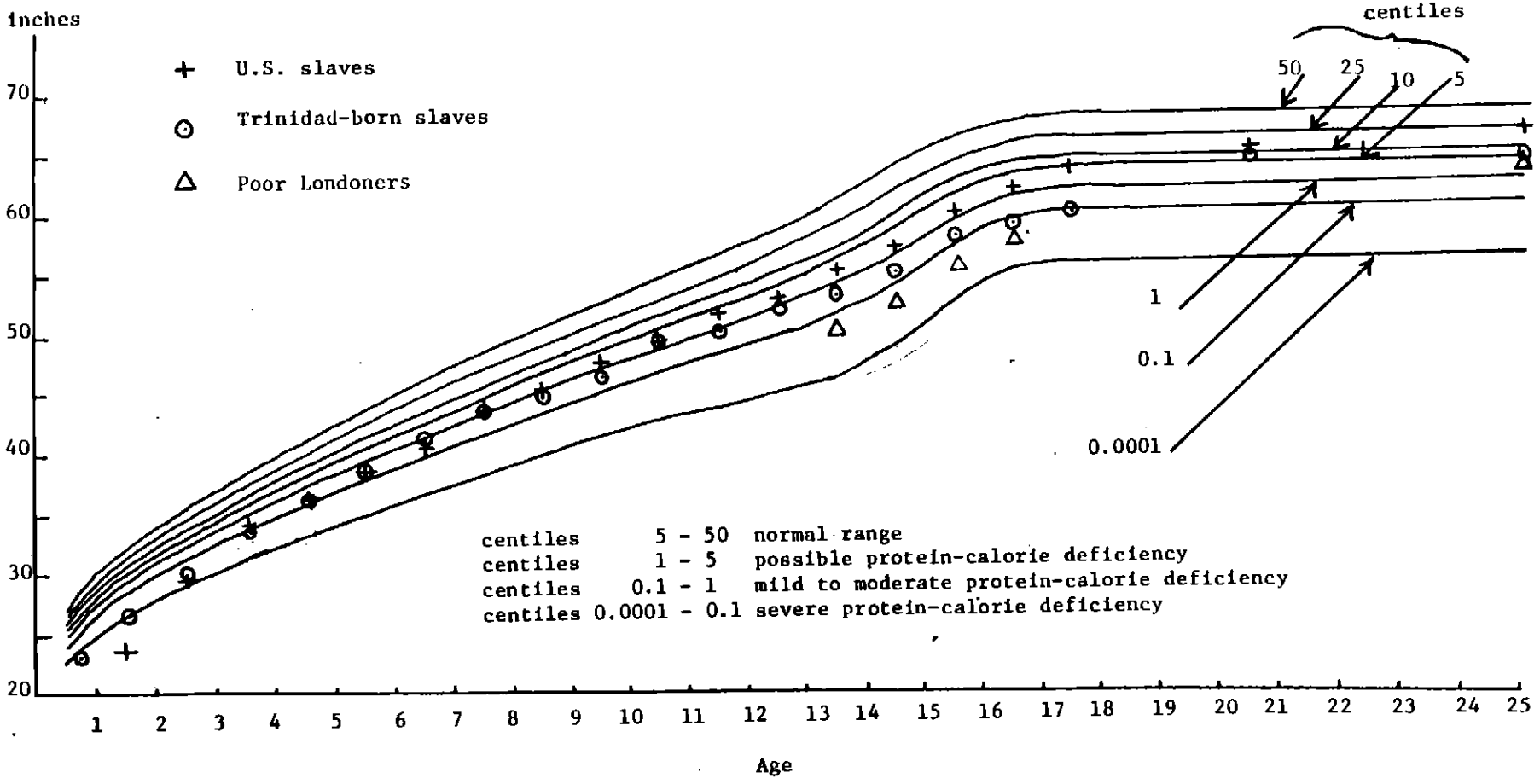
Source: Friedman, 1982

Figure 2 indicates how bad their nutritional status was. In this diagram 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-fed 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 the 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 English laborers at maturity c. 1780.

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

Figure 2

The Extent to Which Mean Height of Male Slaves and Poor Londoners
Deviated From The Modern Height Standard



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

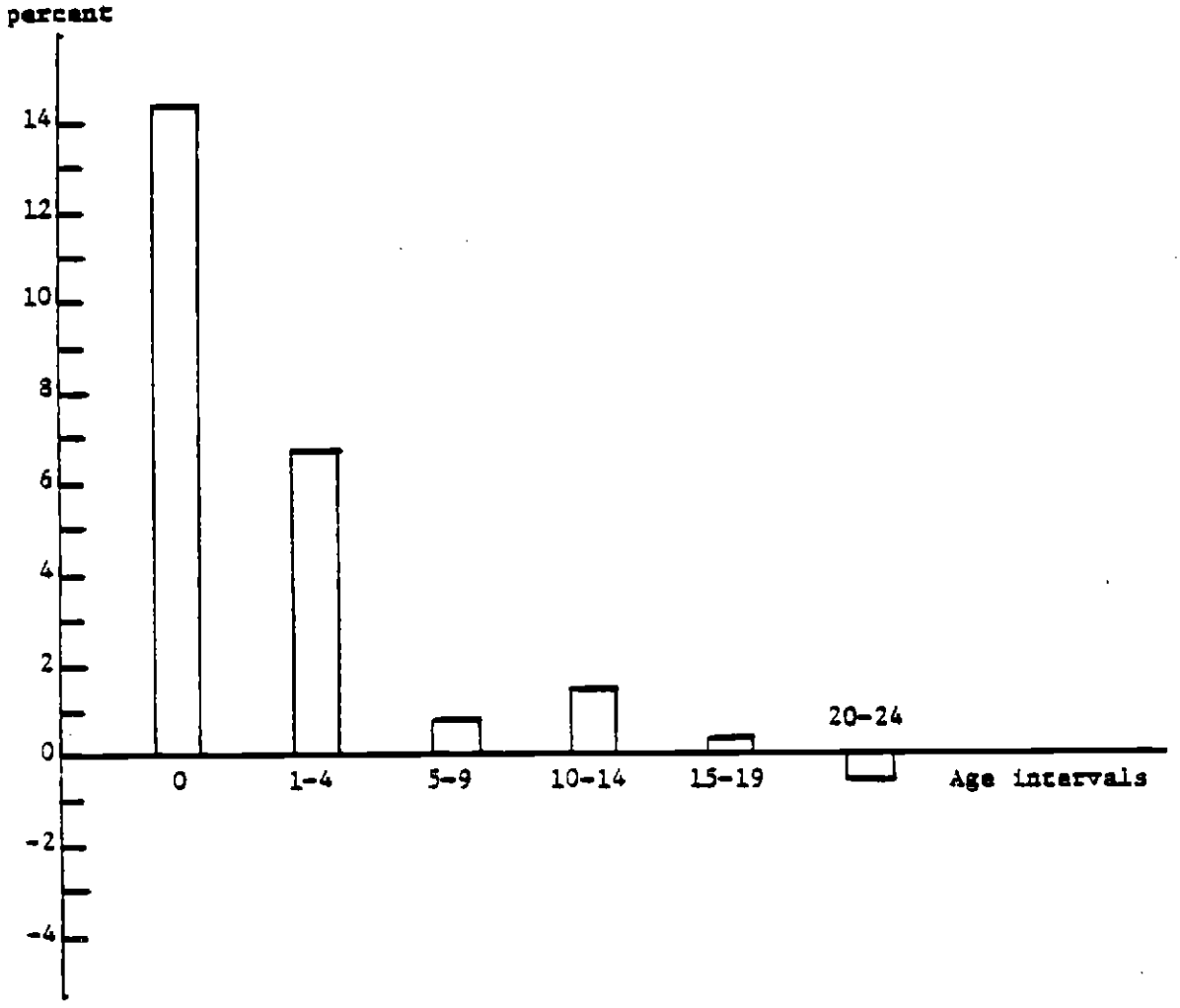
Figure 2 suggests that nutritional deficiencies of early childhood, rather than the overwork or underfeeding of adults, was the main cause of the relatively high death rate of U.S. slaves. This possibility is supported by available data on the death rates. Figure 3 indicates that it was excess death 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

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 1985

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, 1985). 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 intra-uterine 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. Moreover, the adult height of the English laborers who formed the pool from which the Royal Marines were recruited was about one inch less than the adult height of the Trinidad-born slaves. It thus appears likely that some combination of intra-uterine 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 are 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 working 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.

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.[10] 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) 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.[11] 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):

$$(2) \quad \hat{C} = \underset{(5.292)}{30.7877} \underset{(-4.534)}{-5.3851}\ \hat{H} \underset{(-0.382)}{-0.0363}\ \hat{Y} \underset{(-4.040)}{-0.006647}\ T$$

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

$$(3) \quad \hat{I} = \underset{(12.327)}{88.9781} \underset{(-10.797)}{-15.9106}\ \hat{H} \underset{(-3.294)}{-0.3889}\ \hat{Y} \underset{(-4.213)}{-0.00837}\ T$$

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

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)

$\hat{}$ = 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 decline 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.[12]

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 much more sensitive to nutritional status than the mortality rates of adolescents and adults.[13]

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

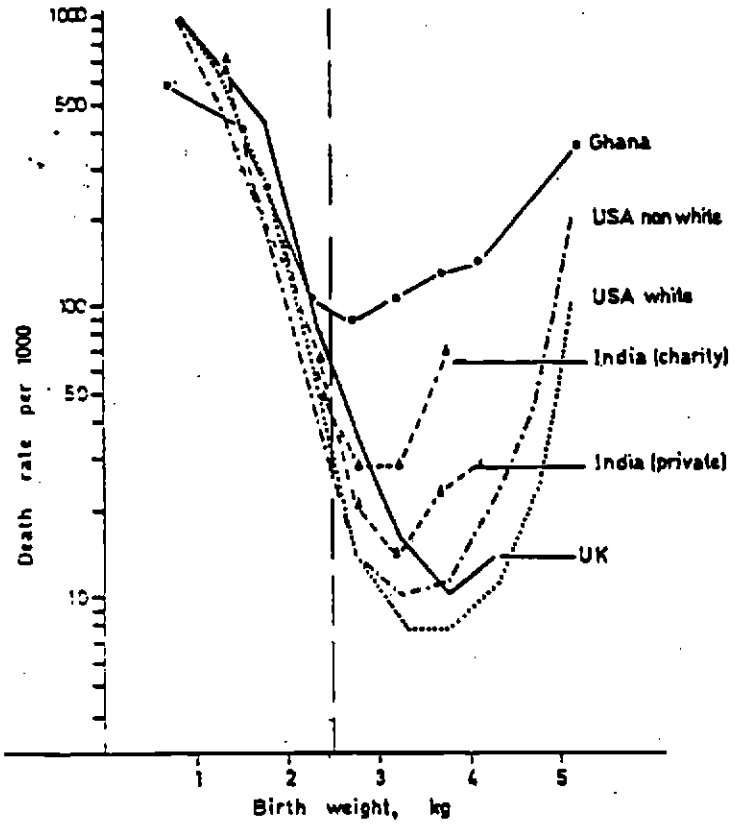
I will first estimate the impact of improvements in nutritional status on non-infants. Because of the absence of data a more indirect approach is required for infants. This illustrative calculation will be applied to the British case.

Let us begin by supposing that the nutrition of lower class English males of 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. 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.[14] 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.[15]

Figure 4

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



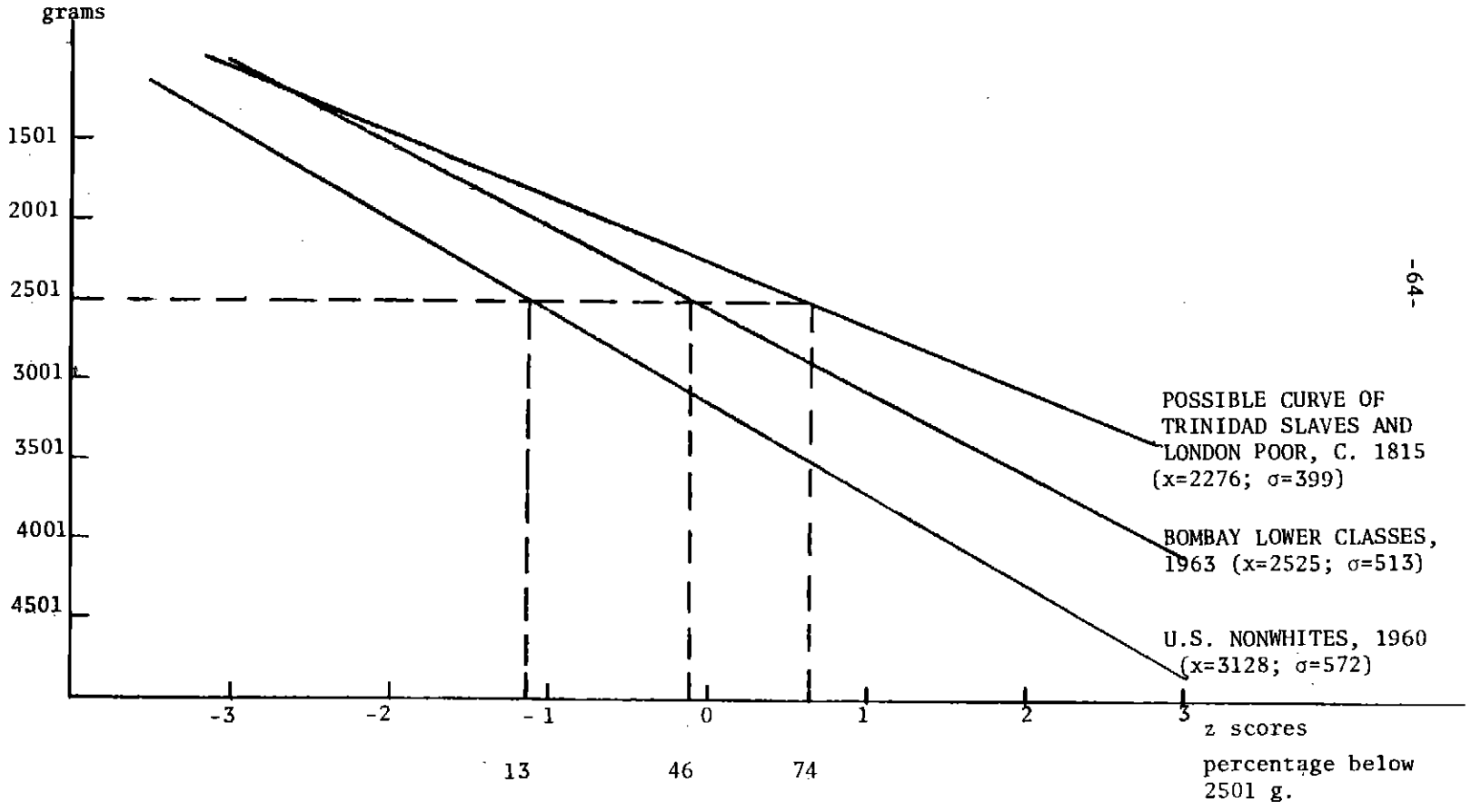
Source: Hytten & Leitch, Physiology of Human Pregnancy (2nd ed.) p. 324.

Mean birth weights vary greatly with the nutritional status of populations (Eveleth and Tanner, 1976; WHO, 1980). This point is illustrated in Figure 5. The lines on this graph are normal approximations of the frequency distributions of birth weights.[16] 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 London poor c.1800.[17] In deriving this distribution I employed established correlations between height and birth weight as well as both published and unpublished information on the heights of the London poor developed by Floud and Wachter.[18] It is probable that the distribution of the birth weights of the London poor in 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 the London poor of c. 1800 were at weights below 2,501 grams.[19]

FIGURE 5

THE PERCENTAGE OF MALE BIRTHS WITH WEIGHTS BELOW 2501 GRAMS IN TWO MODERN POPULATIONS AND THE POSSIBLE PERCENTAGE AMONG TRINIDAD SLAVES AND THE LONDON POOR DURING THE EARLY NINETEENTH CENTURY



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 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 London poor of 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 86 percent [1 - (48.9÷ 346.0) = 0.86].[20]

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.[21]

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

$\overset{*}{S}$ = 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[22] as indicated by the data in Wrigley and Schofield.

Substituting into equation (5) we obtain:

(6) $29 = 0.24\ (86) + 0.76\ (11)$.

Since the age-standardized death rate acutually declined by about 69 percent, equation (6) implies that improvements in

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 ($\bar{x}$=3128 g.; σ=572 g.)	(4) Distribution of birth weights in a population with $\bar{x}$ = 2276 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	346.0

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

Sources: Columns 2 and 3: U.S. Nat. Off. Vital Stat., 1954; Column 4: See footnotes 16 and 17.

nutritional status accounted for about 42 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.[23] Plausible upper and lower bounds on the variables in equation (5) indicate that 42 $\pm$ 10 probably bounds the nutritional contribution at all ages. It should be emphasized that these figures refer not merely to the diet but also to the other factors that affected the nutrients available for growth.[24]

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 middle 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?

Two points seem relevant here. Nutrition does not have an equal influence on the outcome of every disease. Table 11 classifies diseases according to whether nutritional status is likely to influence their outcome. The point to notice is that the diseases on which nutritional influence is minimal were probably the greatest killers before 1700 (McNeill, 1976). For reasons still unclear, the prevalence of these diseases declined between 1600 and 1800, and the main infectious diseases became those in which the influence of nutrition is large. Consequently it is

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
Pertussis	Influenza	Tetanus
Most Intestinal Parasites	Syphilis	Yellow Fever
Cholera	Systemic Worm Infections	Encephalitis
Leprosy		Poliomyelitis
Herpes		

a Morbidity or mortality

Source: JIH, 1983

unlikely that improvements in nutrition had as large an impact on the level of mortality before 1700 as it had afterwards.

The second point 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, nutrition has its greatest impact on the mortality of infants, not of adults. The fact that dukes and earls ate well 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, clothing, and 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 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.

This finding suggests that the peerage did benefit from improved nutritional status during infancy, the weaning years, and perhaps in utero. Several new questions now come to the fore: Did noble women consume large amounts of alcohol during their pregnancy? Was it fashionable in the 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.

4.3. The Wrigley/Schofield/Lee Paradox

In interpreting the regressions between mortality rates and wheat prices, it has often been assumed that the price of wheat was so highly correlated with all other grain prices that it could serve as a proxy for the price of food. It has also been assumed that food shortages would be reflected in their price. Although the second assumption is quite reasonable, it does not follow that a large rise in prices necessarily implies an equally large decline in the supply of food. That would be the case only if the demand elasticity for food was one. The demand elasticity for a food that was a small part of the diet, such as lamb chops or buckwheat, might be equal to or greater than one. But since grains as a whole probably accounted for 75 percent or more of the consumption of English calories during the eighteenth century, they must have had a very low elasticity of demand.

Table 12 shows the decline in the quantity of grain implied by a given price rise under various values of ε, the demand elasticity. For a food as vital as grains were, ε was probably below 0.25, and perhaps even as low as 0.1. It follows that even the so-called "extreme" fluctuations in grain prices (20 percent or more above trend) were probably between 2 and 5 percent below the trend. If that is so, then even the weak relationship between mortality and wheat prices found by various investigators is consistent with the nutritional case. Their regressions imply that mortality rose even when declines in the supply of food were quite small. The fact that their regressions revealed any connection between mortality and grain prices is all the more remarkable, since the dependent variable was dominated by non-infant deaths which are much less sensitive to nutritional status than infant deaths.[25]

Table 12

The Changes in the Price of Grains Associated With Changes in the Quantity Supplied, 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

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.[26] During the next century the final heights of Americans oscillated in a narrow band or declined fairly sharply, losing about 3 centimeters in just two decades beginning with cohorts born about 1835. 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; Fishman and Walker, 1984).

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.1815 and c.1840 appears to have experienced a fairly rapid upward shift in growth schedules, so that the final heights of the c.1840 cohort was about 4 centimeters taller than the c.1815 cohort. Thereafter, the upward shift in growth

schedules slowed down so that the final heights of cohorts born half a century later were only about a centimeter taller 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 Scandanavian 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 cm per decade, which is about 5 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).[27]

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 nearly 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 height, weight, housing conditions, and mortality rates began to be abandoned in favor of the newer and presumably more comprehensive index. Numerous 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 of economists and historians 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 be resolved and I will not attempt to do so here. Rather, I want briefly to sketch some of the new issues about the course of the standard of living that are suggested by the anthropometric and demographic data. When "real wages," per capita income, and other measures all move in the same direction there is little need to probe into their exact meaning. The interesting issues arise when the measures diverge, and it is on some of these issues that I wish to focus.

The evidence so far developed on height and mortality rates suggests that improvements in the living conditions of workers during the nineteenth century may have been more sporadic and uneven (both in time and among subgroups of workers) than is suggested by indexes of real wages or the movements in per capita income. In England the period of rapid improvement in 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 rapid increases in per capita income? Since Williamson and Lindert (1980) have provided weighty evidence that the inequality in the income distribution increased during this period, the answer is that they probably will not. Quite the contrary, by combining the prevailing estimates of per capita income with the information in the series on heights it is possible to obtain a measure of the magnitude of the increase in inequality 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 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 indicate 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; Williamson and Lindert, 1980). Thus the decline in final heights of native-born U.S. white males appears to add to the evidence recently developed by others which suggests that the middle quarters of the nineteenth century witnessed significant but not remarkable increases in the inequality of the American income distribution (Williamson and Lindert, 1980; Pessen, 1973).

It may seem odd to those unfamilar 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 opening up.

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 laboring poor 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 two-thirds of the increase in the mean final height of British males since c. 1830 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).[28] 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

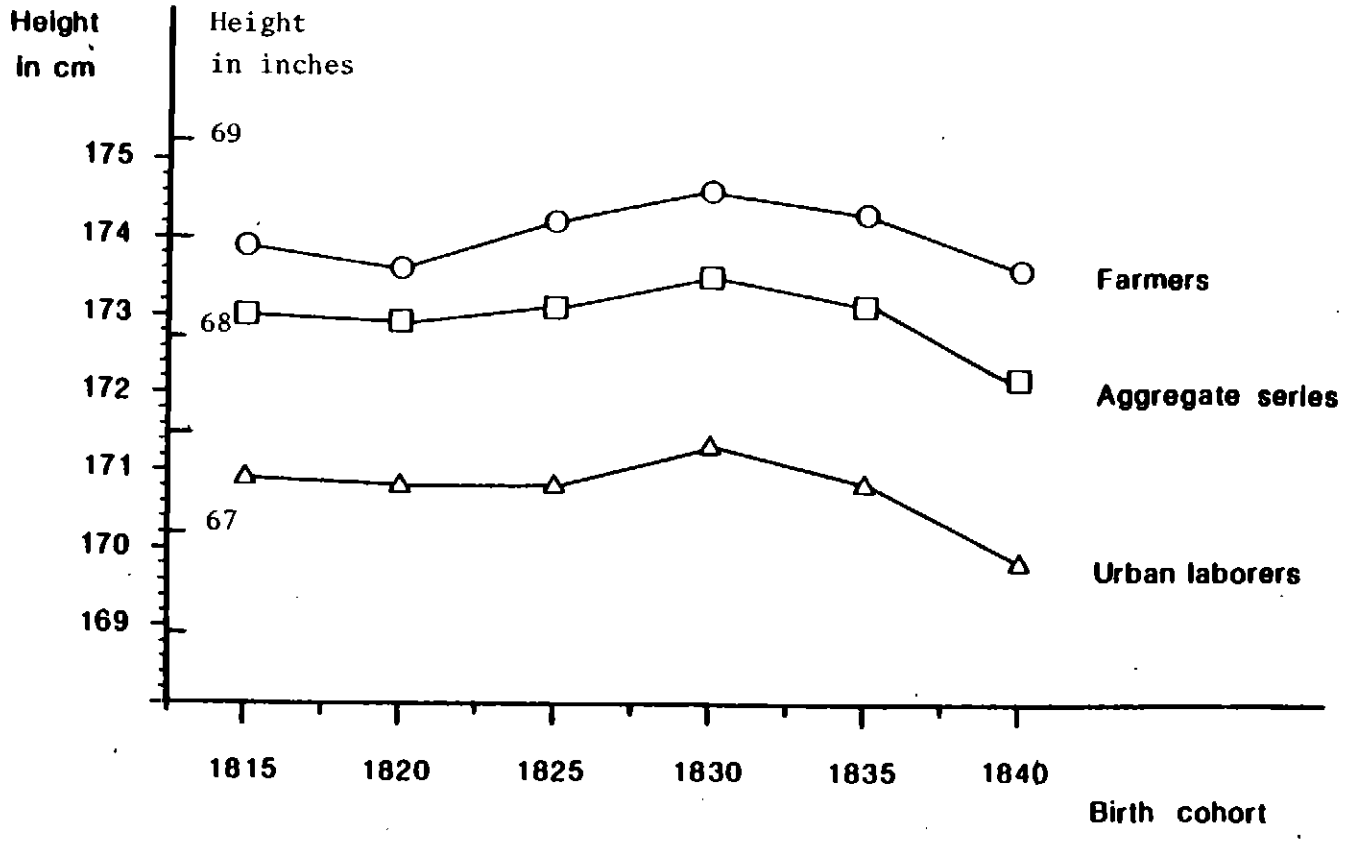
nations, such as the U.S. and Great Britain, by several centimeters (U.S. Bur. Cen., 1983).

The second example pertains to the effect of urbanization on changes in inequality. 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 other mechanisms at work. 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 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 proportion of the population experiencing the poor nutritional and health conditions of the cities as well as to declining heights among urban residents (Margo and Steckel, 1983; Fishman and Walker, 1984).

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 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: Fishman and Walker (1984)

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 malnourishment of foreign-born mothers had an effect on children both in the cities and in the countryside, but that effect was greater in the cities than in the countryside. It is plausible that as much as half of the urban-rural differential in native-born heights was associated with parental ethnicity.[29]

The decline of heights in the rural areas is puzzling, since the ethnic effect could only have accounted for a small share of the rural decline. Another possibility is that exposure of farmers to disease increased as farming pushed into swampy areas in the northern states that were by-passed during the earlier waves of farm settlement. There is also the possibility that part of the decline is a statistical artifact. It may be that children of the

farm families who enlisted during the peacetime years of the early 1880s came on average from poorer families than those who enlisted during the wartime years.[30] 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, 1982).

5. Conclusion

The decline in mortality rates since 1700 is one of the greatest events of human history. 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 human intervention and how much 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 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 desease-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, nutrition 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. The concentration of the impact of improved nutrition in this age group raises the possibility that increases in diarrhea and other diseases which divert ingested nutrients from growth, 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.

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. In commenting on this paper at the Williamsburg conference, Paul David suggested that urbanization could have contributed to the decline in rural nutrition, and to the possible rural increase in mortality, by exporting pollution into the rural counties immediately surrounding them. Using Chicago as a case in point, he noted that the direction of the Chicago River was reversed so that it emptied into the Mississippi River, carrying city waste with it, instead of into Lake Michigan. Agricultural historians have called my attention to the extensive draining of rural swamp lands during the last two-thirds of the nineteenth century, especially in the central states, which might have impaired the health and nutritional status not only of rural adults, but of their children who became more exposed to water-borne diseases that diverted nutrients from growth.

These possibilities point to new issues in the standard-of-living controversy. If David is correct it may turn out that the difficulties created by the 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 insults delivered early in 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). Close examination of these 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 is strongly associated with variations in the rate of immigration.[31]

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 diet, claims on nutritional intake, 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

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

An earlier version of this paper was commissioned by Gunter Steinmann and other organizers of the "Conference on Economic Consequences of Population Change in Industrialized Countries," which was held in Paderborn, West Germany, during June 1983. Successive versions of the paper were presented to seminars at Caltech, the London School of Economics, the Graduate Institute of International Studies (Geneva), Harvard, Chicago, Birkbeck College, Minnesota, Northwestern, Pennsylvania, Princeton, Toronto, and Rochester. Numerous revisions were made as a consequence of points

raised during these sessions. I have also benefitted from comments and criticisms by R.K. Chandra, E. Crimmins, P.A. David, L.E. Davis, A. Fishlow, R.A. Easterlin, F. Furet, D. Galenson, H. Goldstein, M.R. Haines, S. Horton, L. Neale, D.C. North, G.H. Pelto, S.H. Preston, R.S. Schofield, N.S. Scimshaw, S.G. Scrikantia, J.L. Simon, C.E. Taylor, B. Thomas, S.C. Watkins, and E.A. Wrigley.

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 instituions or funding agencies.

[1]From comments made at the Bellagio Conference on Hunger and History, June, 1982.

[2]The principal disadvangtage 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.

[3]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 date 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 percent age 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 which this information was sought.

[4]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. Their 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 groups sheets is drawn from the Main Section.

[5]The first term was suggested by P.H. Lindert at the Williamsburg meeting.

[6]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. See Fishman and Walker, 1984, for a more detailed description of the height series. See Bourne et al, 1984, for a more detailed description of the e_{10} series.

[7](This footnote will describe briefly the derivation of the two curves in Figure 1. Cleaning and processing of the data are still in progress so that the current version of Figure 1 is still provisional and subject to change, as is the discussion of Figure 1).

[8]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).

[9]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.

[10]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 < age 15 (p. 42), females age < 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:

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

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

$$\varepsilon_{m\geq 15} = -2.50$$

$$\varepsilon_{f\geq 15} \approx 0$$

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 or -1.33. Population

weights for England and Wales for 1851 and 1961 (Mitchell, 1975, p. 52) yield elasticities of -1.41 and -1.32.

[11]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).

[12]Total differentiation of equation (3) yields:

$$(3.1) \quad \dot{I} = -15.9106\dot{H} - 0.3889\dot{Y} - 0.00837,$$

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 = 33) of the average annual decline in the infant death rate.

[13]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 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.

[14]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 has been 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). With infant death rates in the neighborhood of 50-100 per thousand, neonatal deaths are about 35 to 60 percent of infant deaths (Shapiro, et al, 1968, Table 1; 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.

[15]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).

[16]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 distributions 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).

17 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 16 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) 2001 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 2001 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 2001 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 2001 grams in the original normal distribution was inflated by the ratio of all births to full-term births in the Delhi sample, but the additional births were distributed over two intervals: under 1501 and 1501-2000. This adjustment for pre-term births produced a convoluted distribution with a mean of 2276 grams (down 24 grams from the underlying normal) and a standard deviation of 399 grams (down 21 grams from the underlying normal).

[18]Several different approaches lead to the same conclusion. The fact that age-2 height is a good predictor of final height (Tanner, 1978) and the fact that the final heights of the London poor was below that of Trinidad slaves suggests that they were shorter at age two. The inference is supported by their mean heights in adolescence which were below those of Trinidadians. The small stature in early adolescence suggest that poor nutritional circumstances in utero, infancy, and early childhood set the London poor on the low growth path indicated by both the data for adolescents and for final heights. Correlations between height and weight during fetal development, at birth, and in early childhood are discussed in Birkbeck, 1976; Southgate, 1978; and Cole, 1979. See Steckel (1984a) and Fogel (1985) for a discussion of methods of estimating birth weight from birth length and early childhood height.

[19]My estimate of the mean birth weight of the London poor 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 in this and in other European lying-in hospitals at the time, women who chose to have deliveries in hospitals tended to have babies that were relatively heavy for their length and that a significant proportion were coming to the hospitals because of difficulties during pregnancy. This possibility is suggested by the exceedingly high death rate among deliveries in maternity hospitals, which were as much as seven times as high as home deliveries (McKeown, 1976), and by the excessive proportion of babies who appear to have had weights that were

more than 1.65 standard deviations above the mean (Tanner, 1981). It is also likely that the left tail of the distribution was truncated because some infants who died immediately after birth were treated as still births.

Two recent studies, one on births between 1851 and 1905 at the University Lying-In Hospital in Montreal (Ward and Ward, 1984), the other of 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. 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.

[20]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 of 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; Evelyth and Tanner, 1976; Hytten and Leitch, 1971; and the sources cited in WHO, 1980.

[21]Equation (5) is derived from the identitiy:

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

which may be rewritten as

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

Total differention of (5.2) yields

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

Since

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

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 $\overset{*}{\alpha} = \overset{*}{\beta} = 0$.

[22] Wrigley and Schofield (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 1000 persons in the total population. Consequently, infant deaths were 23.7 percent of all deaths (6.42 ÷ 27.08 = 0.237).

[23] 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 1801-1805 (.763 x 27.08 = 20.66). Then 20.66 x .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 = .119).

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

the British case) requires confirmation and may have to be modified as additional evidence becomes available.

[25]Even small reductions in the quantity of grain could have had severe nutritional consequences on particular groups or classes. If, for example, the maldistribution of grain increased during a shortfall, the mortality of the poor, especially the urban poor, might have risen even though the food consumption of most of the population changed little (Sen, 1981; Tilly, 1983). Highly aggregated analysis might miss effects that would show up in regressions targeted at groups whose consumption was most likely to suffer even when declines in food production were quite moderate. One of the advantages of the height data and the genealogies is that they permit a far finer differentiation in the experiences of particular socioeconomic groups than has hitherto been possible, so that this sort of disaggregated analysis can be pursued.

[26]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.

[27]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).

[28]The equation used in this computation can be derived as follows:

$$(7.1) \qquad H = (1-\pi)H_u + \pi(H_u - D).$$

Hence

$$(7.2) \qquad H = H_u - \pi D.$$

Differentiating (7.2) totally yields:

$$(7.3) \qquad \overset{*}{H} = \psi \overset{*}{H}_u - (\psi - 1)(\overset{*}{\pi} + \overset{*}{D})$$

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

$\psi = H_u \div H$

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

The following estimates were used (rates of change are in percentage change per annum; the period of change was assumed to be 150 years):

$$\overset{*}{H} = 0.047 \quad (H^0 = 64.75;\ H^1 = 69.5)$$

$$\overset{*}{H}_u = 0.015 \quad (H_u^0 = 69.5;\ H_u^1 = 71)$$

$$\psi = 1.0734 \quad (69.5 \div 64.75)$$

[29]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.

[30]It is possible that the inequality of the income and wealth distributions of farmers and other rural occupations increased and that an increasing share of rural households experienced impoverishment during the relevant period, even though the mean income and the mean wealth of the rural population were rising. Leaders of farm protest movements during the nineteenth century claimed that the incomes of sizable groups of farmers were declining both relatively and absolutely.

[31]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 (1975) 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-borne 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 Chaney (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. REFERENCES

Adams, J.W. and Kasakoff, A.B., 1983, Migration and life-cycles in the American north: age-specific migration rates 1700 to 1850, mimeo, Univ. of N. Carolina.

Appleby, A.B., 1975, Nutrition and disease: the case of London, 1550-1750, J. Interdiscip. Hist. 6:1.

Ashworth, A., 1982, International differences in infant mortality and the impact of malnutrition: a review, Hum. Nut. : Clin, Nut. 36c:7.

Atwater, W.O. and Woods, C.D., 1897, Dietary studies with reference to the food of the Negro in Alabama in 1895 and 1896, U.S.D.A. Off. Exp. Stat. Bull. 38

Beal, V.A., 1980, Nutrition in the Life Span, John Wiley, New York.

Bennett, M.K. and Pierce, R.H., 1961, Changes in the American national diet, 1879-1959, Food Res. Instit. Stud. 2:95.

Birkbeck, J.A., 1976, Metrical growth and skeletal development of the human fetus, in: The Biology of Human Fetal Growth (D.F. Roberts and A.M. Thomson, eds.), p. 39, Taylor and Francis, London.

Bourne, J., Fogel, R.W., Pope, C., and Wimmer, L., 1984, A description and analysis of the data in the DAE/CPE pilot sample of genealogies, mimeo, Univ. of Chicago.

Bouvier, L.F. and van der Tak, J., 1976, Infant mortality: progress and problems, Pop. Bull. 31: No. 1

Brundtland, G.H., Liestøl, K., and Walløe, L., 1980, "Height, Weight, and menarcheal age of Oslo schoolchildren during the last 60 years, Ann. Hum. Bio. 7:307.

Campbell, B.M.S., 1983, Arable productivity in medieval England: some evidence from Norfolk, J. Econ. Hist. 43:379.

Campbell, J., 1984, Work pregnancy, and infant mortality among southern slaves, J. Interdiscip. Hist. 14:793.

Carr, L.G. and Walsh, L.S., 1980, Inventories and the analysis of wealth and consumption patterns in St. Mary's County, Maryland, 1658-1777, Hist. Meth. 13:81.

Case, R.A.M., Coghill, C., Harley, J.L., and Pearson, J.T., 1962, Chester Beatty Research Institute Abridged Life Tables, England and Wales 1841-1960, Part 1, London, 1962.

Chase, H.G., 1969, Infant mortality and weight at birth: 1960 United States birth cohort, Am. J. Pub. H. 59:1618.

Coale, A.J. and Demeny, P., 1966, Regional Model Life Tables and Stable Populations, Princeton Univ. Press, Princeton.

Cole, T.J., 1979, A method for assessing age-standardized weight-for-height in children seen cross-sectionally, Ann. Hum. Bio. 6:249.

Condran, G.A. and Cheney, R.A., 1982, Mortality trends in Philadelphia: age- and cause-specific death rates 1870-1930, Demography 19:97.

Condran, G.A. and Crimmins, E., 1980, Mortality differentials between rural and ruban areas of states in the northeastern United States 1890-1900, J. Hist. Geog. 6:179.

Condran, G.A. and Crimmins-Gardner, E., 1978, Public health measures and mortality in U.S. cities in the late nineteenth century, Hum. Ecol. 6:27.

David, P.A., and Solar, P., 1977, A bicentenary contribution to the history of the cost of living in America, Res. Econ, Hist. 2:1.

Demos, John, A Little Commonwealth: Family Life in Plymouth Colony, Oxford Univ. Press, New York.

Easterlin, R.A., 1977, Population issues in American economic history: a survey and critique, Research in Econ. Hist., 1 Supplement:131.

Eveleth, P.B. and Tanner, J.M., 1976, Worldwide Variations in Human Growth, University Press, Cambridge.

Feigin, R.S., 1981, Interaction of infection and nutrition in Textbook of Pediatric Infectious Diseases (R.D. Feigin and J.D. Cherny eds.), p. 18, W.B. Saunders, Philadelphia.

Fischer, D.H. and Dobson, M.S., 1979, Dying times: changing patterns of death and disease in England and America 1650-1850, mimeo, Brandeis Univ.

Fishman, M.J. and Walker, J.R., 1984, Trends in American stature, 1708-1877, mimeo, University of Chicago.

Floud, R., 1983a, Inference from the heights of volunteer soldiers and sailors, mimeo, NBER.

Floud, R., 1983b, The heights of Europeans since 1750; a new source for European economic history, mimeo, Birkbeck College, London.

Floud, R., 1984, Measuring the transformation of European economies: income, health and welfare, mimeo, Birkbeck College.

Floud, R. and Wachter, K.W., 1982, Poverty and physical stature: evidence on the standard of living of London boys 1770-1870, Soc. Sci. Hist. 6:422.

Floud, R. and Wachter, K.W., 1983, The physical state of the British working class, 1860-1914: evidence from army recruits, mimeo, Birkbeck College, London.

Fogel, R.W., 1985, Without Consent or Contract: The Rise and Fall of American Slavery, Norton, New York.

Fogel, R.W., Engerman, S.L., Floud, R., Margo, R.A., Sokoloff, K., Steckel, R.H., Trussell, J., Villaflor, G.C., and Wachter, K.W., 1983, Secular changes in American and British stature and nutrition, J. Interdiscip. Hist. 14:445.

Fogel, R.W., Engerman, S.L., and Trussell, J., Exploring the uses of data on height: the analysis of long-term trends in nutrition, labor welfare, and labor productivity, Soc. Sci. Hist. 6:401.

Fogel, R.W., Engerman, S.L., Trussell, J., Floud, R., Pope, C.L., Wimmer, L.T., 1978, The economics of mortality in North America, 1650-1910: a description of a reserach project Hist. Meth. 11:75.

Friedman, G.C., 1982, The heights of slaves in Trinidad, Soc. Sci. Hist. 6:482.

Frisancho, A.R., 1978, Nutritional influences on human growth and maturation, Yrbk, Phy. Anthro. 21:178.

Frissell, H.B. and Bevier, I., 1899, Dietary studies of Negroes in Eastern Virginia in 1897 and 1898, U.S.D.A. Off. Exp. Stat. Bull, 71.

Galenson, D.W., 1981, White Servitude in Colonial America: An Economic Analysis, Cambridge Univ. Press, Cambridge.

Gallman, J.M., 1980, Mortality among whites: colonial North Carolina, Soc. Sci. Hist. 4:295.

Gallman, R.E., 1960, Commodity output, 1839-1899, in: Trends in the American Economy in the Nineteenth Century (Confer. on Research in Income and Wealth), pp. 13-71, Princeton University Press, Princeton.

Gallman, R.E., 1966, Gross national product in the United States, in: Output, Employment and Productivity in the United States after 1800 (Confer. on Research in Income and Wealth) p. 3, Columbia Univ. Press, New York.

Gallman, R.E., 1969, Trends in the size distribution of wealth in the nineteenth century: some speculations, in: Six Papers on the Size Distribution of Wealth and Income (Confer. on Research in Income and Wealth),. p. 1, Columbia Univ. Press, New York.

Gallman, R.E., 1982, Influences on the distrubution of landholdings in early colonial North Carolina, J. Econ. Hist. 42:549.

Goldin, C.D., 1979, Evolution of the female labor force in the United States, mimeo, NBER.

Goldin, C.D., and Margo, R.A., 1984, The poor at birth: birth weights and lengths at Philadelphia Alms House, 1847-1874, mimeo, University of Pennsylvania.

Goldstein, H., 1976, Birthweight, gestation, neonatal mortality and child development, in: The Biology of Human Fetal Growth (D.F. Roberts and A.M. Thomson, eds.), pp. 81-102, Taylor and Francis, London.

G.B. Cent. Stat. Off., 1983, Annual Abstract of Statistics No. 119, London, HMSO.

Greven, P.J. Jr., 1970, Four Generations: Population, Land and Family in Colonial Andover, Massachusetts, Cornell Univ. Press, Ithaca.

Guha, D.K., Rashmi, A., and Kochar, M., 1973, Relationship between length of gestation, birth weight, and certain other factors, Indian J. of Pediat. 40: 44.

Habicht, J.-P., Yarbrough, C., and Martoreel, R., Anthropometric field methods: criteria for selection, in Nutrition and Growth (D.B. Jelliffe and E.F.P. Jelliffe, eds.), pp. 365-387, Plenum Press, New York.

Haines, M.R., 1979, The use of model life tables to estimate mortality for the United States in the late nineteenth century, Demog. 16, 289.

Haines, M.R., 1983, Inequality and child mortality: a comparison of England and Wales, 1911, and the United States, 1900, mimeo, Wayne State Univ.

Haines, M.R. and Avery, R.C., 1980, The American life table of 1830-1860: an evaluation, J. Interdiscip. Hist. 11:73.

Henripin, J., 1954, La Population Canadienne au Début du XVIIIe Siècle: Nuptialité-Fecondité-Mortalitité Infantile, I.N.E.D., Paris.

Higgs, R., 1973, Mortality in rural America, 1870-1920: estimates and conjectures, Explor. Econ. Hist. 10:177.

Higgs, R., 1979, Cycles and trends of mortality in 18 large American cities, 1871-1900, Explor. Econ. Hist. 16:381.

Hollingsworth, T.H., 1977, Mortality in the British peerage families since 1600, Population, numéro spécial :323.

Holmes, G.K., 1907, Meat supply and surplus, U.S. Bur. Stat. Bull. 55.

Hytten, F.E. and Leitch, I., 1971, The Physiology of Human Pregnancy, 2nd ed. Blackwell Sci. Pub. Oxford.

Imhof, A.E., 1977, Historical demography in Germany: a research note, Hist. Meth. 10:122.

Jayant, K., 1964, Birth weight and some other factors in relation to infant survival: a study of an Indian sample, Ann. Hu. Genet. 27:261.

John, A.M., 1984, The demography of slavery in nineteenth century Trinidad, unpublished Ph.D. thesis, Princeton Univ.

Jones, A.J., 1980, Wealth of a Nation to Be: The American Colonies on the Eve of the Revolution, Columbia Univ. Press, New York.

Jones, R.A.K., Cummins, M., and Davies, P.A., 1979, Infants of very low birth weight: a 15-year analysis, Lancet, i:1332.

J. Interdiscip. Hist., 1983, The relationship of nutrition, disease, and social conditions: a graphical presentation, J. Interdiscip. Hist. 14:503.

Kahn, C., 1978, The death rate in Memphis in 1860: an application of an alternative estimating technique, mimeo, Harvard Univ.

Kielmann, A.A., Ajello, C.A., and Kielmann, N.S., 1982, Nutrition intervention: an evaluation of six studies, Stud. Fam. Plan. 13:246.

Kielmann, A.A., De Sweemer, C., Chernichovsky, D., Uberoi, I.S., Masih, N., Taylor, C.E., Parker, R.L., Reinke, W.A., Kakar, D.N., and Sarma, R.S.S., 1983, Child and Maternal Health Services in Rural India: The Narangwal Experiment, Vol. 1: Integrated Nutrition and Health, Johns Hopkins Press, Baltimore.

Kiil, V., 1939, Stature and Growth of Norwegian Men during the Past Two Hundred Years, Oslo.

Kiple, K.F. and King, V.H., 1981, Another Dimension to the Black Diaspora: Diet, Disease, and Racism, Cambridge Univ. Press, Cambridge.

Komlos, J., 1984, Stature and nutrition in the Habsburg monarchy, a study of secular changes in the standard of living and economic development, 1720-1920, mimeo, Univ. of Chicago.

Kulikoff, A., 1976, Tobacco and slaves: population, economy, and society in eighteenth-century Prince George's county, Maryland, unpub. Ph.D. thesis, Brandeis Univ.

The Lancet, 1980, The fate of the baby under 1501 g. at birth, Lancet:461.

Lee, R., 1981, Short-term variation: vital rates, prices and weather, in: The Population History of England, 1541-1871 (E.A. Wrigley and R.S. Schofield), p. 356, Harvard Univ. Press, Cambridge, MA.

Levy, D., 1984

Lindert, P.H., 1980, English occupations, 1670-1811, J. Econ. Hist. 40:685.

Lindert, P.H., 1983, English living standards, population growth and Wrigley-Schofield, Explor. Econ. Hist. 20:134.

Lindert, P.H., and Williamson, J.G., 1983, English worker's living standards during the industrial revolution: a new look, Econ. Hist. R. 36:1.

Lindgren, G., 1976, Height, weight, and menarche in Swedish urban school children in relationship to socio-economic and regional factors, Ann. Hum. Bio. 3, 501.

Livi-Bacci, M., 1983, The nutrition-mortality link in past times: a comment, J. Interdiscip. Hist. 14:293.

Lockridge, K.A., 1966, The population of Dedham, Massachusetts, 1636-1736, Econ. Hist. R. 19:318.

McCusker, J.J., 1970, The rum trade and the balance of payments of the thirteen continental colonies, 1650-1775, unpub. Ph.D. thesis, Univ. of Pittsburgh.

McKeown, T., 1976, The Modern Rise of Population, Academic Press, New York.

McKeown, T., 1978, Fertility, mortality and causes of death: an examination of issues related to the modern rise of population, Pop. Studies 32:535.

McKeown, T., 1983, Food, infection, and population, J. Interdiscip. Hist. 14:227.

McKeown, T. and Brown, R.G., 1955, Medical evidence related to English population changes in the eighteenth century, Pop. Studies 9:119.

McKeown, T. and Gibson, J.R., 1950 and 1951, Observations on all births (23,970) in Brimingham, 1947, Brit. J. Soc. Med. 4:221, 5:98, 5:177.

McMahon, S.F., 1980, Provisions laid up for the family, Hist. Meth. 14:4.

McNeill, W.H., 1976, Plagues and People, Doubleday, Garden City, N.Y.

Main, G.L., 1982, Tobacco Colony: Life in Early Maryland, 1650-1720, Princeton Univ. Press, Princeton.

Margo, R.A. and Steckel, R.H., 1982, The heights of American slaves: new evidence on slave nutrition and health, Soc. Sci. Hist. 6:516.

Margo, R.A. and Steckel, R.H., 1983, Heights of native-born whites during the antebellum period, J. Econ. Hist. 43:167.

Mata, L.J., 1978, The Children of Santa Maria Cauque: A Prospective Field Study of Health and Growth, MIT Press, Cambridge, MA.

Matthews, R.C.O., Feinstein, C.H., and Odling-Smee, 1982, British Economic Growth 1856-1973, Stanford Univ. Press, Stanford.

Meeker, E., 1972, The improving health of the United States, 1850-1915, Explor. Econ. Hist. 9:353.

Meeker, E., 1976, Mortality trends of southern blacks, 1850-1910: Some preliminary findings, Explor. Econ. Hist. 13, 13.

Meindl, R.S. and Swedlund, A.C., 1977, Secular trends in mortality in the Connecticut Valley, Hum. Bio. 49:389.

Menard, R.R., 1975, Economy and society in early colonial Maryland, unpub. Ph.D. thesis, Univ. Iowa.

Meredith, H.V., 1970, Body weight at birth of viable human infants: a world-wide comparative treatise, Hum. Bio. 42:217.

Mitchell, B.R., 1975, European Historical Statistics 1750-1975, Macmillan, London.

Mitchell, B.R. and Deane, P., 1962, Abstract of British Historical Statistics, Cambridge Univ. Press, Cambridge.

Moseley, W.H. and Chen, L.C., 1983, An analytical framework for study of child survival in developing countries, mimeo.

Naeye, R.L., 1981, Nutritional/nonnutritional interactions that affect the outcome of pregnancy, Am. J. Clin. Nut., 34:727.

Overton, M., 1979, Estimating crop yields from probate inventories: an example from East Anglia, 1585-1735, J. Econ. Hist. 39:363.

Overton, M., 1980, English probate inventories and the measurement of agricultural change, A.A.G. Bijdragen 23:205.

Pessen, E., 1973, Riches, Class, and Power Before the Civil War, D.C. Heath, Boston.

Pharoah, P.O.D. and Alberman, E.D., 1981, Mortality of low birthweight infants in England and Wales 1953 to 1979, Arch. Dis. Child. 56:86.

Pollard, S., 1981, Sheffield and sweet Auburn--Amenities and living standards in the British Industrial Revolution: a comment, J. Econ. Hist. 41, 902.

Preston, S.H., 1976, Mortality Patterns in National Populations: With Special References to Recorded Causes of Death, Academic Press, New York.

Preston, S.H., Keyfitz, N. and Schoen, R., 1972, Causes of Death: Life Tables for National Populations, Seminar Press, New York.

Preston, S.H., and van de Walle, E., 1978, Urban French mortality in the nineteenth century, Pop. Studies 32:275.

Preston, S.H. and Haines, M.R., 1983, New estimates of child mortality in the United States at the turn of the century, mimeo, Univ. of Pennsylvania.

Raman, L., 1981, Influence of maternal nutritional factors affecting birthweight, Am. J. Clin. Nut. 34:775.

Razell, P.E., 1973, An interpretation of the modern rise of population--a critique, Pop. Studies 28:5.

Rothenberg, W.B., 1984, Markets and Massachusetts farmers: a paradigm of economic growth in rural New England, 1750-1855, unpublished Ph.D. thesis, Brandeis Univ.

Rutman, D.B. and Rutman, A.H., 1979, Now-wives and son-in-law: parental death in a seventeenth-century Virginia County, in: Chesapeake in the Seventeenth Century: Essays in Euramerican Society and Politics (T.W. Tate and D.L. Ammerman, eds.).

Sandberg, L.G. and Steckel, R.H., 1980, Soldier, soldier what made you grow so tall?, Econ. and Hist. 23:91.

Sawyer, M., 1976, Income distribution in OECD countries, OECD Economic Outlook: Occasional Studies., July:3.

Schofield, R., 1983, The impact of scarcity and plenty on population change in England, 1541-1871, J. Interdiscip. Hist. 14:265.

Schuurman, A., 1980, Probate inventories: research issues, problems and results, A.A.G. Bijdragen 23:19.

Sen, A., 1981, Poverty and Famines: An Essay on Entitlement and Deprevation, Oxford Univ. Press, Oxford.

Shapiro, S., Schlesinger, E.R., and Nesbitt, R.E.L., 1968, Infant Perinatal, Maternal, and Childhood Mortality in the United States, Cambridge, MA., Harvard Univ. Press.

Sheridan, R.B., 1985, Doctors and Slaves: A Medical History of Slavery in the British West Indies, 1680-1834, Cambridge Univ. Press, Cambridge.

Smith, D.S., 1973, The demographic history of colonial New England, J. Econ. Hist. 32:165.

Sokoloff, K.L. and Villaflor, G.C., 1982, The early achievement of modern stature in America, Soc. Sci. Hist. 6:453.

Sokoloff, K.L., 1982, The heights of Americans in three centuries: some economic and demographic implications, mimeo, U.C.L.A.

Soltow, L., 1971, Patterns of Wealthholding in Wisconsin Since 1850, U. of Wisconsin Press, Madison.

Soltow, L., 1975, Men and Wealth in the United States 1850-1870, Yale Univ. Press, New Haven.

Southgate, D.A.T., 1978, Fetal measurements, in: Human Growth: Principals and Prenatal Growth (F. Falkner and J.M. Tanner, eds.), v. 1, p. 379, Plenum Press, New York.

Steckel, R.H., 1982a, Height, health, nutrition and labor productivity in the antebellum South, mimeo, NBER.

Steckel, R.H., 1982b, Height, nutrition, and mortality in Ohio, 1870-1900, mimeo, NBER.

Steckel, R.H., 1983, Height and per capita income, Hist. Meth. 16:1.

Steckel, R.H., 1984a, Adversity and diversity: the nutrition, health, and mortality of American slaves from conception to maturity, mimeo, Ohio State Univ.

Steckel, R.H., 1984b, Characteristics of men rejected by the Union Army, mimeo, Ohio State Univ.

Tanner, J.M., 1978, Fetus into Man: Physical Growth from Conception to Maturity, Cambridge, MA, Harvard Univ. Press.

Tanner, J.M., 1981, A History of the Study of Human Growth, Cambridge Univ. Press, Cambridge.

Tanner, J.M., Whitehouse, R.H., and Takaishi, M., 1966, Standards from birth to maturity for height, weight, height velocity, weight velocity: British children, 1965, Arch. Dis. Child. 41: 454-71, 613-35.

Taylor, A.J., ed., 1975, The Standard of Living in Britain in the Industrial Revolution, Methuen, London.

Taylor, C.E., 1982, Infections, famines, and poverty, mimeo, Johns Hopkins Univ.

Taylor, F.K., 1979, The Concepts of Illness, Disease, and Morbus, Cambridge University Press, Cambridge.

Terrenato, L. and Ulizzi, L., 1983, Genotype-environment relationships: an analysis of stature distribution curves during the last century in Italy, Ann. Hum. Bio. 10:335.

Thirsk, J., 1983, The horticultural revolution: a cautionary note on prices, J. Interdiscip. Hist. 14:299.

Thomson, A.M. and Billewicz, W.Z., 1976, The concept of the "light-for-dates" infant, in: The Biology of Human Fetal Growth (D.F. Roberts and A.M. Thomson, eds.) pp. 69-80, Taylor and Francis, London.

Thompson, W.S. and Whelpton, P.K., 1933, Population Trends in the United States, McGraw-Hill, New York.

Tilley, L.A., 1983, Food entitlement, famine, and conflict, J. Interdiscip. Hist. 14:333.

Towne, M.W. and Rasmussen, W.D., 1960, Farm gross product and gross investment in the nineteenth century, in: Trends in the American Economy in the Nineteenth Century (Confer. on Research in Income and Wealth), pp. 255-315, Princeton Univ. Press, Princeton.

Trussell, J. and Bloom, E.E., 1979, A model distribution of height or weight at a given age, Hum. Bio. 51:523.

Trussell, J. and Wachter, K.W., 1984, Estimating covariates of height in truncated samples, mimeo, NBER.

Udjus, L.G., 1964, Anthrometrical Changes in Norwegian Men in the Twentieth Century, Universitetsforlaget.

U.N. Stat. Off., 1983, Demographic Yearbook, New York.

U.S. Bur. Cen., 1975, Historical Statistics of the United States Colonial Times to 1970, Washington D.C.

U.S. Bur. Cen., 1983, Statistical Abstract of the United States 1984. Washington, D.C.

U.S. Dept. Labor, 1897, Conditions of Negroes in various cities. Bull. No. 10.

U.S. Nat. Center Health Stat., 1974, Vital Statistics of the United States 1970, Vol. II, Mortality Part A. Rockville, MD.

U.S. Nat. Cent. Health Stat., 1983, Advance report of final mortality statistics, 1980, Monthly Vit. Stat. Rep. 32, No. 4.

U.S. Nat. Off. Vital Stat., 1954, Weight at birth and its effect on survival of the newborn in the United States, early 1950, Vital Stat. Spec. Dept. v. 39, No. 1.

Van Wieringen, J.C., 1978, Secular growth changes, in: Human Growth, vol. II Postnatal Growth (F. Falkner and J.M. Tanner, eds.), pp. 445-473, Plenum Press, New York.

Vinovskis, M.A., 1972, Mortality rates and trends in Massachusetts before 1860, J. Econ. Hist. 32:184.

Von Tunzelmann, G.N., 1979, Trends in real wages, 1750-1850, revisited, Econ. Hist. R. 32:33.

Wachter, K.W., 1981, Graphical estimation of military heights, Hist. Meth. 14:31.

Wachter, K.W. and Trussell, J., 1982, Estimating Historical Heights, J. Amer. Stat. Assn. 77:279.

Walsh, L.S. and Menard, R.R., 1974, Death in the Chesapeake: two life tables for men in early colonial Maryland, Maryland Hist. Mag. 69:211.

Ward, W.P. and Ward, P.C., 1984, Infant birth weight and nutrition in industrializing Montreal, Am. Hist. R. 89:324.

Waterlow, J.C., Buzina, R., Keller, W., Lane, J.M., Nichaman, M.Z., and Tanner, J.M., 1974, The presentation and use of height and weight data for comparing the nutritional status of groups of children under the age of 10 years, Bull. WHO, 55:489.

Weir, D.R., 1982, Fertility transition in rural France, 1740-1829, unpub. Ph.D. thesis, Stanford Univ.

Wilkin, J.C., 1981, Recent trends in the mortality of the aged Soc. of Aged Trans. 33:11.

Williamson, J.G., 1976, American prices and urban inequality since 1820, J. Econ. Hist. 36:303.

Williamson, J.G., 1981a, Urban disamenities, dark satanic mills, and the British standard of living debate, J. Econ. Hist. 41:75.

Williamson, J.G., 1981b, Some myths die hard--urban disamenities one more time: a reply, J. Econ. Hist. 41:905.

Williamson, J.G., 1982, Was the Industrial Revolution worth it? Disamenities and death in 19th century British towns, Explor. Econ. Hist. 19:221.

Williamson, J.G. and Lindert, P.H., 1980, American Inequality: A Microeconomic History, Academic Press, New York.

Wimmer, L.T., 1984, Phases and strategies in the collection and linking of genealogical data: the status of the DAE/CPE pilot sample as of May, 1984.

Winich, M. and Brasel, J.A., 1980, Nutrition and cell growth, in: Modern Nutrition in Health and Disease (R.S. Goodhart and M.E. Shils, eds.), pp. 592-607, Lea and Febiger, Philadelphia.

World Health Org., 1980, The incidence of low birth weight: a critical review of available information, World Health Stat. Q. 33:197.

Wrigley, E.A. and Schofield, R.S., 1981, The Population History of England, 1541-1871: A Reconstruction, Harvard Univ. Press, Cambridge, MA.

Wrigley, E.A. and Schofield, R.S., 1983, English population history from family reconstitution: summary results 1600-1799, Pop. Studies, 37:157.

Yasuba, Y., 1962, Birth Rates of the White Population in the United States, 1800-1860, Johns Hopkins Press, Baltimore.