

Commentary: 'The child is father of the man.' The relationship between child health and adult mortality in the 19th and 20th centuries

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In the early years of the twentieth century, enormous concern was focused on the relationship between the health of the child and the welfare of the nation. In 1902, Leslie Mackenzie and Matthew Hay informed the Royal Commission on Physical Training (Scotland) that large numbers of children in Aberdeen and Edinburgh were suffering from serious medical conditions which could pose a threat to them in later life, and in 1904 the Interdepartmental Committee on Physical Deterioration devoted more than one-third of its report to conditions affecting the 'juvenile population'.¹ In 1905 Leslie Mackenzie told the Royal Society of Edinburgh that the most important single lesson to emerge from the enquiries was the need to make the individual citizen fit for the environment around them. 'One truth we have already realised—if we would fit the man for his environment, we must begin with the child. "The child is father of the man."²

As this statement indicates, contemporary medical opinion had already begun to emphasize the importance of child health and its relationship to the adult, but it was not until the 1930s that epidemiologists began to devote serious attention to the role which factors operating in early childhood might play in determining the chronological pattern of adult mortality. In 1934, Kermack, McKendrick and McKinlay suggested that if the decline of mortality in England, Scotland, Wales and Sweden in the second half of the nineteenth century was examined in terms of the experience of particular cohorts, then 'it would seem that the actual calendar year is of relatively little importance in determining ... mortality. What is of importance is year of birth of generation or group of individuals under consideration'. They went on to suggest that the first fifteen years of life were particularly important in shaping the health of the adolescent and the adult, and that 'if the above hypothesis is correct, it would be implied that the decreased death rates were largely the result of ... improved physique'.³

The aim of this paper is to examine the impact of Kermack, McKendrick and McKinlay's work on subsequent approaches to the history of health and mortality. The paper begins with an examination of the relationship between infant and child health and adult mortality, and focuses on the history of mortality decline in Sweden and Scotland. The second section seeks to extend Kermack, McKendrick and McKinlay's work on the history of mortality decline in England and Wales back into the eighteenth century, and also highlights some important differences in the relationship between childhood and adult

mortality in the case of men and women. The third section examines the impact of their work on the development of research in the field of anthropometric history. The final section will discuss the implications of this work for our understanding, not only of the causes of mortality decline in the past, but also for the projection of mortality trends in the future.

Infant and child health and adult mortality in Scotland and Sweden

In 1986, Barker and Osmond published a ground-breaking paper on the relationship between infant and childhood mortality and the incidence of ischaemic heart disease in later life.⁴ They found that there was strong correlation between the geographical distribution of infant mortality in the counties of England and Wales in the early-1920s and the distribution of ischaemic heart disease mortality in the same counties in the late-1960s and 1970s. This paper has since provided the foundation for a highly influential research programme, focusing on the links between fetal and infant nutrition and health in later life.^{5–6} However, it is interesting to note that when Kermack, McKendrick and McKinlay published their paper in 1934, they explicitly rejected the argument that infant mortality and, to a lesser extent, mortality in early childhood, played any role in determining the level of mortality among the survivors of each cohort as they grew older. After looking at the chronology of mortality decline at different ages in both Scotland and England and Wales, they concluded: 'the fall in infantile death rate was delayed relatively to the expected by more than a generation. In the case of the 1–4 age-group, it is found that the fall in its death rate was also somewhat delayed ... but to a lesser extent. This group would appear to be intermediate in behaviour between the infants on the one hand and the older children and the adults on the other' (p.700).

In view of the obvious importance of this question, it is interesting to look more closely at the pattern of mortality decline in the countries concerned. In their paper, Kermack, McKendrick and McKinlay sought to demonstrate the anomalous behaviour of infant mortality by comparing the decline in infant mortality with the decline in mortality more generally, but we can highlight the point more clearly by disaggregating the mortality figures in Scotland for children aged 0–1 and those aged 1–4, as shown in Tables 1 and 2. These Tables provide a very clear illustration, not only of the anomalous behaviour of the infant mortality rate, but also of the intermediate status of young-child mortality. They also help to highlight the relative consistency of the pattern of cohort-related mortality during the

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Table 1 Age-specific male mortality rates in Scotland, 1860/2–1980/2. 1860/2 = 100

	0–1	1–4	5–14	15–24	25–34	35–44	45–54	55–64	65–74
1860–2	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0
1870–2	104.9	94.1	106.3	106.9	108.7	112.9	115.6	109.8	98.9
1880–2	98.4	78.9	84.6	81.6	86.4	100.8	112.1	112.8	98.1
1890–2	104.6	73.5	64.3	74.7	80.6	98.4	120.2	125.7	111.3
1900–2	104.2	61.0	49.7	57.5	72.8	94.4	112.7	128.4	110.5
1911–2	90.3	47.6	41.3	46.6	53.9	69.0	90.5	107.1	102.0
1920–2	79.3	38.2	33.1	39.8	45.0	58.1	83.2	107.3	103.9
1930–2	66.4	27.9	31.0	34.5	36.9	53.5	67.2	82.3	93.7
1940–2	61.6	16.1	26.1	39.5	42.1	51.6	74.0	88.2	90.5
1950–2	28.6	4.9	9.6	15.3	19.1	28.5	58.6	87.5	88.4
1960–2	20.8	3.1	7.2	10.7	13.9	25.3	52.8	86.3	91.0
1970–2	14.4	2.4	6.3	11.9	12.3	23.9	50.9	81.4	89.7
1980–2	8.6	1.7	4.2	11.5	11.3	20.7	46.6	73.5	82.0

Source: Mitchell BR. *Abstract of British Historical Statistics*. Cambridge: Cambridge University Press, 1988, pp.66–67; Registrar-General for Scotland, *Annual Report 1982*: 32.

Table 2 Age-specific female mortality rates in Scotland, 1860/2–1980/2. 1860/2 = 100

	0–1	1–4	5–14	15–24	25–34	35–44	45–54	55–64	65–74
1860–2	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0
1870–2	103.5	94.4	107.9	111.8	108.3	107.8	111.8	111.6	96.5
1880–2	96.0	77.0	85.7	92.1	95.8	97.4	103.5	108.0	94.3
1890–2	102.5	72.5	71.4	81.6	91.7	100.9	109.7	122.1	109.2
1900–2	99.7	57.5	57.1	67.1	74.0	87.0	108.3	131.3	106.4
1911–2	86.4	45.3	44.3	51.3	57.3	70.9	86.8	101.4	93.6
1920–2	71.6	35.1	33.1	45.2	51.0	60.6	85.2	108.2	99.4
1930–2	61.3	25.7	28.1	37.7	39.6	47.2	64.8	78.3	84.3
1940–2	55.2	15.2	22.9	39.0	35.1	38.3	56.0	72.6	79.0
1950–2	26.5	4.1	7.1	16.7	20.1	25.5	43.5	61.6	73.7
1960–2	18.5	2.5	5.0	5.7	9.0	19.1	37.0	52.9	63.5
1970–2	13.4	1.8	3.8	4.8	7.6	17.1	38.2	50.3	54.8
1980–2	8.1	1.1	2.9	3.9	6.3	13.9	33.6	48.5	52.5

Source: Mitchell BR. *Abstract of British Historical Statistics*. Cambridge: Cambridge University Press, 1988, pp.67–68; Registrar-General for Scotland, *Annual Report 1982*: 32.

period covered by Kermack, McKendrick and McKinlay, namely 1860–1932.

However, whilst Kermack, McKendrick and McKinlay’s argument about the anomalous behaviour of infant mortality is supported by an analysis of the pattern of mortality decline in Scotland, it is less readily apparent in the case of the Swedish data. In a series of publications, Gunnar Fridlitzius⁷ and Tommy Bengtsson and Mark Lindström⁸ have reinforced Kermack, McKendrick and McKinlay’s basic contention about the importance of a cohort-base approach to mortality, whilst simultaneously challenging the way in which the original authors developed it. According to these Swedish authors, developments in infancy and early-childhood may not have exerted a great influence on mortality in the middle years of life, but they exercised a profound effect on the pattern of old-age mortality. Moreover, in contrast to Barker and his colleagues, they also argued that the most important mechanism influencing both infant mortality and the relationship between infant mortality and old-age mortality was not the standard of nutrition, but the disease environment. In his study, Fridlitzius drew particular attention to the role played

by smallpox, whilst Bengtsson and Lindström highlighted the disease environment more generally.

It is clear that these accounts offer a profound challenge, not only to Kermack, McKendrick and McKinlay, but also to David Barker and his colleagues, but it is not clear how far this should be regarded as a general challenge, as opposed to being a reflection of what one might term the ‘peculiarities’ of the Swedish data. When Kermack, McKendrick and McKinlay published their article in 1934, they pointed out not only that Swedish mortality rates began to decline earlier than mortality rates in either Scotland or England and Wales, but also that there appeared to be a much closer relationship between infant mortality and general mortality in Sweden than in the other countries they studied (p.702). The case for Swedish ‘exceptionalism’ is also supported by an analysis of the French data for this period. Drawing on an analysis of mortality changes in the urban *départements* of Seine, Rhône and Bouches-du-Rhône, Preston and van de Walle argued that ‘the first ages to experience improved mortality were those below ages 15 and 20 (*except infancy*), and the cohorts so benefiting tended to retain

Table 3 Age-specific male mortality in Sweden, 1781/90–1971/80. 1751–80 = 100

	0–10	10–20	20–30	30–40	40–50	50–60	60–70	70–80
1751–1780	100	100	100	100	100	100	100	100
1781–1790	98	108	113	113	104	100	110	108
1791–1800	89	72	79	82	89	93	103	114
1801–1810	89	102	107	105	103	117	118	122
1811–1820	86	80	92	101	103	110	115	115
1821–1830	81	64	85	102	105	108	110	110
1831–1840	66	67	87	108	106	110	109	114
1841–1850	64	62	74	90	92	98	102	107
1851–1860	71	76	78	86	88	95	98	105
1861–1870	64	63	70	71	73	83	92	98
1871–1880	58	58	71	69	65	69	76	87
1881–1890	49	56	65	58	56	60	66	77
1891–1900	40	54	65	57	52	57	64	74
1901–1910	31	50	62	50	48	52	59	69
1911–1920	24	52	76	57	45	50	57	69
1921–1930	17	33	44	36	35	43	54	65
1931–1940	13	25	33	28	32	42	54	67
1941–1950	9	16	23	20	23	36	48	61
1951–1960	5	9	12	13	18	31	46	60
1961–1970	4	8	11	12	18	30	46	60
1971–1980	2	8	11	12	19	32	46	58

Source: Fridlitzius G. The deformation of cohorts: nineteenth-century mortality decline in a generational perspective. *Scandinavian Economic History Review and Economy and Society* 1989;37(3):5.

Table 4 Age-specific female mortality in Sweden, 1781/90–1971/80. 1751–80 = 100

	0–10	10–20	20–30	30–40	40–50	50–60	60–70	70–80
1751–1780	100	100	100	100	100	100	100	100
1781–1790	98	107	102	98	96	102	110	106
1791–1800	88	77	85	86	90	96	104	110
1801–1810	88	104	106	102	103	123	119	118
1811–1820	83	86	94	96	93	110	111	109
1821–1830	68	70	83	84	86	102	102	101
1831–1840	63	70	84	88	91	106	102	105
1841–1850	60	67	71	74	76	94	94	99
1851–1860	67	77	76	79	79	95	96	98
1861–1870	62	65	69	66	68	82	85	88
1871–1880	57	65	72	65	60	71	71	78
1881–1890	48	65	68	60	54	63	63	70
1891–1900	39	64	70	58	52	59	60	68
1901–1910	29	60	69	54	49	53	55	64
1911–1920	22	60	76	56	48	56	55	64
1921–1930	15	39	50	38	40	48	53	62
1931–1940	11	25	36	29	33	44	53	65
1941–1950	7	13	20	18	23	36	45	59
1951–1960	4	6	7	9	16	28	39	53
1961–1970	3	5	5	8	14	23	31	45
1971–1980	2	5	6	7	13	21	27	39

Source: Fridlitzius G. The deformation of cohorts: nineteenth-century mortality decline in a generational perspective. *Scandinavian Economic History Review and Economy and Society* 1989;37(3):5.

their favoured position relative to earlier cohorts as they passed through life' (p. 284; emphasis added).⁹ Indeed, one of the most interesting features of this study was the close similarity between these authors' analysis of the pattern of mortality

change in France and that identified by Kermack, McKendrick and McKinlay in England, Scotland and Wales, despite important differences in the cause-specific nature of mortality.

In addition to the role played by infant and child mortality in determining cohort mortality, both the Scottish and Swedish figures also raise some interesting questions about the different factors which may have disrupted the general pattern by causing the mortality of a particular cohort to depart from its original trend. In a number of cases these deviations are clearly related to the experience of different genders. In the case of Scotland, as we have already seen, there is a striking degree of consistency in the values of the relative mortality statistics for both males and females during the period 1860–1932, but the relationship is much less clear-cut in the following years. This may be partly attributable to the distorting effect of the Second World War on both male and female civilian mortality in 1940–1942, but it also reflects the impact of tobacco-related cancer and other diseases on the mortality of older people from the 1920s onwards. It is interesting to note that the increase in relative mortality values among male cohorts precedes the increase in female cohorts, reflecting sex-specific patterns of tobacco consumption.¹⁰

Although the pattern of mortality decline in Sweden is broadly consistent with a cohort-based hypothesis, both Kermack, McKendrick and McKinlay and Fridlitzius drew attention to what Fridlitzius called 'the deformation of cohorts'. Kermack, McKendrick and McKinlay noted that there was 'a disturbance limited to a rectangular block from 1855 onwards [i.e. from 1855 through to 1925], and affecting the age-groups centred at 10, 20 and 30 years', but they declined to investigate the reasons for this, and simply concluded that 'when this block is omitted, it will be found that the contours run with moderate regularity down the diagonals' (pp.701–02). In his analysis, however, Fridlitzius argued that there were three major 'deformations' in the pattern of cohort-related mortality decline in Sweden between 1750 and *circa* 1920. These included (a) an increase in the mortality rates of men aged 30–50 between 1820 and 1850; (b) a rise in childhood mortality in the 1850s and 1860s; and (c) an increase in both male and female mortality between the ages of 10 and 30 in the 1890s and early-1900s. He also drew attention to two other major features of the Swedish data: a much more simultaneous decline in mortality at all ages in Stockholm during the second half of the nineteenth century; and an increase in nicotine-related mortality, first among males and then among females, among older adults in the twentieth century.

This is not the place in which to engage in a detailed analysis of the reasons for these 'deformations', but it may be worth summarizing the explanations which Fridlitzius provided. He argued that the first 'deformation', the rise in the mortality of males aged 30–50 between 1820 and 1850, was closely-related to the consumption of alcohol, but he was less able to explain the rise in childhood mortality in the 1850s and 1860s. Lars Sandberg and Richard Steckel have suggested Sweden experienced a major nutritional crisis during this period,¹¹ but Fridlitzius rejected this interpretation on the grounds that the greatest increases in childhood mortality resulted from diseases which in themselves had little to do with nutritional deficiencies. He argued that the third 'deformation'—the rise in young adult mortality at the end of the nineteenth century—was most probably related to the increase in tuberculosis mortality. The combination of increased childhood mortality during the 1850s and 1860s and the rise in tuberculosis-related mortality in the

1890s was directly responsible, in his view, for the existence of the disturbing 'rectangle' which Kermack, McKendrick and McKinlay had first highlighted, and then glossed over, in the 1930s.

The decline of mortality in England and Wales since 1700

Although Kermack, McKendrick and McKinlay devoted some attention to the pattern of mortality change in Sweden during the second half of the eighteenth century, they were mainly concerned with the interpretation of changes in mortality in the second half of the nineteenth century. However, it is important to place this work in a more long-term context. During the last 20–30 years, historical demographers have made enormous advances in the use of techniques such as back-projection and family-reconstitution to frame new estimates of mortality change over a much longer period. This research has shown that mortality rates began to fall in England from the 1730s onwards. The decline continued until the end of the 1820s, and was resumed during the second half of the 1860s (Figure 1).¹² It is therefore important to recognise that Kermack, McKendrick and McKinlay's analysis of the pattern of mortality decline focuses on what one might regard as the second phase of mortality decline, from the 1860s onwards.

In addition to shedding new light on the overall pattern of mortality, Wrigley, Davies, Oeppen and Schofield have also shed new light on patterns of age-specific mortality. It now appears that child and adult mortality followed rather different paths during the eighteenth century. The child mortality rate rose during the first half of the eighteenth century, and only began to fall consistently from the 1770s onwards, whilst the fall in adult mortality began somewhat earlier.¹³ Whilst these findings do not invalidate the conclusions which Kermack, McKendrick and McKinlay reached for the nineteenth century, they do help to highlight the importance of specific historical circumstances in influencing demographic relationships.

Although this research suggests that there was no obvious relationship between child mortality and adult mortality in the eighteenth century, there does appear to have been a much closer relationship between the mortality rates of boys and men in the nineteenth century. As we can see from Table 5, the first age group to experience a sustained decline in mortality, with mortality rates falling consistently below 90% of the rate in 1838–1842, were those aged 5–9, whose mortality rates began to fall during the 1850s. As the children in this age group grew older, they carried their enhanced survival prospects with them. Death rates among those aged 10–19 began to fall during the 1860s; death rates among those aged 20–24 began to fall at the beginning of the 1870s; and death rates among those aged 25–34 in the early-1880s. This was what Kermack, McKendrick and McKinlay noticed when they observed that 'each generation after the age of five seems to carry along with it the same relative mortality throughout adult life, and even into extreme old age'.³

In addition to shedding new light on the chronology of mortality decline, Kermack, McKendrick and McKinlay also raised some interesting questions about its causation. In their article, they suggested that the initial improvement in child health reflected the beneficial impact of an improved environment,

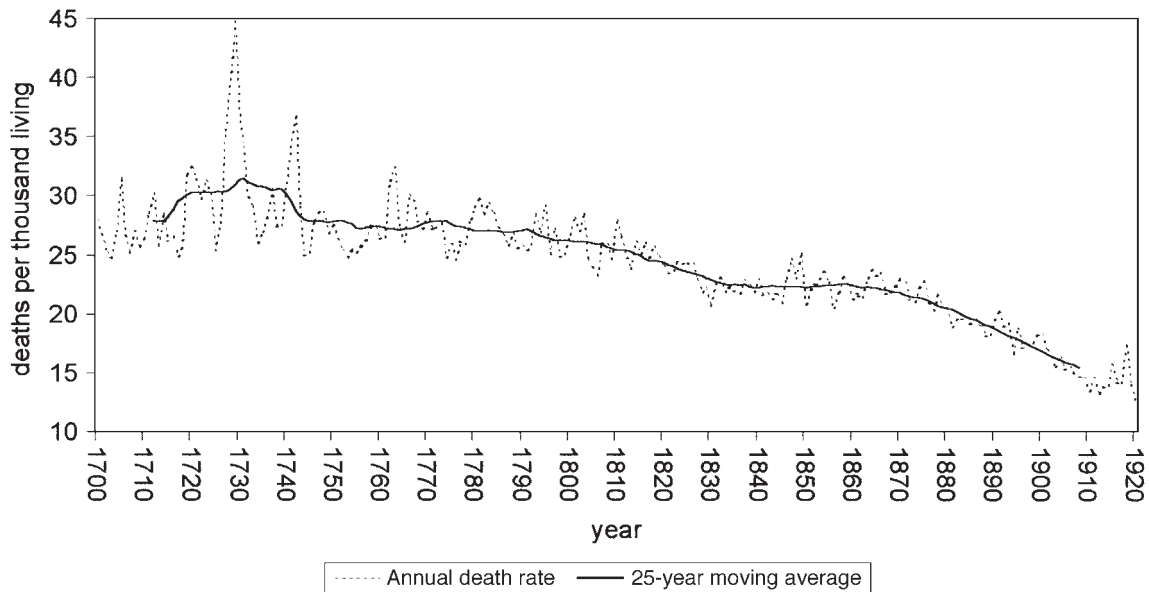


Figure 1 Mortality in England and Wales, 1700–1920

Sources: Fogel RW. Economic growth, population theory and physiology: the bearing of long-term processes on the making of economic policy. *American Economic Review* 1994;**84**:371; Mitchell BR. *British Historical Statistics*. Cambridge: Cambridge University Press, 1988, pp.57–59; Wrigley EA, Schofield RS. *The Population History of England, 1541–1871: A Reconstruction*. Cambridge: Cambridge University Press, 1981, Table A3.3.

and Preston and van de Walle, in their study of French mortality in the nineteenth century, claimed that 'Kermack, McKendrick and McKinlay suggest that improved physique resulting from reduced exposure to infectious diseases in childhood may have exerted a favourable influence on cohort mortality at subsequent ages'.^{3,9} However, whilst it would clearly be premature to rule out the importance of environmental improvements altogether, it is important to recognise that the initial improvement in childhood mortality in England and Wales appears to have predated the period of major improvement in public health conditions.¹⁴ By drawing attention to this fact, Kermack, McKendrick and McKinlay's work suggests that we still need to pay more attention to the role played by improvements in nutrition, as well as public health provision, in determining the pattern of mortality change.¹⁵

Although the figures in Table 5 provide clear evidence of the kind of cohort-based pattern of mortality decline which Kermack, McKendrick and McKinlay identified more than fifty years ago, it is important to note that the relationship appears to be slightly more obvious in the case of male mortality rates than female mortality rates. There are some slight, but not insignificant, differences in the timing of the onset of mortality declines (for example, the death rates among women aged 25–34 and 35–44 began to decline somewhat earlier than the death rates among men in these age-groups), and the pattern of age-specific female mortality decline is slightly more irregular than the pattern of age-specific male mortality decline (Table 6). These differences probably reflect the impact of other factors that had different effects on the health and welfare of men and women in early and middle adulthood. These include: changes in the distribution of household resources (which probably conferred particular advantages on women, in comparison with earlier

generations); changes in access to paid work and in the nature of paid work itself; and, perhaps most importantly, in this period, the decline in the birth rate, and the consequent reductions, not only in the proportions of maternal deaths, but also in the frequency of health impairments associated with repeated childbearing.¹⁶

In recent years, epidemiological research has cast doubt on some of the assumptions made by Kermack, McKendrick and McKinlay, whilst reinforcing others. Their expectation that the process of mortality decline might continue to follow a step-by-step pattern in the twentieth century has not been borne out, but their underlying contention—that the conditions which affect people in early life have a profound effect on their subsequent mortality—has been supported by a wealth of recent studies.^{17,18} This research has had a major impact, not only in the field of contemporary epidemiology, but also in the area of anthropometric history, a branch of economic and social history which uses records of human height and weight to measure changes in the health and well-being of historical populations.

Health, height and mortality

Although epidemiologists and other social and medical observers have long recognised the potential value of anthropometric measurements as indicators of health and well-being, it was not until the 1960s and 1970s that historians started to pay any detailed attention to these records.^{19,20} During the 1970s, Robert Fogel and his colleagues began to collect large amounts of information about the heights and weights of more than 200 000 individuals who were weighed or measured in the US between 1750 and 1977. The results showed not only that there were significant fluctuations in the average heights of successive

Table 5 Age-specific male mortality rates in England and Wales, 1838/42–1978/80. 1838–42 = 100

	0–4	5–9	10–14	15–19	20–24	25–34	35–44	45–54	55–64	65–74	75–84	≥85
1838–1842	100.00	100.00	100.00	100.00	100.00	100.00	100.00	100.00	100.00	100.00	100.00	100.00
1843–1847	100.48	91.39	95.37	96.96	102.14	98.79	101.59	100.22	99.75	104.27	104.77	106.11
1848–1852	101.85	99.79	102.70	97.24	101.28	102.42	104.13	104.59	100.82	100.76	100.49	97.62
1853–1857	101.26	86.34	95.75	93.92	96.57	98.59	100.79	101.90	98.73	100.79	103.65	102.68
1858–1862	100.95	85.92	87.26	88.12	89.51	92.54	98.09	99.55	98.35	99.57	100.58	101.75
1863–1867	104.51	89.29	89.58	87.85	95.07	103.23	110.65	110.51	107.47	103.72	103.48	106.74
1868–1872	100.81	84.66	83.01	82.60	89.51	103.02	109.54	108.72	104.75	102.14	100.72	99.59
1873–1877	95.77	68.70	71.04	72.93	79.01	95.16	111.92	113.87	111.77	107.50	104.58	107.62
1878–1882	90.78	65.97	64.09	64.09	67.02	84.27	103.34	109.40	109.75	105.40	104.55	105.77
1883–1887	87.89	56.72	58.30	60.77	62.31	79.03	99.05	107.72	109.30	108.18	103.47	102.34
1888–1892	86.74	50.00	51.35	56.35	57.82	74.80	98.09	113.20	116.46	113.61	105.36	101.92
1893–1897	87.30	45.59	47.49	52.49	53.53	66.53	88.55	101.45	105.32	101.98	96.38	88.41
1898–1902	84.52	42.02	43.63	48.62	51.82	64.72	87.44	103.47	109.24	105.89	100.28	94.25
1903–1907	72.39	36.55	39.38	42.54	44.54	56.65	72.97	91.05	101.33	98.11	95.17	92.99
1908–1912	59.41	34.24	37.84	40.33	40.69	50.40	65.18	83.89	95.51	97.50	95.77	89.40
1913–1917	52.12	35.92	41.31	44.75	59.96	64.72	68.04	82.21	95.25	102.20	101.05	93.22
1918–1922	47.77	37.18	43.24	50.28	65.31	82.66	66.14	72.82	83.16	92.19	93.64	84.13
1923–1927	34.12	25.84	30.50	35.08	35.12	38.31	49.76	63.53	76.20	87.92	93.53	93.93
1928–1932	31.37	25.00	30.89	35.91	35.12	35.89	46.90	64.54	76.33	89.20	94.71	97.41
1933–1937	26.58	23.11	26.64	31.22	32.12	31.65	40.86	61.86	75.70	85.60	93.85	91.64
1938–1942	23.44	20.38	25.48	32.32	43.90	36.49	39.90	59.96	77.97	84.44	93.85	87.94
1943–1947	19.90	13.87	19.31	24.86	41.54	33.06	33.07	52.13	71.52	77.64	83.00	75.48
1948–1952	11.30	7.98	11.20	14.92	15.42	16.94	23.85	46.31	71.65	81.57	84.76	84.21
1953–1957	9.34	5.46	8.49	11.60	12.42	12.90	20.35	42.73	69.94	82.18	86.36	81.87
1958–1962	8.66	5.04	7.72	12.15	11.99	11.49	19.08	40.72	69.05	82.79	85.50	80.94
1963–1967	7.46	4.83	7.72	13.81	11.35	10.89	19.40	40.83	67.66	80.90	82.35	81.83
1968–1972	6.48	4.20	7.72	12.43	10.06	10.08	18.28	39.93	66.33	81.85	83.79	83.35
1973–1977	5.13	3.57	5.79	12.15	10.71	9.68	17.01	39.60	62.41	77.09	80.07	79.16
1978–1980	4.91	3.15	5.79	11.97	9.99	9.07	15.63	36.91	59.07	73.47	77.20	77.11

Source: Mitchell, BR. *Abstract of British Historical Statistics*. Cambridge: Cambridge University Press, 1988, pp.60–65.

generations of native-born white males in the US, but also that these fluctuations appeared to be closely-related to fluctuations in life expectancy (Figure 2). The average height of native-born white males was already quite close to modern standards by the middle of the eighteenth century. However, average heights declined during the middle years of the nineteenth century, and only began to recover during the early years of the twentieth century.²¹

When Fogel and his colleagues began their research, they were primarily interested in the relationship between height, as a measure of nutritional status, and current levels of mortality, but it soon became apparent that height may also have an impact on mortality experience in the future. In 1984, the Norwegian epidemiologist, Hans Waaler, examined the relationship between height, weight and body mass index and a range of different causes of mortality among men and women who were weighed and measured during a national radiography survey between 1963 and 1979. Although he found that there was no clear relationship between adult height and cancer mortality, height was inversely related to the risk of mortality from tuberculosis, obstructive lung disease and cardiovascular disease. His overall conclusion was that height was inversely associated with mortality as a whole, and that the long-term trend towards greater height should mean that 'the new cohorts

which have grown up under optimal nutritional conditions will have better health in the future'.²²

One of the first clear indications of a relationship between height and adult mortality in historical populations was provided by Floud, Wachter and Gregory in their study of the main trends in height and nutritional status in the UK between 1700 and 1980. They found that the average heights of men who joined the British Army fell sharply between the birth cohorts of the 1820s and late-1840s, and only began to recover after about 1850. However, one of their most striking conclusions was that there appeared to be a close correspondence between the timing of the increase in stature and the onset of the improvement in age-specific mortality rates. 'The height data make the link between nutrition (although in a wider sense) and mortality which McKeown [in *The Modern Rise of Population*] could only infer. [They] ... also provide concrete support for the assertions made by Kermack, McKendrick and McKinlay ... that the pattern of the decline of mortality—in which each successive cohort exhibited relatively lower mortality throughout life—could best be explained by improvements in the health of children'.²³

In recent years, anthropometric historians have broadened the focus of their enquiries to include not only height, but also weight and the body mass index (BMI). In 1993, Dora Costa compared the results of Waaler's investigations into the relationship

Table 6 Age-specific female mortality rates in England and Wales, 1838/42–1978/80. 1838–42 = 100

	0–4	5–9	10–14	15–19	20–24	25–34	35–44	45–54	55–64	65–74	75–84	≥85
1838–1842	100.00	100.00	100.00	100.00	100.00	100.00	100.00	100.00	100.00	100.00	100.00	100.00
1843–1847	100.20	91.14	93.97	94.87	100.22	100.78	101.28	100.77	101.58	106.13	105.88	106.34
1848–1852	102.38	99.78	98.94	96.58	103.36	105.66	105.44	104.74	102.01	102.69	101.44	98.46
1853–1857	102.61	87.04	88.65	91.69	96.87	98.44	99.20	98.98	98.71	101.55	103.77	101.73
1858–1862	101.83	88.34	84.75	85.82	91.05	93.75	94.40	95.52	96.76	101.17	101.74	101.43
1863–1867	105.72	87.26	82.62	82.89	92.62	98.05	99.84	103.07	102.44	103.75	105.10	103.76
1868–1872	101.93	80.99	75.89	77.26	85.91	92.19	94.56	99.10	99.14	100.48	100.81	96.86
1873–1877	94.71	66.09	65.60	66.50	76.96	85.16	94.40	101.41	103.67	105.54	103.27	105.89
1878–1882	90.04	63.93	59.57	59.41	67.34	76.95	88.32	97.31	102.80	103.93	102.43	101.22
1883–1887	86.21	57.45	56.74	55.50	63.53	73.83	85.60	97.70	102.73	105.13	101.21	97.36
1888–1892	84.81	51.19	50.35	49.88	55.93	66.99	83.20	98.98	107.69	110.06	104.28	97.41
1893–1897	85.85	47.73	46.10	45.23	50.11	59.38	75.52	90.65	97.70	99.10	94.48	86.82
1898–1902	82.78	44.49	42.20	39.61	44.97	53.71	71.52	91.42	98.78	101.65	98.38	92.06
1903–1907	70.73	38.66	38.65	35.70	39.15	46.68	60.80	80.54	89.79	92.77	91.27	89.15
1908–1912	57.86	35.64	36.17	33.01	36.47	41.21	53.12	74.01	83.75	89.70	89.56	85.04
1913–1917	50.08	36.29	38.65	35.45	37.14	39.45	50.08	70.68	79.44	89.43	91.15	88.02
1918–1922	45.61	38.66	42.20	42.54	48.99	52.73	48.80	63.25	70.38	79.86	85.97	82.13
1923–1927	32.08	24.62	27.66	29.83	33.33	32.81	38.08	54.29	64.56	76.72	84.49	88.65
1928–1932	29.11	22.89	26.60	28.85	32.66	32.03	36.32	53.14	63.62	76.07	85.16	89.93
1933–1937	24.57	21.81	22.34	24.69	29.31	28.52	33.12	49.81	60.17	72.80	81.62	86.94
1938–1942	21.53	17.49	19.50	23.47	28.41	25.78	29.12	45.84	56.22	68.60	78.42	84.47
1943–1947	18.23	10.80	14.18	17.36	23.49	21.29	23.68	38.41	48.17	60.40	71.03	71.74
1948–1952	10.23	5.62	7.80	9.78	12.98	14.45	18.72	33.55	44.93	59.06	71.96	76.07
1953–1957	8.46	3.67	5.67	5.62	6.49	9.38	15.84	29.96	40.76	54.65	67.74	75.65
1958–1962	7.87	3.24	4.96	4.89	5.37	7.42	14.24	28.43	38.75	52.34	66.15	75.37
1963–1967	6.80	3.24	5.32	4.89	5.15	6.64	14.08	28.04	37.10	49.31	61.35	70.75
1968–1972	5.65	3.24	3.90	4.89	4.70	5.86	13.12	27.91	37.24	47.73	59.89	71.36
1973–1977	4.54	2.59	3.55	4.40	4.47	5.47	12.00	27.53	36.52	44.90	56.68	68.54
1978–1980	4.52	2.16	3.55	3.67	4.47	5.53	10.93	26.03	35.23	43.16	53.00	67.38

Source: Mitchell BR. *Abstract of British Historical Statistics*. Cambridge: Cambridge University Press, 1988, pp.60–65.

between height, weight, BMI and mortality among modern Norwegians with the experience of men who had served in the Union Army during the American Civil War in the 1860s. In both cases, she found that both height and BMI were closely correlated with the risk of mortality. She concluded if the mean height of the Union Army veterans had been shifted one standard deviation to the right, they might have experienced 9% less mortality over a 20-year period. If it had been possible to shift their BMI one standard deviation to the right, then mortality might have been reduced by 14%.^{24–26}

These findings also have important implications for our understanding of the relationship between stature and morbidity. In their examination of the Union Army records, Fogel and his colleagues found that individuals who were shorter and had lower BMI also suffered from a significantly higher prevalence of chronic diseases. They also showed that just as height and BMI increased during the course of the twentieth century, so the prevalence of these conditions decreased. This research challenges notions that the decline of mortality has been associated with an increase in the incidence of age-specific morbidity, and suggests that the relationship between infectious and chronic conditions may be more complex than some authorities have supposed.^{27–30}

Conclusions

As this paper has shown, there is now considerable interest among historians in the relationship between indicators of health in early life and subsequent mortality. Whilst this research has been reinforced by contemporary epidemiological research, it has also contributed to it, by suggesting that many of the relationships found among modern populations may also have existed in the past. As Dora Costa has noted, the finding that there appears to be a consistent relationship between height and mortality across the generations suggests that it is certainly legitimate to use standards derived from contemporary industrialized populations to investigate aspects of the health and mortality of people in the past, and among people living in less industrialised countries today.²⁴

Whilst this paper has been mainly concerned with the relationship between height and mortality in the past, it has obvious implications for the prediction of future health trends. In a recent paper, Davey Smith *et al.* have shown that increased height may be positively associated with the risk of mortality from a number of different diseases, including prostate cancer, lymphoma and colorectal cancer, whose incidence in the UK is currently increasing, but increased height continues to be

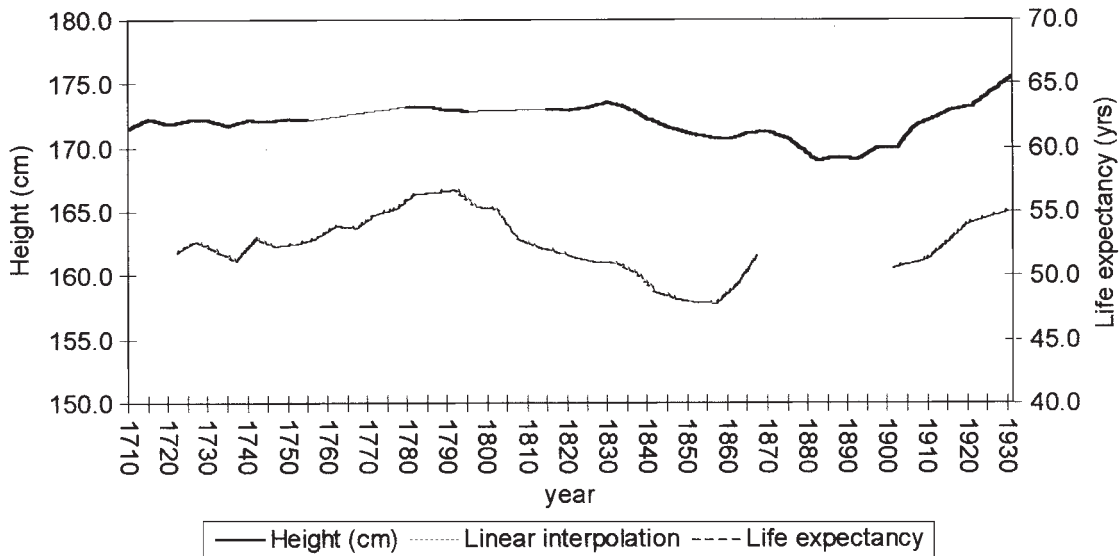


Figure 2 Height and life expectancy at age 10 in the United States, 1710–1931

Notes: Heights are arranged by year of birth. Life expectancy is expectation of life at age 10. The figure shown for e^0_{10} in 1722 is the average figure for the period 1720–24.

Source: Fogel RW. Nutrition and the decline in mortality since 1700: some preliminary findings. In: Engerman SL, Gallman RE (eds). *Long-term Factors in American Economic Growth*. Chicago: University of Chicago Press, 1986, pp.462–65, 510–12.

inversely related to mortality as a whole.³¹ When one considers the global pattern of the causes of premature mortality, it still seems likely that improvements in the health and nutrition of children will continue to play a major part in the reduction of adult mortality for many years to come.^{32–33}

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Commentary: William Ogilvy Kermack and the childhood origins of adult health and disease

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The 1934 Lancet paper by Kermack, McKendrick and McKinlay was a landmark in the discussion of birth cohort influences on adult disease risk.¹ It pre-dated Wade Hampton Frost's paper on cohort influences on tuberculosis mortality,² and, whilst it was pre-dated by several demographic analyses highlighting birth cohort influences,^{3,4} was prescient in using these analyses to inform hypotheses regarding early-life exposures and their influence on later disease. As Kermack, McKendrick and McKinlay concluded, the data behaved as if 'the expectation of life was determined by the conditions which existed during the child's early years', and concluded, 'the health of the child is determined by the environmental conditions existing during the years 0–15, and ... the health of the man is determined preponderantly by the physical constitution which the child has built up'.¹

The idea that adult disease is determined in early-life is currently most influentially reflected in the 'fetal origins' work of Professor David Barker and his colleagues. In this formulation

the critical period of early-life development which is influenced by environmental exposures and has long-term effects on later health is the intra-uterine period.⁵ Interestingly Kermack *et al.* noted that one exception to the general pattern they observed—of mortality rates initially declining in the young, and only declining at older ages when the cohorts experiencing the initial mortality declines had entered these older age groups—was mortality under one year of age, which started to fall only after mortality at later ages had done so. They suggested that infant mortality was dependent upon the health of the mother, and thus improvement in infant mortality followed the generational improvement in the vitality of women of childbearing age. Kermack *et al.* suggested that the fall in the death rate at ages 1–4 was also somewhat delayed, but to a lesser extent than infant mortality. They did not provide data to support this assertion, however, and as Harris shows, at least in the case of Scotland, the fall in 1–4 year-old mortality fitted in well with the cohort-specific falls, occurring 10 years before the fall in 5–14 year-old mortality.⁶ The data suggest that very early-life development—before or within the first year of life—could not determine the mortality risk of birth cohorts as they aged. Other recent research—taking a lead from the seminal work of Anders Forsdahl^{7,8}—has investigated the possibility that

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