Public health, nutrition and the decline of mortality: the McKeown thesis revisited

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SUMMARY. The medical writer, Thomas McKeown, can justifiably claim to have been one of the most influential figures in the development of the social history of medicine during the third quarter of the twentieth century. Between 1955 and his death in 1988, he published a stream of articles and books in which he outlined his ideas about the reasons for the decline of mortality and the ‘modern rise of population’ in Britain and other countries from the early-eighteenth century onwards. Although McKeown’s main aim was to deflate the claims made by the proponents of therapeutic medicine, his publications have sparked a long and protracted debate about the respective roles of improvements in sanitation and nutrition in the process of mortality decline, with particular emphasis in recent years on the impact of sanitary reform in the second half of the nineteenth century. This paper attempts to place the debate over the ‘McKeown thesis’ in a more long-term context, by looking at the determinants of mortality change in England and Wales throughout the whole of the period between circa 1750 and 1914, and pays particular attention to the role of nutrition. It offers a qualified defence of the McKeown hypothesis, and argues that nutrition needs to be regarded as one of a battery of factors, often interacting, which played a key role in Britain’s mortality transition.

Keywords. Public health, sanitation, diet, nutrition, mortality, living standards, real wages, housing, state intervention, ‘McKeown thesis’.
Between 1700 and 1911-15, the crude death rate in England and Wales declined from 27.9 deaths per thousand living to 14.4 and average life expectancy at birth increased from 37.1 (in 1701) to 53.5 (in 1910-12). These figures reflect a major improvement in the life-chances of the British population over the course of the period, and therefore it is hardly surprising that the ‘McKeown thesis’, which attempts to account for the decline of mortality and the ‘modern rise of population’, should have played such an important part, not only in debates about economic, social and medical history, but also in the fields of population studies and historical epidemiology. However, in recent years opinion has undoubtedly moved against McKeown and his coauthors, and it has even been suggested that the time may have come ‘to draw a line under the McKeown interpretation and simply acknowledge that its greatest strength has proved to be its enduring ability to stimulate debate’. If this is right, then this paper may, in some respects, seem somewhat ill-timed.

Although McKeown examined a wide range of factors in his efforts to account for the decline of mortality, he attached the greatest importance to the improvement of nutrition, and this has led many of his critics to complain that he failed to take sufficient account of such factors as changes in the virulence of infectious organisms, improvements in personal and domestic hygiene, medical intervention and, perhaps most importantly, the beneficial effects of the sanitary revolution of the second half of the nineteenth century. However, as Hionidou has recently reminded us, epidemiologists have continued to emphasize the importance of the role which nutrition can play in combating disease, during famines and at other times. This suggests that the real issue is not whether nutrition is capable of influencing trends in
mortality, but whether changes in nutrition did have an influence on the pattern of mortality in England and Wales during the eighteenth and nineteenth centuries. Although it is difficult to offer any categorical answers to this question, this paper will suggest that it would be wrong to exclude the role of nutritional change altogether, and that nutrition should be regarded as one of a battery of factors, often interacting, which played a key role in Britain’s mortality transition.

The synergistic relationship between nutrition, infection and mortality

During the course of the last century, there have been many advances in our knowledge and understanding of the essential requirements for human nutrition. Seebohm Rowntree confined his analysis of essential nutrients to fats, carbohydrates and proteins, but during the interwar period, nutrition researchers became increasingly interested in the role played by vitamins and minerals in the aetiology of what subsequently became known as ‘deficiency diseases’. After the Second World War, this research led to the identification of what is now known as ‘protein-energy malnutrition’ or ‘protein-calorie malnutrition’, which Tomkins has defined as a condition in which ‘deficiencies of major body nutrients, resulting from a diet which is generally inadequate in energy and protein, are frequently accompanied by deficiencies of micronutrients’. Nutrition researchers have also drawn an important distinction between the concept of nutrition, or diet, and nutritional status. The concept of nutrition refers to the amount and quality of the food consumed by each individual, whereas nutritional status refers to the balance between the food consumed by each individual and the
claims made upon it. As Dasgupta and Ray have argued, the most important category of claims are those required for the maintenance of the body's basic functions, such as temperature control, the circulation of the blood, and breathing, but human beings also require additional quantities of food in order to enable them to perform work and ward off the effects of disease. Individuals who perform large amounts of energy-intensive work require more food than individuals engaged in more sedentary occupations, whilst individuals who are subjected to repeated bouts of infection require more food than individuals whose environments are largely disease-free. The amounts of food required by individuals also vary according to the size of their bodies. Children require less food than adults, and the average woman requires less food for the maintenance of her normal functions than the average man. 6

Although it is generally accepted that nutrition and infection are interrelated, it can often be difficult to distinguish between the two. This is partly related to the fact that those individuals who are most likely to suffer from defective diets are also the individuals who are most likely to be living in insanitary and overcrowded environments, with the result that those individuals who are most likely to show signs of malnutrition are also the individuals who are most susceptible to outbreaks of infectious disease. 7 However, it is also important to recognize that the effects of malnutrition and infection can also be mutually reinforcing. As Scrimshaw and SanGiovanni have argued, infection can have an adverse effect on nutrition, because it leads to the suppression of appetite and inhibits the body's ability to digest those nutrients which are consumed, and malnourished individuals are less likely to recover from an infection once it has been contracted. 8 As a result, it can
often be difficult to determine which factor plays the greatest role in determining the outcome of an infectious disease.

However, despite these problems, it seems clear that there is a widespread, if not universal, consensus on the part of the epidemiological community that nutritional deficiencies do play an important part in the development of a wide range of infectious diseases. Scrimshaw, Taylor and Gordon argued that even though nutritional factors tended to have little effect on the outcome of an infection in cases where natural resistance was either high or low in relation to the virulence of the infection, nutritional factors were important in situations where there was an equilibrium between the natural or constitutional resistance of the host and the virulence of the infective agent, and this conclusion has been reinforced by subsequent research. In 1991, Lunn concluded that ‘whatever the cause of the deterioration in nutritional status, it has become generally accepted that malnutrition predisposes an individual to infectious diseases’ and that ‘when illness does strike, it is likely to be more severe, prolonged, and carries an increased risk of death or permanent damage’.  

Although it seems clear that nutrition can play an important role in the development of a number of infectious diseases, the nature of this role may not always be straightforward. Aaby argued that nutritional factors played a very limited role in the outcome of measles epidemics, and that the progress of the disease was more likely to be related to the extent of overcrowding and the intensity of the infective dose, but this interpretation has not been accepted uncritically. In a wide-ranging review, Tomkins argued that even though cases of mild to moderate malnutrition had little effect on the initial stages of measles infection, they did have
an effect on the outcome of post-viral complications, and severe cases of malnutrition had an effect on all stages of the disease.\textsuperscript{12}

During the last two decades, epidemiologists have devoted considerable effort to understanding the ways in which nutritional deficiencies can affect a range of different conditions. In 1982, a multidisciplinary group of historians, demographers, economists, food scientists and nutritionists concluded that nutritional status had relatively little effect on the development of such diseases as plague, typhoid, tetanus, smallpox or malaria, and that it only exerted a ‘variable’ impact on the outcomes of diphtheria, influenza, syphilis and typhus.\textsuperscript{13} However, they believed that it had a strong effect on the outcomes of such diseases as cholera, measles, leprosy, tuberculosis, and both bacterial and viral respiratory infections. From the point of view of the later sections of this paper, it may be worth noting that this list includes a number of conditions, including tuberculosis, which made a major contribution to the decline of mortality in Victorian Britain.\textsuperscript{14}

One of the main areas of difficulty in interpreting these arguments concerns the question of whether or not there is a ‘threshold’ at which levels of malnutrition become sufficiently severe to affect disease outcomes, or whether all levels of ‘sub-optimal’ nutrition can have adverse consequences. The majority of observers appear to believe that the relationship between nutrition and infection is not continuous and that, to quote Scrimshaw and SanGiovanni, nutrient deficiencies are only likely to affect disease outcomes if they are ‘sufficiently severe’.\textsuperscript{15} However, Lunn suggested that ‘some deficiencies, particularly those in the cell-mediated immune system, do appear to be affected at an early stage of undernutrition’, and even though it is not yet clear how severe such changes need to be before immune status is affected, ‘it seems probable that where reductions in the immune
components described are found, there will be an overall increased susceptibility to infectious disease\(^\text{16}\).

**Nutrition and the decline of mortality before 1820**

During the last twenty years, there has been considerable debate over the precise pattern of mortality change in England (or England and Wales) since the beginning of the eighteenth century. Razzell suggested that mortality may have declined substantially during the first half of the eighteenth century, but this suggestion was rejected by Wrigley, Davies, Oeppen and Schofield on the grounds that it depended largely on the behaviour of adult mortality, and failed to take sufficient account of countervailing trends in mortality at younger ages.\(^\text{17}\) Although Wrigley and his coauthors now accept that mortality change exerted a stronger influence on eighteenth-century population trends than they had previously thought, their latest findings reinforce their earlier view that the secular decline in English mortality began during the second half of the eighteenth century and continued into the first two decades of the nineteenth century. There was then an arrest of progress between the 1820s and 1860s, followed by a resumption of mortality decline from the late-1860s onwards.

In addition to these disputes over the course of mortality decline, there has also been a vigorous debate over its causes. In a series of publications, McKeown and his coauthors denied that either sanitary improvement or therapeutic intervention (in the form of smallpox inoculation) had any significant impact on mortality before 1800,\(^\text{18}\) but these conclusions have been strongly challenged by a number of
historians. Razzell and Mercer have argued that both inoculation and vaccination led to a sharp reduction in the incidence of smallpox mortality, and Dobson has shown that the draining of marshlands in Essex, Kent and Sussex helped to reduce mortality from malaria in the south-eastern corner of England between 1670 and 1800. Jones and Falkus have drawn attention to the various environmental improvements which took place in the market towns of southern England, and both Porter and Landers have demonstrated the importance of environmental improvement in London during the second half of the eighteenth century. However, this research does not mean that the influence of nutritional factors should be discounted altogether. Even though McKeown and his colleagues have often been accused of basing their arguments on inference rather than direct evidence, there is now a growing body of evidence which does suggest that nutritional factors may also have played a part in reducing mortality.

As we have already seen, there is now a growing debate among nutritionists as to whether it is possible to identify a ‘threshold’ below which individuals can be said to be sufficiently malnourished for the degree of malnutrition to result in increased susceptibility to infectious disease, and this debate has important implications for our efforts to examine the relationship between malnutrition and mortality among populations in the past. As Floud, Wachter and Gregory have shown, eighteenth-century Britons were both shorter and lighter than their modern equivalents, but this does not mean that their food requirements were necessarily lower. One of the most important factors which needs to be considered is the extent to which individuals were able to digest the nutrients they consumed. Dasgupta and Ray showed that individuals who are subjected to repeated bouts of diarrhoeal infection and whose diets contain large proportions of dietary fibre are only able to
digest approximately eighty per cent of the nutrients they consume, whilst conventional estimates of food adequacy tend to assume that around 95 per cent of nutrients will be digested.\textsuperscript{23} This implies that, just as conventional estimates of dietary need are likely to underestimate the food needs of deprived populations in the modern world, they are also likely to underestimate the food needs of populations in the past.

In addition to considering the adequacy of the standards used to establish dietary standards for individuals, it is also important to consider the distribution of food within populations. Livi-Bacci has argued that ‘a population which could rely on a normal consumption of 2000 calories per head would have been, in centuries past, an adequately-fed population, at least from the point of view of energy’,\textsuperscript{24} but this view takes little account of inequalities in the distribution of food within either households or populations.\textsuperscript{25} It is also difficult to reconcile Livi-Bacci’s view that nutritional deprivation was not an important factor in the high mortality of pre-industrial societies with his account of the role played by nutrition in the improvement of mortality in Britain after 1850. If one assumes that there was little or no relationship between nutrition and mortality in the eighteenth century, it is difficult to see how ‘improved nutrition certainly did play a positive role in increasing life expectancy’ during the second half of the nineteenth century,\textsuperscript{26} unless one is also prepared to argue that there was a significant reduction in the level of food consumption, or an increase in dietary needs, between the two dates.

In view of these arguments, it seems clear that a substantial proportion of the British population was inadequately fed during the first half of the eighteenth century, but it does not automatically follow that the subsequent reduction of mortality was caused by an improvement in the population’s diet. In the years following the
publication of McKeown’s original findings, both Wrigley and Schofield and Livi-Bacci argued that it was extremely unlikely that dietary standards would have improved during the second half of the eighteenth century because the average value of real wages was falling, but it is important to recognize the extent to which these conclusions are dependent on the accuracy and reliability of the price and wage indices used to calculate real wages. In 1998, Feinstein published new estimates of the value of real wages (or, to be more precise, real earnings) in England between 1770 and 1870 which went a long way towards reconciling some of the apparent contradictions between real wages and mortality. Previous accounts had suggested that real wages rose substantially during the first half of the eighteenth century, fell back between 1750 and circa 1810, and then rose sharply from the 1820s onwards. In contrast, Feinstein’s figures suggested that real earnings rose by 12.5 per cent in Great Britain between 1770/2 and 1818/22, and by 23.1 per cent between 1818/22 and 1848/52 (see Figure 1).

In view of these disagreements over the trend in real wages, it is not surprising that other historians have looked elsewhere for direct evidence of nutritional standards. Towards the end of the eighteenth century, Davies and Eden presented information about the consumption patterns of 213 labourers’ households, and these data have been analysed by modern historians to yield new estimates of the nutritional value of the diets available to labourers’ families in the preindustrial period. In 1995, Clark, Huberman and Lindert estimated that the average value of the diet consumed by the typical Davies-Eden household was equivalent to 1508
calories per person per day and 27.9 grammes of protein per person per day, whilst Shammas, using a restricted sample of 22 Davies-Eden households, estimated that mean daily consumption was equal to 1734 calories per person in the south of England, and 2352 calories per person in the north.\textsuperscript{29} It is not entirely clear why these estimates should differ, and neither set of figures includes any allowance for the food which was provided for individuals at work or which they were able to produce for themselves, but it still seems clear that the overall figures are very low. In her 1984 article, Shammas concluded that ‘the south’s 2100-2500 [calories per day per adult equivalent] would not seem to provide the energy for hard labour or growth in children and the north’s 2800-3200 was, at best, barely adequate.’\textsuperscript{30}

One of the greatest challenges posed by the analysis of dietary information in the past in the difficulty of knowing exactly how the groups represented in any one survey should be compared with the population as a whole. As we have already seen, Clark, Huberman and Lindert believed that the value of the diets consumed by the Davies-Eden households was very low, but they also believed that these households were drawn from the very poorest sections of the population. By contrast, Shammas does not appear to have regarded the families in her sample as being especially poor, and Fogel has argued that they were in fact close to the median for the population as a whole.\textsuperscript{31} In 1989, Fogel used the information provided by Shammas to estimate the distribution of food consumption among the population as a whole in 1790. He estimated that the average value of the diets consumed by the poorest decile of the population was equivalent to 1545 calories per adult equivalent per day, whilst the bottom twenty per cent of the population still consumed less than 2000 calories per adult equivalent per day. By contrast, the
richest decile of the population consumed a diet with an average daily value of more than 4000 calories per adult equivalent (or ‘consuming unit’).  

In addition to estimating the level of food consumption at the end of the eighteenth century, it is also important to obtain some impression of the likely trends in consumption over time. Shammas argued that the nutritional value of the average diet may have declined because the proportion of bought foods increased and the consumption of milk and dairy products declined. By contrast, Fogel has estimated that the nutritional quality of the average diet increased between 1750 and 1800 (because of an increase in the proportion of calories derived from meat products), and that the average calorific value rose by just under seven per cent between 1700 and 1800, and by a further 5.6 per cent between 1800 and 1850. Nevertheless, even in 1850, the average British consumer only consumed approximately 75 per cent of the calories consumed by his or her successors at the end of the 1980s.

As the above paragraphs have demonstrated, it is difficult to reach any categorical conclusions regarding the main trends in dietary standards before 1800, but it seems reasonably clear that the average level of nutrition was low and that a substantial proportion of the population subsisted on diets which fell significantly below the standards recommended by modern nutritional experts. The main area of uncertainty concerns the question of change. As we have already seen, there are some grounds for believing that the quality of the average diet may have declined, but other evidence suggests that the proportion of meat may have increased and that overall energy levels may also have risen, even though the extent of any increase was not very great, and the value of the food consumed remained very low. Thus, even if the decline of mortality between 1780 and 1820 was related to
improvements in nutrition, the limited nature of these improvements may help to explain why mortality rates only fell at the rate they did.

Although there is a considerable body of evidence to show that the poorer members of the population were clearly undernourished by modern standards, it is rather more difficult to argue that the high mortality rates experienced by the better-off were related to malnutrition. As we can see from Figure 3, there was very little difference between the life expectancy of aristocratic infants and that of the population at large before the early years of the eighteenth century, and even during the eighteenth and nineteenth centuries, members of the aristocracy experienced life expectancies which fell well below modern standards. These facts have led Razzell and Livi-Bacci to question the whole basis of the argument that the decline in mortality as a whole can be related to improvements in nutrition. 35

In The modern rise of population, McKeown suggested that even though the aristocracy were not themselves malnourished, they were affected by the presence of malnutrition among the population at large. As we have already seen, modern epidemiologists believe that the presence of severe malnutrition can render a population more susceptible to infection, as well as impeding their prospects of recovery, and McKeown argued that the health of the aristocracy was undermined because the poor nutritional status of the general population increased the incidence of infection in the population as a whole. However, he was unable to explain why the improvement in nutrition which was central to his explanation for the decline of aristocratic mortality should only have led, in the first instance, to an increase in the
average life expectancy of the better off, without any immediate or equivalent impact on the life expectancy of the poor.\textsuperscript{36}

In view of these problems, it is difficult to argue that the decline in aristocratic mortality was initiated by a general improvement in nutrition, but this does not mean that nutrition was unimportant overall. As we have already seen, Scrimshaw, Gordon and Taylor suggested that nutritional factors were only likely to exert a decisive influence on the outcome of an infectious disease in situations where there was an epidemiological balance between the natural resistance of the host and the virulence of the infective organism, and this may help to explain why nutritional factors do not appear to have played a leading role in the determination of mortality before \textit{circa} 1750.\textsuperscript{37} Kunitz and Engerman argued that during the sixteenth and seventeenth centuries, the major causes of premature death were epidemic or pandemic diseases, but these either became less important (in the case of plague) or more endemic (in the case of smallpox) during the course of the eighteenth century, and this allowed social factors, such as differences in hygiene, domestic arrangements and nutritional status, to become more important as the century progressed.\textsuperscript{38} Consequently, the real significance of the ‘peerage paradox’ is not that it demonstrates the irrelevance of nutritional explanations for mortality decline, but, rather, that it demonstrates the need for a more sophisticated understanding of the relationship between these factors and the changing nature of the disease environment.
Although there are good grounds for believing that nutritional factors were involved in the decline of mortality before 1820, it is rather more difficult to argue that the cessation of progress which followed this period was caused by a deterioration in nutritional standards between circa 1820 and 1850 (or even 1870), and there are even grounds for believing that nutritional standards may even have risen. However, the most obvious explanation for the cessation of progress between 1820 and 1850 is that the beneficial effects of any improvement in nutrition were undermined by the impact of urbanization on the nature of the disease environment in which a growing proportion of the population lived.

During the last century, few debates in economic history have attracted more attention, or generated more controversy, than the debate over the ‘standard of living’ during the industrial revolution. In 1816, the government statistician, John Rickman chastised his friend, Robert Southey, for entertaining the belief that ‘[the] state [of the poor] has grown worse and worse of late’, and in 1926 Sir John Clapham pointed out that, according to the latest estimates, ‘the purchasing power of wages in general – not, of course, everyone’s wages’ was definitely increasing between 1820 and 1850. However, in 1930 the socialist historian, J. L. Hammond retorted that even though the industrial revolution may have made life easier and more comfortable for thousands of men and women, ‘the ugliness of the new life, with its growing slums, its lack of beautiful buildings, its destruction of nature and its disregard of man’s deeper needs, affected not this or that class of worker only, but the entire working class population’.
During the following decades, a new generation of economic historians tried to challenge Clapham on his own territory. In 1957, Eric Hobsbawm criticized the evidence which Clapham had used to measure real wages, and contrasted this with the available evidence on food consumption. He argued that there were declines in the per capita consumption of wheat, milk and cheese, and that the consumption of meat also declined, principally because the number of cattle and sheep slaughtered at London’s Smithfield Market failed to keep pace with the growth of the capital’s population. However, many of these claims were strongly rebutted by Max Hartwell in 1961. In particular, he argued that there was no firm evidence for the view that grain consumption declined, and he challenged Hobsbawm’s arguments about meat consumption by demonstrating that it was impossible to generalize from the experience of Smithfield market at a time when London’s other meat markets were growing much more rapidly.

The difficulties associated with the measurement of trends in the consumption of domestically-produced goods have prompted a number of writers to focus rather more attention on the consumption of imported items, such as tea and sugar. Burnett argued that the consumption of sugar fell between 1811 and 1821, and did not begin to rise significantly before the mid-1840s, whilst the consumption of tea (which he regarded as a much truer indicator of standards) only began to increase in the 1830s. Mokyr used similar information to estimate changes in real wages overall. He concluded that the average level of real wages remained largely unchanged in the three decades following the end of the Napoleonic Wars, and only began to improve substantially from the mid-1840s onwards.

These findings highlight the need for caution in assessing changes in real wages, but most economic historians have continued to argue that the average level
of real wages did increase during the first half of the nineteenth century, and the main areas of contention concern the question of when they began to increase and by how much. As we have already seen, many commentators, including Wrigley and Schofield, Lindert and Williamson, Crafts and Schwarz, believed that real wages stagnated or fell during the second half of the eighteenth century, before rising sharply from the early-1800s onwards, but Feinstein’s more recent calculations suggest that these authors may have been wrong to suppose that real earnings were falling in the earlier period, and that they may have exaggerated the extent to which they increased in the later period. In his article, Feinstein tended to focus most attention on the movement of real earnings after 1820, and it was for this reason that his findings appeared to lend most support to the ‘pessimist’ case. However, it is worth noting that even his revised figures implied that real wages rose by 0.7 per cent per year between 1818/22 and 1848/52, and by 23.15 per cent over the whole period.

Although it is widely accepted that real wages did increase, on average, over the course of this period, it does not necessarily follow that aggregate levels of food consumption should have risen as a consequence. Clark, Huberman and Lindert attributed part of the gap between the available estimates of food consumption and their own estimates of the increase in the level of real wages to the fact that urban and industrial occupations tended to be less energy-intensive than traditional rural occupations, and to the possibility that urban workers ‘chose’ to consume a less nutritious diet. However, as Feinstein pointed out, much of the ‘food puzzle’ identified by Clark, Huberman and Lindert was based on their own, possibly inflated, estimates of the growth in real earnings. If the growth in real earnings was, as he
believed, significantly lower than they argued, then the food puzzle, as such, would largely disappear.\textsuperscript{50}

In addition to examining questions of taste and income elasticity, economic historians have also focused attention on the question of relative prices. Komlos has suggested that the quality of the average diet may have deteriorated because increases in the relative price of meat encouraged consumers to transfer expenditure from meat and dairy products to grains, but it is not entirely clear how far the available data support his view.\textsuperscript{51} He appears to have based his interpretation on Horrell’s study of home demand in Britain during the industrial revolution, but her data suggest that the proportion of expenditure devoted to meat and dairy products rose by seventeen per cent between 1801 and 1841, and Feinstein’s data suggest that the share of total expenditure devoted to meat and dairy products remained unchanged between 1828/32 and 1858/62.\textsuperscript{52}

In view of these findings, it is unlikely that we can attribute a deterioration in the quality of the working-class diet to changes in the relative price of meat and dairy products, but other writers have suggested that the demand for food may have been affected by changes in the price of industrial goods.\textsuperscript{53} It is also worth noting that even though food prices were falling between 1820 and 1850, the cost of housing increased, and it is possible that workers may have attempted to recoup some of their additional housing costs by economising on the purchase of food.\textsuperscript{54} However, even though the proportion of working-class expenditure devoted to food fell between 1828/32 and 1858/62, the extent of this reduction was outweighed by the overall increase in the real value of working-class earnings.\textsuperscript{55} Consequently, even though working-class families devoted a smaller proportion of their overall budget to the purchase of food, the increase in the real value of their earnings meant that that
the amount of food which they were able to purchase was more likely to have increased than decreased. 56

As a result of these calculations, it seems unlikely that the amount of food consumed by the wage-earning population of Britain declined between 1820 and 1850, and there are clear grounds for believing that it ought to have increased, even though the extent of this increase was probably small, and may not necessarily have been reflected in the aggregate accounts. This conclusion is reinforced by an examination of the most recently-published analyses of eighteenth- and nineteenth-century household budgets, but these also need to be treated with care. During the 1990s, both Oddy and Clark, Huberman and Lindert estimated the average nutritional value of the food consumed by working-class families at different points in time between the end of the eighteenth century and the middle of the nineteenth century, but there are significant differences between the results which they reported and those reported by Shammas, even though all their findings were derived from substantially the same sources. 57 Nevertheless, all three sets of results still suggest that there were significant improvements in both the quantity and quality of the diets consumed by poor English workers, and their families, during this period (see Table 1). 58

Table 1 about here

These findings make it unlikely that the cessation of mortality decline after 1820 can be attributed, to any significant degree, to a deterioration in the level of nutrition, but it may be attributable to a change in the nature of the disease environment in which a growing proportion of the population lived. During the period
between 1800 and 1850, several commentators drew attention to the high death rates, associated with urban areas, and these fears were compounded by the pace at which the size of the urban population was increasing. As Wohl has pointed out, the number of towns and cities containing more than 100,000 inhabitants increased from one to eight between 1801 and 1851, and by the end of the period the population of London had risen to more than one million. During the period as a whole, the proportion of the population residing in towns containing more than 10,000 inhabitants rose from 24 per cent to 44 per cent, and the proportion of the population residing in towns with more than 100,000 inhabitants rose from eleven per cent to 25 per cent. Between 1801 and 1851, the population of Manchester increased from 75,000 people to 303,000, whilst the population of Liverpool rose from 82,000 to 376,000. The population of Bradford rose from 13,000 to 104,000.

During the last two decades, a number of authors have argued that urban conditions were not only less healthy than rural conditions, but that conditions within towns may have grown worse. Huck showed that there was a significant increase in the level of infant mortality in nine urban parishes in central and northern England between 1813/18 and 1831/6, and evidence from four of these parishes (Walsall, West Bromwich, Wigan and Ashton) suggested that this deterioration was unlikely to have been reversed, and may even have continued, into the 1840s. This evidence is supported by Floud, Wachter and Gregory’s account of changes in the average height of British army recruits who were born in different parts of Britain between the 1820s and 1850s. They found that there was a substantial deterioration in the average heights of men born in London and other urban centres, and that this was at least partly responsible for the decline in the average height of the male population as a whole during this period.
Although there is strong evidence of a deterioration in infant mortality and in stature, it has proved rather more difficult to establish whether or not there was any deterioration in mortality rates as a whole within urban areas. In 1985, Woods used data for 1861 and 1911 to estimate the main trends in mortality in four different types of area between 1811 and 1911. He suggested that average life expectancy at birth rose in all parts of Britain during the first half of the nineteenth century, including rural areas, small towns, large provincial towns and the capital, and that the absence of any clear improvement in national mortality rates was a direct result of changes in the proportions of the population inhabiting different types of districts but Szreter and Mooney have argued that average life expectancy at birth declined sharply in the largest provincial cities during the 1830s, and only began to improve consistently from the 1870s. However, as Woods himself has pointed out, Szreter and Mooney’s figures for the early part of the nineteenth century were largely (though not entirely) dependent on the evidence for Glasgow, and even if one accepts the reliability of the Glasgow data, it is still open to question how far they can be used to estimate the main trends in mortality in cities as diverse as Bradford, Newcastle, Sheffield, Bristol, Leeds, Birmingham, Manchester and Liverpool.

In view of the obvious dangers associated with these figures, it is clearly advisable to treat any estimates regarding the pattern of mortality change in different parts of the country in the early part of the nineteenth century with a high degree of caution, but some points are beginning to become a little more clear. Although Woods has questioned the validity of Szreter and Mooney’s overall conclusions, he has also revised his own estimates of changes in life expectancy in the different types of area, and he now believes that average life expectancy may have declined after all in both large and small towns between the 1830s and 1850s. However,
whilst this represents an important concession to supporters of a more pessimistic interpretation of the impact of urbanization, it also raises other questions about the pattern of mortality change in the country as a whole. As we can see from Table 2, the new estimates suggest that the average level of life expectancy at birth in towns containing between 10 and 100,000 inhabitants fell by 0.3 years during the 1840s, but this figure may not have been true of all these areas. In their paper, Szreter and Mooney emphasized the fact that it was not the size of population *per se* which mattered, but the rate at which the population was increasing, and this suggests that even though many smaller and medium-sized towns experienced a deterioration in their standards of public health, others may have witnessed some improvement, and this impression is reinforced by the fact that the average level of life expectancy in rural areas also appears to have increased.⁶⁹

Woods’ figures also raise questions about the pattern of mortality changes in the country as a whole. Szreter and Mooney argued that there was a catastrophic fall in life expectancy in the largest provincial cities during the 1830s, and even though their figures suggested that the average level of life expectancy rose by four years during the 1850s, they nevertheless concluded that there was no substantial increase in life expectancy before the 1870s. By contrast, Woods’ figures point to a somewhat smaller reduction in average life expectancy in the 1830s and 1840s, followed by a small but not insignificant improvement from the 1850s onwards. These figures suggest that the improvement in urban life expectancy cannot simply be attributed to sanitary intervention, since even Szreter concedes that there is little evidence of effective sanitary intervention in these areas before 1870, and Bell and Millward have argued that sanitary intervention is unlikely to have made much difference to mortality rates before the 1890s or even the early-1900s.⁷⁰
These calculations suggest that even though urbanization was undoubtedly the most important influence on Britain’s mortality rate during this period, it was not the only influence. As we have already seen, the evidence of real wages, expenditure patterns and food consumption suggests that dietary standards were also improving, and this may help to explain why mortality rates continued to improve within certain types of area, even though they failed to improve in the country as a whole. This interpretation is reinforced by Floud, Wachter and Gregory’s analysis of changes in the average height of army recruits, which began to improve with the birth cohorts of the 1850s. Although they recognized that diet was only one of the factors which was likely to influence stature, they nevertheless concluded that ‘the height data make the link between nutrition (although in a wider sense) and mortality which McKeown could only infer’. 71

Floud, Wachter and Gregory’s findings also raise questions about the timing of the factors which determine both childhood and adult mortality. In 1934, Kermack, McKendrick and McKinlay highlighted ‘certain regularities in the vital statistics of Britain’, 72 in which each cohort or generation appeared to experience a consistently lower level of mortality throughout the life course than its predecessor, and this has led many subsequent researchers to devote much more attention to the impact of cohort-specific factors on mortality and to the ways in which nutritional and environmental influences in early life can influence mortality at later ages. 73 These investigations certainly raise important new questions about the pattern of mortality change in the second and third quarters of the nineteenth century. 74
Although the McKeown hypothesis was concerned with the whole of the period from 1700 onwards, much of the fiercest debate has focused on the critical period between 1850 and 1914. In 1962, McKeown and Record examined changes in the main causes of death and concluded that ‘it seems unquestionable that the decline of mortality between 1851/60 and 1891/1900 was attributable almost exclusively to a reduction in the frequency of death from infectious disease’. In 1976, McKeown argued that 33.30 per cent of the decline in mortality between 1848/54 and 1901 was caused by a decline in the incidence of deaths from water- and food-borne diseases, whilst 43.63 per cent of the decline was caused by a decline in the frequency of deaths from airborne diseases. He argued that a substantial proportion of the decline in the incidence of deaths from water- and food-borne diseases was caused by improvements in hygiene, but that there was little evidence to show that environmental improvements had any substantial effect on exposure to airborne diseases before the start of the twentieth century. He therefore concluded that the decline in the death rate associated with these conditions (respiratory tuberculosis; bronchitis, pneumonia and influenza; whooping cough; measles; scarlet fever and diphtheria; smallpox; and infections of the ear, pharynx and larynx) was most likely to have been caused by an improvement in the population’s capacity to resist infection, which was itself the result of improvements in nutrition.

During the last two decades, several historians have attempted to cast doubt on the form of McKeown’s argument and on the conclusions he drew from it. One of
the most intriguing features of the argument concerns the treatment of the decline
in the number of deaths attributable to scarlet fever. In 1962, McKeown and Record
argued that ‘scarlet fever was responsible for about nineteen per cent of the
reduction of mortality during the second half of the nineteenth century’ and that ‘there
is no reason to differ from the general opinion that [the decline in the number of
deaths attributed to scarlet fever] … resulted from a change in the nature of the
disease’, but in 1976 McKeown included scarlet fever and diphtheria in the
category of airborne diseases whose decline he attributed to an improvement in
nutrition. If the decline in the number of deaths attributed to scarlet fever and
diphtheria had been excluded from this category, then the overall contribution of the
remaining airborne diseases to the overall decline in mortality would have fallen from
43.63 per cent to 31.20 per cent – i.e. less than the figure associated with the decline
in the incidence of mortality from water- and food-borne diseases (see Table 3
below).

Despite the obvious importance of scarlet fever to the construction of
McKeown’s argument, his critics have often tended to focus rather more attention on
the question of tuberculosis. In 1988, Szreter argued that McKeown had
exaggerated the extent of the contribution made by pulmonary tuberculosis to the
overall decline in mortality, and that this had led him to underestimate the
significance of improvements in the incidence of mortality from water- and food-
borne diseases. However Szreter’s own interpretation of the tuberculosis statistics
has also been challenged, and he subsequently conceded that ‘the recorded fall in
respiratory tuberculosis was probably genuine, [even though] it also confirms the full
extent of the contradictory rise in bronchitis/pneumonia/flu’. This conclusion has
since been reinforced by Woods’ analysis of the cause-of-death data in the
Registrar-General’s Decennial Supplements for the period 1861/70 to 1891/1900. Although Woods’ figures cover a slightly different period to those employed by McKeown, they are broadly consistent with his original interpretation (see Table 3).

Table 3 about here

One of the most interesting attempts to account for the decline of mortality in the latter part of the nineteenth century in recent years has come from attempts to relate the history of mortality to that of morbidity. Riley has argued that one of the reasons for the decline of mortality was that improvements in medical attendance had enabled sick people to manage their diseases (and, especially, respiratory and organ diseases) in such a way as to enable them to survive for longer and postpone mortality. However, much of the increase in the duration of sickness episodes on which Riley based his case may have been caused by increases in the average age, and changes in the age distribution, of the population who submitted sickness claims. It is difficult to know how far this may have been true of the friendly society members whose health Riley investigated, but it certainly appears to provide the most likely explanation for changes in the pattern of morbidity experienced by members of the Hampshire Friendly Society, in the south of England, over the same period.

Although Riley has raised important new questions about the effects of medical attendance and the relationship between mortality and morbidity, his work offers a much less sustained critique of the McKeown thesis than Woods’ exhaustive analysis of the main changes in causes of death in each of Britain’s 614 registration districts between the 1860s and the 1890s. Woods’ most striking finding was that
even though the decline in the death rate from pulmonary tuberculosis accounted for more than one-third of the total decline in mortality during this period, it appeared to decline at much the same rate in all parts of the country. He therefore concluded that this decline was unlikely to have been caused by changes in either diet or environmental conditions, and that the most likely explanation was a change in virulence of the infective organism.\textsuperscript{83} However, this argument has not been accepted uncritically. Landers thought it was ‘rather unlikely’ that ‘tuberculosis underwent a spontaneous reduction in virulence … given the apparent evolutionary age and stability of the tubercle bacillus’, and Szreter noted that Woods’ discussion of the main trends in tuberculosis was ‘uncharacteristically lacking’ in references to the most recent medical research.\textsuperscript{84}

In view of these criticisms, it is worth looking more closely at recent studies of the recrudescence of tuberculosis in Britain since the beginning of the 1980s. Spence \textit{et al.} found that there was ‘a strong relation … between notification rates of tuberculosis and poverty in the last decades of the twentieth century’ and Bhatti \textit{et al.} argued that ‘the evidence favours a major role for socio-economic factors that affect all residents in accounting for the increase in tuberculosis in poor districts’. Although these authors recognized that poverty was ‘multifaceted’, they also argued that it was most likely to influence levels of tuberculosis through its association with poor diet and overcrowding.\textsuperscript{85} Spence \textit{et al.} concluded that ‘where social deprivation is rife, particularly in areas of poor housing, high unemployment, and low incomes, an increased awareness of tuberculosis as a possible diagnosis is necessary. Far from diminishing, tuberculosis both worldwide and in the United Kingdom is increasing. Poverty may be a factor causing this increase’.\textsuperscript{86}
During the last three decades, a number of researchers have examined the relationship between housing standards and tuberculosis mortality in the latter part of the nineteenth century. Pooley and Pooley found that housing density accounted for only seventeen per cent of the variance in crude mortality rates in Manchester between 1871 and 1875,\(^8\) and Vögele argued that there was surprisingly little relationship between housing and population density and either all-cause mortality or tuberculosis mortality in the country as a whole.\(^8\) Moreover, both Vögele and McKeown believed that there was comparatively little evidence of any substantial improvement in housing conditions before the start of the twentieth century.\(^8\)

However, other historians have challenged this view. Daunton suggested that the introduction of wallpaper and linoleum in working-class homes during the second half of the nineteenth century ‘possibly contributed to the reduction in tuberculosis’,\(^9\) and Cronjé argued that there was a considerable amount of evidence to show that ‘housing improved considerably in the second half of the nineteenth century, with a rise in the quality of new building, a reduction in excessive overcrowding, and increased government regulation bringing better sanitation and water supply’.\(^9\)

In view of the conflicting nature of these statements, it is worth looking more closely at the extent of housing change during this period. As many historians have pointed out, a very large number of working-class families continued to inhabit ill-ventilated and overcrowded accommodation at the end of the nineteenth century, and this may well have contributed to the persistence of high rates of tuberculosis mortality at the end of the century.\(^9\) However, this does not mean that there had been no improvement in housing conditions before this point. As Daunton has shown, the substantial increase in the value of real wages after 1850 enabled a significant proportion of working-class families to pay higher rents in return for
improved accommodation, and this was reinforced by the introduction of new building bye-laws which helped to establish higher standards for the construction of new houses from the 1870s onwards. Consequently, even though it may be difficult to establish a precise relationship between housing improvement and mortality decline, there are strong circumstantial grounds for believing that these improvements did make an important contribution to the decline of tuberculosis (and, possibly, other diseases) after 1850.

Although many historians might be prepared to accept the view that housing conditions did play a part in the decline of tuberculosis mortality, the relationship between diet and mortality has proved even more contentious. Hardy has claimed that ‘recent research … reinforces the conclusion that, for a significant proportion of Britain’s population, rising real incomes had little direct impact on improving nutritional standards’, but the evidence for this is far from clear. The statement is apparently based on the third edition of Burnett’s classic study of Plenty and Want, but Burnett himself argued that the period between 1873 and 1896 ‘brought increased purchasing power … and a bigger margin which could go towards providing a better and more varied diet’ and that ‘improvements are observable in the general standard of the working-class diet’ during the last quarter of the nineteenth century.

In view of the apparently contradictory nature of these statements, it is clearly worth looking more closely at some of the available evidence on food consumption during this period. Oddy estimated that the calorific value of the food consumed each day by the average working-class consumer fell by 360 calories between the 1860s and the 1890s, but he also argued that quality of the diet improved, and that this was reflected in the reduced consumption of bread and an increase in the
consumption of meat and sugar. Oddy’s emphasis on the quality of the average diet was supported by Dewey’s analysis of changes in food consumption between 1880 and 1954. He argued that ‘well before 1914, consumption had begun to shift away from “inferior” foods (cereals, potatoes) towards “superior” ones (mainly meat and dairy produce). This process reflected mainly the rise in average real income which became apparent from about 1850, but it was also related to the greater availability of certain foods and in some, but not all, cases, a lowering of the price of individual commodities.’

Although this paper has been particularly concerned with the impact of nutritional factors on the decline of mortality, it is also important to consider the role played by sanitary improvements, especially after 1870. As we have already seen, McKeown believed that up to one-third of the total decline in mortality between 1848/54 and 1901 was associated with a decline in the incidence of mortality from water- and food-borne diseases, and that a substantial part of this decline could be explained by improvements in the sanitary condition of the urban environment. Szreter believed that it was no accident that the onset of more rapid mortality decline after 1870 should have coincided with an increase in the level of local public health activity, and Hardy has argued that ‘the problems of nineteenth century cities called for a systematic and professional administrative response, which was provided with the establishment of sanitary authorities and, more especially, of Medical Officers of Health in London in 1855 and elsewhere in 1872…. It was [the Medical Officers of Health] … who spearheaded the Victorian struggle against infectious disease, and it was their initiative and their labours which led to the eventual eradication of the epidemic streets.’

In view of the importance of arguments about the effectiveness of social intervention, it is worth examining this question in a little more detail. In his recent book, Robert Woods argued that fourteen of the 614 registration districts in England and Wales (containing thirteen per cent of the total population) were responsible for 25 per cent of the national decline in the number of deaths from diarrhoea and typhus, but these two diseases were responsible for more than 28 per cent of the overall decline of mortality in the country as a whole. These findings demonstrated the importance of sanitary intervention and showed that ‘if sufficient progress could be made in sanitary reform in even a small number of the most populous places its effects might outweigh more substantial advances in places with smaller populations, such as the small towns’. However, even though Woods was anxious to emphasize the importance of sanitary reform, especially when compared to the effects of any possible nutritional improvements, he also warned against placing excessive reliance on what he called ‘simple conclusions’. One of the most important problems for supporters of a ‘public health’ explanation for mortality change is the persistence of high rates of infant mortality before 1900. Even though the aggregate rate of mortality from diarrhoeal diseases fell by 23.25 per cent between 1861/70 and 1891/1900, the rate of diarrhoeal mortality among infants only began to decline after the turn of the century.

Although it would clearly be wrong to underestimate the importance of sanitary intervention, it is also important to ask why its impact on mortality should not have been greater. In 1998, Frances Bell and Robert Millward pointed out that most of the initial increase in sanitary expenditure was concerned with the provision of better water supplies, but such measures were often counterproductive because the authorities failed to make any equivalent investment in sewerage facilities.
argued that it was only in the 1890s that many local authorities began to make significant investments in the full range of sanitary measures and that, consequently, it was only after this date that the ‘sanitary revolution’ really began to take hold. As a result, although many local authorities began to invest more heavily in their sanitary infrastructure from the 1870s onwards, it was not until the first decade of the twentieth century that these efforts began to exercise a decisive effect on the decline of mortality in the country as a whole.104

Conclusions

In some respects, this paper may be said to have raised more questions than answers. During the last three decades, McKeown’s critics have often accused him of failing to provide any direct evidence to justify the weight he attached to nutritional factors in his interpretation of the onset of mortality decline. This paper has shown that such evidence may exist, but it remains difficult to attach precise quantitative estimates to the impact of different determinants at different points in time.

Although the paper has, therefore, offered a qualified defence of McKeown’s work, it is also important to acknowledge some particular weaknesses. One of the most important problems with McKeown’s work is his failure to distinguish between the concepts of nutrition (or diet) and nutritional status. In his work, he argued that a large part of the decline of mortality between 1700 and 1914 must be attributable to improvements in diet because it could not be explained by changes in any of the other ‘obvious’ causes, but this ignores the extent to which the adequacy of a person’s diet may also be affected by changes in their epidemiological environment
and in the nature and amount of the work they are expected to perform. It is for this reason that many students of this question have tended to prefer the term ‘nutritional status’. Even though some critics may feel that this concept tends to blur the boundaries between dietary issues and epidemiological issues, it also reflects a much better understanding of the synergistic relationship between nutrition and infection on which so many aspects of human health depend.¹⁰⁵

The distinction between nutrition and nutritional status is particularly important when one considers the way in which McKeown attempted to draw a clear line between the different types of disease and the factors which may (or may not) have contributed to their development. Whilst a number of writers, including Szreter, have drawn attention to the inconsistencies which existed between the diseases which McKeown listed in the same categories (such as, for example, the differences between the trends in respiratory tuberculosis and bronchitis), it is also important to recognize the synergies which may exist between diseases in different categories. For example, in his work McKeown drew a clear distinction between water- and food-borne diseases such as diarrhoea, which were amenable to the effects of sanitary intervention, and airborne diseases, such as respiratory tuberculosis, whose decline could only have come about through improvements in nutrition, but this ignores the extent to which reductions in the incidence of water- and food-borne diseases may have contributed to the decline in nutrition-based diseases by improving the population’s capacity to absorb essential nutrients.¹⁰⁶

It is also arguable that McKeown failed to pay sufficient attention to the way in which urbanization affected the nature of the disease environment within which people lived. As we have seen, there was a substantial increase in the proportion of the population which was exposed to urban influences in the first half of the
nineteenth century, and it is highly likely that the urban environment itself also
deteriorated, even if this was not true of the entire country. Consequently, even
though there are grounds for believing that the general standard of nutrition may
have improved, it is difficult to deny that the expansion of urban conditions either
created or exacerbated a public health problem which could only be addressed
properly by effective sanitary intervention.

Although McKeown emphasized the importance of improvements in the
‘standard of living’ in the second half of the nineteenth century, it is arguable that he
interpreted the consequences of this improvement rather too narrowly. In The
modern rise of population, he tended to argue that improvements in mortality could
only come about because of a change in the biological relationship between
organism and host, or because of therapeutic improvements, or as a result of
sanitary intervention, or because of improvements in the standard of nutrition, and it
is therefore arguable that he failed to pay sufficient attention to the ways in which
increases in the value of real wages also enabled people to purchase better housing.
This factor has sometimes appeared to be obscured by an emphasis on the role of
housing legislation (though the impact of this should not be neglected), and it seems
particularly relevant to the analysis of changes in the incidence of mortality from
tuberculosis.

In his famous critique of McKeown, Szreter claimed that one of the major
weaknesses of the ‘McKeown thesis’ was the extent to which it appeared to offer
support to ‘the extreme laissez-faire position that health and welfare gains may be
generated most effectively as a by-product of economic growth’, but this criticism
is surely misplaced. Even if it could be shown that McKeown had intended to offer
his analysis as part of an argument for the promotion of free-market economics, the
fact remains that the improvements which did occur in Britain’s health were not
that great, as the recruiting officers who surveyed the physical state of army
volunteers discovered to their cost in 1914. It would therefore be wrong to
assume that the slow and tortuous path which Britain took towards better health in
the eighteenth and nineteenth centuries represents a model to which other countries
should now aspire at the start of the twenty-first century.

Acknowledgements

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University of Birmingham, on 21 September 2002, and has also been presented in
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participants in these meetings for their helpful comments. I am also grateful to Javier
Birchenall, Claudia Edwards, Robert Fogel, Andrew Hinde, Leonard Schwarz, Simon
Szreter, Robert Woods and Tony Wrigley for their comments on written versions of
the paper.
Table 1. Average daily consumption of calories and proteins per person, 1787-1863.

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<th>1787-96S</th>
<th>1787-96N</th>
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Notes.

a. Shammas’ figures were derived from Davies’ study of the diets of rural families between 1787 and 1793, and from Eden’s study of the diets of urban and rural families in 1796. She based her results on the analysis of 7 northern families (1787-96N) and 15 southern families (1787-96S).

b. Oddy’s figures for the eighteenth century were derived from the same Davies and Eden studies as Shammas’ figures. However, he based his findings on the analysis of 32 families from the Eden study (mainly northern labourers) and 119 families from the Davies study (mainly southern families). His figures for 1837-41 were based on William Neild’s study of the diets of Manchester and Dukinfield and ‘several other contemporary budgets’. The figures for 1863 are based on the information contained in Edward Smith’s survey for the Medical Department of the Privy Council.

c. Clark, Huberman and Lindert based their figures for 1787-96 on 195 households in the Davies-Eden surveys. Their analysis of the 1841 data was based on the figures supplied by Neild for Manchester and Dukinfield, Purdy (for agricultural labourers) and two Parliamentary commissions on mining and child labour. Their figures for 1863 were also derived from Edward Smith’s surveys.

Table 2. Average life expectancy at birth in different parts of England and Wales, 1801-1900.

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<th>1831</th>
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<td>32</td>
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<td>44</td>
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<tr>
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<td>30</td>
<td>31</td>
<td>32</td>
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<td>35</td>
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<td>39</td>
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<td>Large provincial towns</td>
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<td>30</td>
<td>34</td>
<td>34</td>
<td>38</td>
<td>40</td>
<td>42</td>
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<tr>
<td>Other centres</td>
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<td>41</td>
<td>42</td>
<td>42</td>
<td>42</td>
<td>42</td>
<td>44</td>
<td>47</td>
<td>47</td>
</tr>
<tr>
<td>England and Wales</td>
<td>40</td>
<td>41</td>
<td>41</td>
<td>41</td>
<td>41</td>
<td>41</td>
<td>43</td>
<td>45</td>
<td>45</td>
<td>46</td>
</tr>
</tbody>
</table>

<table>
<thead>
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<tbody>
<tr>
<td>Large provincial towns</td>
<td>32.0</td>
<td>32.5</td>
<td>32.7</td>
<td>32.6</td>
<td>32.0</td>
<td>32.3</td>
<td>33.0</td>
<td>36.6</td>
<td>39.0</td>
<td>39.6</td>
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<tr>
<td>Other centres</td>
<td>40.4</td>
<td>41.4</td>
<td>41.6</td>
<td>41.4</td>
<td>41.3</td>
<td>42.4</td>
<td>42.9</td>
<td>44.6</td>
<td>47.2</td>
<td>48.6</td>
</tr>
<tr>
<td>England and Wales</td>
<td>40.3</td>
<td>41.1</td>
<td>41.1</td>
<td>40.7</td>
<td>40.4</td>
<td>41.1</td>
<td>41.2</td>
<td>43.0</td>
<td>45.3</td>
<td>46.1</td>
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</tbody>
</table>

Table 3. The contribution made by different diseases to the decline of mortality in England and Wales during the second half of the nineteenth century.

<table>
<thead>
<tr>
<th></th>
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<tbody>
<tr>
<td></td>
<td>1851/60</td>
<td>1891/1900</td>
<td>1861-70</td>
</tr>
<tr>
<td></td>
<td>Deaths per million</td>
<td>Deaths per million</td>
<td>%</td>
</tr>
<tr>
<td>Phthisis/Respiratory tuberculosis</td>
<td>2,772</td>
<td>1,418</td>
<td>43.89</td>
</tr>
<tr>
<td>Bronchitis, pneumonia and influenza</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Diseases of the lung/respiratory system</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Whooping cough</td>
<td>433</td>
<td>363</td>
<td>2.27</td>
</tr>
<tr>
<td>Measles</td>
<td>357</td>
<td>398</td>
<td>-1.33</td>
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<tr>
<td>Scarlet fever</td>
<td>779</td>
<td>152</td>
<td>20.32</td>
</tr>
<tr>
<td>Diphtheria</td>
<td>99</td>
<td>254</td>
<td>-5.02</td>
</tr>
<tr>
<td>Scarlet fever &amp; diphtheria</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Smallpox</td>
<td>202</td>
<td>13</td>
<td>6.13</td>
</tr>
<tr>
<td>Infections of the ear, pharynx and larynx</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Cholera, diarrhoea, dysentery</td>
<td>990</td>
<td>715</td>
<td>8.91</td>
</tr>
<tr>
<td>Non-respiratory tuberculosis</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Tabes mesenterica and other tuberculous and scrofulous diseases</td>
<td>706</td>
<td>603</td>
<td>3.34</td>
</tr>
<tr>
<td>Typhus</td>
<td>891</td>
<td>184</td>
<td>22.92</td>
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<tr>
<td>Typhoid and typhus</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Cancer</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Violence</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Others (McKeown &amp; Record)</td>
<td>13,890</td>
<td>14,024</td>
<td>-4.34</td>
</tr>
<tr>
<td>Others (McKeown)</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Others (Woods)</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

Note. According to Woods (The Demography of Victorian England and Wales, p.351), the total number of deaths in 1891-1900 was 5,575,375. However, the figures in his column sum to 5,575,435, and this figure has been used to calculate the figures shown in this table.

Figure 2. Average life expectancy at birth, 1541/46-1866/71

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13 Although the Bellagio conferees argued that nutritional factors played a very small role in the aetiology of smallpox, Duncan, Scott and Duncan have claimed that nutritional factors were responsible for the timing of smallpox epidemics in Penrith between 1630 and 1800. See Bellagio Conference, ‘The Relationship of Nutrition, Disease and Social Conditions: A Graphical Presentation’, in R. Rotberg and T. K. Rabb, eds., *Hunger and History: the Impact of Changing Food Production and Consumption Patterns on Society* (Cambridge, 1985), 305-8; S. R. Duncan, S. Scott and C. J. Duncan, ‘The dynamics of smallpox epidemics in Britain, 1550-1800’, *Demography*, 30


Scrimshaw and SanGiovanni, ‘Synergism’, p. 464S.


M. Dobson, Contours of Death and Disease in Early-Modern England (Cambridge, 1997).


Dasgupta and Ray, ‘Adapting to Undernourishment’, pp. 215-6. I am grateful to Robert Fogel for directing my attention to this article.


Robert Fogel estimated for the bottom decile of the whole population. However, since Fogel’s estimate was calculated on the basis that the Davies-Eden households were actually quite close to the median of the overall distribution, it is difficult to see how the two sets of figures can be reconciled. See Clark, Huberman and Lindert, ‘A British Food Puzzle’, p. 222 (note 16); Fogel, ‘Second thoughts’ (1989), pp. 37-43; idem., ‘Second Thoughts’ (1992), pp. 268-71.


34 R. Fogel, The Escape from Hunger and Premature Death 1700-2100: Europe, America and the Third World, (Cambridge, in press), pp. 10-11. I am grateful to Robert Fogel for permission to cite material from this book, prior to its publication. In the current paper, page references refer to the manuscript of Fogel’s text.


37 Scrimshaw, Gordon and Taylor, Interactions of Nutrition and Infection, pp. 144-5.


According to Feinstein, the proportion of total working-class expenditure which was devoted to food fell from 65 per cent to 61 per cent between 1828/32 and 1858/62. However, the real value of working-class earnings (after allowing for changes in the rate of unemployment) rose by 23.4 per cent over the same period (Feinstein, ‘Pessimism Perpetuated’, pp. 640, 648, 653).


The view that dietary standards improved during this period is also supported by Roger Scola’s analysis of the food supply of Manchester between 1770 and 1870. See R. Scola, Feeding the Victorian City: the Food Supply of Manchester 1770-1870 (Manchester, 1992), pp. 261-2.


Ibid., p. 4.


might have been somewhat surprised by Huck’s assertion that these midlands parishes were situated ‘in the industrial north of England’.


Ibid., p. 358.

Ibid., pp. 350, 358.


See also Kim, ‘Nutrition and the Decline of Mortality’, pp. 1384-6.


This, it seems to me, is one of the main lessons to be drawn from James Riley’s recent review of *Rising life expectancy: a global history* (Cambridge, 2001).