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# **THE DECLINE OF MORTALITY IN THE NINETEENTH CENTURY, With Special Reference to Three English Towns**

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**Imogen Anderson**  
**University of Durham**  
**Submitted for the Degree of M.A. by Research, October 1993**



28 OCT 1994

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## ACKNOWLEDGEMENTS

I would like to thank the following people for their help in the preparation of this dissertation:

Alan Heesom for his entertaining supervision.

Professor Anthony Fletcher for his support.

The Staff of the Local Archives for their patience and co-operation.

Rebecca Reader for her encouragement and company; and for the professional appearance of the title, contents and bibliography.

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THE DECLINE OF MORTALITY IN THE NINETEENTH CENTURY  
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ABSTRACT

This dissertation is intended to explore the relationship between mortality and the environment and to compare the national pattern with various local examples. The core of the analysis is based on the McKeown model and hypothesis, which seek to explain the national decline in mortality in the nineteenth century by reference to specific diseases and their interaction with the environment. This theory will be expounded and assessed in the first part of the discussion; it will then be applied to local data to discover whether McKeown's findings for the national pattern correlate with the information for certain specific urban localities. Necessarily, within the confines of the dissertation, it is impossible to explore many towns, so three have been selected for use with this model. It is not intended to deal with any local issues beyond the immediate concerns of mortality and disease, and factors in the environment affecting these.

It is, therefore, hoped to discover whether arguments evolved to explain the national mortality decline in the nineteenth century can be reconciled with specific local evidence. In the course of this examination, information regarding disease and living conditions will be described, thus adding to the plethora of data available on this subject.

## METHODOLOGY

It is necessary, before beginning the main text, to indicate the sources and limitations of the evidence used. The data for the first chapter has, for the most part, been culled from the work of modern historians. Since so much has been written on the issues of demographic growth and mortality, there is little to be gained from further direct study of the official sources, other than to verify the accuracy of information. The evidence on the local level will, however, be largely primary and it is in this area that original work can be expected.

The towns selected are Bath, Birmingham and Nottingham. These were chosen as representing different aspects of urban life. Bath was an ancient town, whose economic structure was based on a service industry, catering for a large number of leisured inhabitants, and seasonal visitors; Birmingham was an economically and industrially diverse city, which also developed trading and service facilities during the nineteenth century; and Nottingham was a commercial centre with the concentration of its trade resting in textiles, specifically, hosiery and lace.

Primary information was obtained from the local archives of the chosen towns. The data held varies from place to place and is, therefore, not wholly comparable. However, there is generally sufficient evidence to determine general trends in mortality and, often, to trace the progress of specific diseases. The quality and quantity of material depends upon the extent of local provisions and administration established to collect

statistical, and other, data. For example, the evidence for Bath was derived from a selection of weekly Medical Officer of Health reports, 1866-1875(1), and three annual reports, 1880, 1890 and 1900(2); whereas Birmingham sources are in the form of reports by doctors, such as John Postgate(3) and T P Heslop(4), Medical Officer of Health reports for the later years of the century(5), and reports to national institutions(6). In addition, evidence was procured from Royal Commission reports and other official documents.

The second half of the dissertation will be less statistically complete than the first, since the local provisions for collation of data were not as sophisticated and comprehensive as those for the national registers. It is hoped, however, that trends can be established and that the environmental contribution to mortality will become evident. National studies, necessarily, involve generalisations and standardisation of much of the evidence(7). Assumptions are made which may prove flawed when compared to a specific local picture. The local material will be used in this context, to either consolidate the validity of McKeown's theory or to prove it inapplicable to discrete locations.

There are considerable difficulties regarding the available statistical data, both at the national level and locally. Previous to 1837-8, there was no official registration of births and deaths(8), so that evidence for the first four decades of the nineteenth century is as scanty and unreliable as that for the eighteenth century(9). As far as crude statistics for the birth and death rates are concerned, the 'data are so

treacherous that they can be interpreted to fit any hypothesis'(10). Historians attempting to examine the pre-registration trends are forced back upon limited and incomplete evidence, such as that contained in the parish registers and bills of mortality(11). From these, they extrapolate figures for the whole country. These sources are so limited that it is not really possible to depend on any estimates made on this basis(12). Causes for mortality, before the inclusion of the cause of death in certification in 1838, are not known(13), although many theories have been put forward about the behaviour of certain diseases, based on the post-registration statistics(14).

It is, therefore, more valid to begin assessment of the mortality from the point at which relatively secure data can be obtained. However, there are significant reservations regarding post-registration information, which must be kept in mind. McKeown regards certification as deficient until as late as 1851(15); and Robert Woods concludes that collection of data on the cause of death posed difficulties throughout the nineteenth century(16). Much of the problem arises from troubles with diagnosis. In a period which relied on clinical assessment and experience for diagnosis, with none of the modern laboratory aids, there was naturally a degree of misdiagnosis(17). Despite the fact that the nineteenth century saw the emergence of bacteriology, which enabled understanding of the nature of infectious disease, the significance of these methods for diagnosis, even in the period 1891-90, was not fully realised(18). Gillian Cronie suggests that the nineteenth century was also a time of great prejudice against post-mortem examinations(19), and this too would retard developments in diagnosis. Medical men could

identify those ailments which presented sharply differentiated symptoms, thus recognition of smallpox, scarlatina and whooping cough were fairly simple. A disease like tuberculosis, however, was hard to define until the later stages(20), when the organism is well rooted. In the case of — diphtheria, it's chief symptom was a sore throat, yet this was a common and, frequently, trivial affliction, although it could presage a potentially lethal infection. However, in 1870, Dr Robert Semple stated that 'every ailment of the throat...[was] fashionably called diphtheria'(21). Fashion, therefore, could play a part in causing misdiagnosis(22). Tuberculosis was often deliberately misdiagnosed, or at least misreported, by doctors concerned to spare the victim's family the stigma of association with a disease believed by many to afflict only the 'innately susceptible'(23). Clearly, then, it can be expected that a certain proportion of deaths will be misattributed in the statistics..

A further problem was classification. Changes in nomenclature and developments throughout the period in diagnostic method and bacteriology led to inconsistency in the statistical arrangements. Blanket headings obscure the actions of some diseases, such as syphilis, fever and tuberculosis(24). Other causes was the most notorious, representing the greatest single category(25). Other common headings were bowel diseases, and diseases of various organs(26). Atrophy and debility figured highly, particularly for infants, whose deaths were often difficult to explain(27). This fact probably explains the prevalence of marasmus in cause of death statistics, since it was a wasting disease(28). Amalgamation of diseases also occurred, when two diseases

were believed to be of the same origin. Examples of this phenomenon are the confusion over scarlatina and diphtheria, classed together under the former name until 1861, although diphtheria was first named by the Registrar-General in 1855(29); and diphtheria continued to be classed with cynache maligna until 1869(30). Typhus and typhoid were grouped together in the official statistics until 1869(31), despite earlier proof that the two were not related(32). Diarrhoea, dysentery and cholera were often amalgamated(33), perhaps because of the difficulty in distinguishing between them. Diphtheria and croup were listed separately, although the latter is a complication of the former, throughout the nineteenth century(34). An additional problem is that some diseases had a variety of terms, for example, tuberculosis was frequently called both phthisis and consumption(35). Several diseases were probably under-represented because they led to other ailments, which were then named as the cause of death(36). Changes in registration mean that the statistical historian must either continue to class different diseases together for the sake of continuity and comparability; or attempt to determine the proportion of deaths attributable to each disease. The first approach is probably the more satisfactory and is the form used by McKeown(37).

Under-registration was also a possibility, because it was not compulsory to register until 1874(38). Farr confidently asserted in 1839 that his registration represented 98% coverage(39), but there are some doubts about this, not least because of the unofficial interment of infants(40).

The increasing accuracy of diagnosis throughout the period can result in distortion, making declines appear less steep or increases more severe. Cronje is convinced that this is a factor in explaining the less dramatic fall of extra-pulmonary tuberculosis, compared to the pulmonary type(41). Children were more likely to fall victim to the non-respiratory forms of tuberculosis, and diagnosis in this age-group was more problematic(42).

There are many problems and limitations associated with the sources used, even after the advent of official registration. Nevertheless, although it is wise to maintain a degree of scepticism, it is possible to be confident in describing trends. Woods and Hinde regard the official body of vital statistics as 'particularly rich' and potentially fruitful'(43). The statistics given below suffer from the limitations described. Local statistical evidence is subject to the same problems.

## CHAPTER ONE

### NATIONAL STATISTICS

The statistics for the nineteenth century population show the speed of the growth which took place; and, also, the rapidity of urbanisation. These factors are important to the study, since it was in the towns that mortality was most significantly reduced. Population growth is associated with two specific rates - birth and death. An increase will occur only when the birth rate exceeds the death rate. However, a declining death rate will result in a rising population in default of any change in the birth rate or even a negative one. It is argued that for the period in question, declining mortality was of more importance than the birth rate(1), especially since birth rates fell in the later nineteenth century.

TABLE 1.1

Birth rates per 1 000 of the population, England & Wales: 1838-1910(2)

1838	30.3
1840	32.0
1845	32.5
1850	33.4
1855	33.8
1860	34.3
1865	35.4
1870	35.2
1875	35.4
1880	34.2
1885	32.9
1890	30.2
1895	30.3
1900	28.7
1905	27.3
1910	25.1

Before registration there is no firm evidence of either birth or death rates, but it has been suggested by McKeown that a falling mortality was probably responsible for the onset of the increase of population(3). He inclines to this view because of the long term trend. Others(4) argue that the process of urbanisation itself caused a rise in population, since the crowded centres afforded the opportunity to meet partners and increased individual prosperity, which allowed earlier marriage. The younger the couple, the longer the legitimate reproductive period and, hence, the larger the family(5). This theory has many faults, not least of which is the problem of measuring fertility in the nineteenth century, when the age of the mother at the time of the first - or any - birth was not recorded(6). It also seems reasonable to suppose that the abnormally high mortality rates in the towns counteracted any upsurge in the birth rate there. Indeed, it is often claimed(7) that the increase

in the urban population was primarily due to internal migration, not natural increase within the city boundaries.

It is impossible, in the light of the statistical limitations, to state anything conclusively on the subject of population increase, other than the fact that it was increasing very swiftly throughout the century - at over one percent per annum(8). The population figures are below:

TABLE 1.2

Population in 1 000s: England & Wales 1801-1911(9)

1801	9 061
1811	10 332
1821	12 106
1831	13 994
1841	15 929
1851	17 938
1861	20 119
1871	22 789
1881	26 046
1891	29 086
1901	32 612
1911	36 136

The growth of urban concentration was even more significant;

TABLE 1.3

Urban population as a percentage of the total population: 1801-1911(10)  
 [Urban defined as with a population of over 5 000 inhabitants]

1801	33,8
1811	36,6
1821	40,0
1831	44,3
1841	48,3
1851	54,0
1861	58,7
1871	65,2
1881	70,0
1891	74,5
1901	78,0
1911	78,9

Not only was there a great influx of the population into urban habitats, but the majority congregated in a relatively limited number of cities. Thus, in 1801, only London could boast a population in excess of 100 000; by 1851, there were ten towns of this size, containing approximately one quarter of the population as a whole. By 1911, there were thirty-six towns of 100 000 plus, comprising 43,8% of the population(11).

These growth rates were so rapid, although they did slow down in the later nineteenth century, that there was no opportunity to evaluate the difficulties of containing such a vast population. Buildings were erected haphazardly, wherever there was space; there was no attempt at town planning. In such a climate of change and swift growth, it is hardly surprising that the mortality rates in the towns began to

climb(12). That they declined during the century is less explicable, and is the problem which McKeown and his associates seek to solve.

Data on the death rate is available from different sources. The figures given below are the estimates by Mitchell and Deane:

TABLE 1.4

Death rate per 1 000 of the population: 1838-1910(13)

1838	22.4
1840	22.9
1845	20.9
1850	20.8
1855	22.6
1860	21.2
1865	23.2
1870	22.9
1875	22.7
1880	20.5
1885	19.2
1890	19.5
1895	18.7
1900	18.2
1905	15.3
1910	13.5

Decennial statistics are as follows:

TABLE 1.5

Death rate per 1 000 of the population: 1830-1900(14)

1830-9	c21-22
1841-50	22.3
1851-60	22.2
1861-70	22.5
1871-80	21.3
1881-90	19.1
1891-1900	18.2

Friendly societies estimates(15) point to a death rate of 19.3 per 1 000 in the decade 1801-11; and census commissioners in 1831 estimated that the death rate 1816-20 was 17.6 and by 1831, stood at 18.5(16). The pre-registration data must be viewed with caution, since, as Michael Flinn says, 'a resurgence of national mortality rates in the first half of the nineteenth century cannot ... be substantiated unequivocally by sound statistical evidence'(17). The empirical view of Farr in 1849, however, was that the death rate had been climbing since 1816(18).

The statistics show a decline in the later nineteenth century, probably dating from around 1870(19). Wohl believes that over the last quarter of the century, there was an eight percent decline in mortality(20), and the decline is certainly steady and substantial in these years(21). Woodward's interpretation of the data, is that the death rate changed very little between 1841 and 1871, whereafter a decline set in(22).

Crude death rate statistics are not the only means available to evaluate life chances in this era. The estimation of life expectancy and the

calculation of probability of dying at certain ages are also valuable, and can help to pinpoint the specific ages which benefitted from the falling mortality.

### **Probability of dying(23)**

Probability of dying (p.o.d.) was declining for certain age groups during the nineteenth century. By mid-century, p.o.d. for the age group 5-10 had begun to fall, as had that for women aged 25-35. In the period 1850-1875, male p.o.d. at ages 15-20 and 25-35 was also in decline, and even the p.o.d. for 1-5 and 35-45 age brackets were exhibiting a downward trend. Infant p.o.d. (age 0-1) and that for the 45 and above group did not fall. Age specific mortality translates thus: The principal factors in male p.o.d. were, firstly, adulthood 35-64; second, infancy and early childhood, and third, early adulthood 10-24. The female factors were different, with the first group being 45-74; the second, 15-34, and the last, 1-14. The second factor group could be classed as death from risks of childbearing.

### **Life Expectancy(24)**

Life expectancy rises when mortality declines. The first attempts at estimating the English life tables in the 1840s, put male life expectancy at 40; female at 42. However, these estimates were based on the 1841 census data, which is suspect, so confidence cannot be placed in the figures. From 1840-1900, life expectancy probably increased by no more than ten years, with seven years of that rise being achieved in

the period 1851-1901. The era 1901-1960 yielded a life expectancy increase of twenty years.

The key component of the rise in life expectancy figures was the decline in the mortality associated with the 1-35 age group. Improvement could only be sustained, when the infant mortality rates dropped precipitously after 1900.

Differences in life expectancy estimates were useful benchmarks for illustrating the dichotomy between urban and rural regions, in terms of living conditions. A male child, born in Liverpool in 1861, would have a life expectancy of twenty-six years; a female baby, twenty-seven. In Okehampton Devon, the male baby would carry a life expectancy of fifty-five; the female, fifty-seven. This regional diversity in life chance was as wide a gulf as that separating national life expectancy in 1840 from that in 1960, namely thirty years.

Urban districts typically exhibited the higher mortality rate and the lowest life expectancy. Hence, life expectancy below thirty-five could be found in the registration districts of Liverpool, Leeds, Bristol Birmingham, Hull, Sheffield, Derby, Manchester and Newcastle. In 1861, it was estimated that ninety percent of the population lived in districts with life expectancies over thirty-five, but under fifty.

A low life expectancy can be positively correlated with population density - as population density rises, so life expectancy falls. However, life expectancy would only decline to a level of around

thirty-five years, and would then remain fairly static. Male life expectancy in rural districts was generally reckoned in the forties, but was variable, according to the local population density; male life expectancy in the towns was in the thirties, but did not show any alteration consistent with population density levels. Areas of high population density would also be likely to have high infant mortality rates, but the reverse is not true, since rural areas could record infant mortality rates as high as those of the towns. However, whereas the rural districts could register either high or low infant mortality, the towns could not maintain a low level.

Woods and Hinde show that the movement of life expectancy from forty to fifty was specifically associated with the declining mortality in the age group 1-24. However, they maintain that no further upward movement was possible until the infant mortality rate began to drop. Thus, the five year rise in the life expectancy estimates, 1901-11, was intimately connected with the changes in mortality of those under twelve months old.

To summarise, the national statistical evidence shows a decline in mortality from about 1870, with p.o.d. falling in the most productive age groups, and life expectancy rising, particularly after mid century. This evidence suggests that a change occurred during the second half of the nineteenth century, which allowed a previously high level of mortality to be redressed. McKeown's theory seeks to explain what these changes were, and how they translated to a fall in national mortality.

Before embarking on an exposition of the McKeown hypothesis, it is, perhaps, useful to explore, briefly, the subject of infant mortality. Infant mortality failed to show any downward inclination until after the turn of the century, so that it can be discounted in any examination of the declining trend in mortality in the nineteenth century. However, to be credible, any explanation offered for this decline must exclude any factors likely to affect the life chances of those under twelve months old, and, for this reason, it is vital to detail some of the causes which sustained the high infant mortality levels.

### **Infant mortality**

The term infant, in this context, is taken to mean those children below one year old.

Whereas the general death rate was in decline, there was no corresponding fall in the rate of infant mortality during the nineteenth century(25). The death rate of the infant population represented as much as thirty percent of all deaths(26), and from 1840, accounted for almost one quarter of the total mortality(27). Smith regards the infant population as 'the single largest group at risk'(28) during the nineteenth century. The figures given below are from Mitchell and Deane;

TABLE 1.6

Infant death rate per 1000 live births, England and Wales, 1839-1910(29)

1839-40	153
1841-5	147
1846-50	161
1851-5	156
1856-60	150
1861-5	151
1866-70	157
1871-5	153
1876-80	144
1881-5	139
1886-90	145
1891-5	151
1896-1900	156
1901-5	138
1906-10	117

As if to underline the failure of the infant mortality rate to drop in concert with the general rate, the death rate recorded in 1899 was the highest ever, standing at 163 per 1 000(30). These rates translate to the following absolute figures:

TABLE 1.7

Actual Infant deaths: 1841-1901(31)

1841	75 507 infant deaths
1861	106 428
1881	114 976
1901	140 648

Anthony Wohl suggests that the official statistics may have been under-representative, and he quotes William Farr as having estimated the exclusion of 40 000 stillbirths from the official statistics each year(32). Wohl goes on to argue that, as the registration figures became more accurate later in the century (registration became compulsory in 1874(33)), this may have distorted the picture of the trend in infant mortality(34). However, he counterbalances this suggestion with the observation that the evidence shows a decline in the proportion of illegitimate births to total births in the last quarter of the century. This is significant because the death rate among illegitimate babies was higher than that among legitimate infants(35). Cartwright estimates that one third of illegitimates died under one year, whilst one quarter of the legitimate did(36). Thus, argues Wohl, the death rate should have exhibited some evidence of decline, if only because a high risk group was falling in size(37).

Woods and Hinde estimated infant mortality to be 0.130 nationally, but in excess of 0.175 in industrial centres(38). Infant mortality was clearly of more importance in the urban districts, with most large industrial towns containing districts with an infant death rate of over 200, at the end of the nineteenth century(39). In the early 1890s, whilst the national infant death rate was 153, that recorded in the twenty-eight largest towns was, on average, 167(40). Much of this higher mortality can, probably, be ascribed to water and food-borne diseases(41), because of the insanitary living conditions prevailing in towns(42). Woods and Hinde hypothesize that two identical populations exposed to the same disease, but located, one in an urban district and

one in a rural, would show a higher case fatality rate in the urban environment. This is important because some diseases were more prevalent in the towns, especially those spread by factors enhanced by crowding, and a high case fatality rate would have exacerbated their mortality(43). Infant mortality was also found to be most severe among the poor, and least common among the 'comfortable classes'(44). This correlates infant mortality with wealth, which provided the family with the ability to protect a child from hostile environmental influences, such as contaminated food and water, and almost constant exposure to viruses.

The leading malady among artificially fed infants was diarrhoea(45) (which supports the theory that the water/food-borne diseases were a major factor in higher urban infant mortality)(46). Diarrhoea reached a seasonal peak in summer, and attacks were acute and, probably, had a high case fatality rate, since a weak child could perish within forty-eight hours(47). It is impossible to prove case fatality, since the evidence for prevalence is unavailable in official records. Statistical data on diarrhoea was collected by the Registrar-General from the early 1870s, and it was discovered that eighty percent of all diarrhoeal deaths were among children below the age of two years(48). It was more common in the towns; in the eighteen largest cities, the rate was 4.4 per 1 000 deaths; in the fifty next largest, 3.5; and in the rest of England and Wales, 1.5(49). Diarrhoea not only killed directly, but could lead to death from so-called atrophy and debility because, even after the purging was over, the child often developed a loathing of food, followed by fever, thirst and restlessness leading to

emaciation(50). The number of deaths resulting from this cause cannot be accurately calculated, but Dr Hugh Jones estimated that between 1873 and 1875, 17.1 per 1 000 died of diarrhoea, but 39.5 died from atrophy and premature birth(51). A proportion of the latter deaths was probably associated with earlier diarrhoea, although it is not possible to determine how much.

Although doctors had previously regarded diarrhoeal deaths with resignation, believing it to represent a natural hazard, by the 1850s, this attitude had changed(52). Doctors began to equate it with maternal improvidence and insanitary living conditions(53). Nutrition of infants was faulty by modern standards. Much of the medical community's concern in the 1850s, stemmed from the increase in information about adulteration of milk and other foods; and the cooking and housekeeping habits of the lower classes(54). Cheap milk, purchased by the poor, was skimmed, watered and old, and the shops from which it was bought were often very insanitary(55). The practice of giving infants solid food was widespread(56), and mothers tended to feed on demand, often with whatever the rest of the family was having(57). Bread pap (made of bread, doused in boiling water and mashed with sugar and milk), porridge and bouillie (flour mixed with milk and water) were given liberally; and the infants were also frequently dosed with tea, coffee and spirits. Even breast-fed children were given solid foods to 'quiet' them(58).

The use of drugs to quiet infants was widespread(59). Laudanum was easily available and mixtures containing opiates, such as Godfrey's Cordial, were given liberally to wakeful children(60). In 1844, *The*

*Times* stated that most druggists and village stores sold large amounts of opium every week(61). Babies under one month old were given a teaspoonful of Godfrey's Cordial, which soon increased to three(62). The opium was adulterated, which probably saved the population from poisonings, and many more infants died from starvation than overdosing(63). The continual use of narcotics made the baby disinclined to take food, and malnutrition or starvation followed(64). Malnourished children are more likely to succumb to infection, since their resistance is severely diminished. The WHO concluded that malnutrition lowers infants resistance to tuberculosis, measles, acute diarrhoeal disorders, typhus and whooping cough(65). The free sale of opiates was not curtailed until the end of the century(66), although in 1868 a Pharmacy Act, which restricted opium sales to legally registered chemists and qualified pharmacists, was passed at the behest of the Privy Council. This act was ineffective since it did not require any purchaser to give their name and it laid no restriction on the amount sold. Even more importantly, patent medicines were exempt(67).

Other important causes of infant mortality were among the zymotics: whooping cough, measles, smallpox and croup(68). Whooping cough was estimated in 1874, to have a case fatality rate in children under one, of 1:2, with two fifths of deaths from whooping cough being within that first year(69). Charles Creighton, writing in 1891, believed that it was mainly because of infant diarrhoea and whooping cough that the death rate of those under one year had 'fallen but little in successive decennial periods'(70). Scarletina, cholera, typhoid and tuberculosis, were not consistent primary killers of the infant population(71).

Prematurity contributed indirectly to the high level of infant mortality. Premature babies are more susceptible to infection and are, therefore, killed by diseases which a full term child would survive(72). Many deaths recorded as fatal convulsions, nine day fits, atrophy and debility resulted from premature birth(73). The causes of pre-term delivery are not known, but poor maternal diet is believed to be a factor(74). Wohl insists that the mother was the source of infant mortality in the nineteenth century(75). Woods(76) tentatively suggests a possible relationship between fertility levels and high infant mortality. He states that a high infant death rate is usually accompanied by a compensatory boost in fertility. If fertility is high, and women are having high parity births in their forties, then the chances of stillbirth and infant mortality increase significantly. Problems associated with the measurement of fertility are, however, prohibitive, as explained earlier. Nevertheless, Woods's theory seems sensible, especially in view of the potential internal damage, anaemia and exhaustion which were associated with frequent childbirth(77).

As will be discussed below, tuberculosis, particularly the pulmonary type, was a major killer in the nineteenth century. It was the extra-pulmonary types, however, which were associated with children(78). In fact, deaths from scrofula, tabes mesenterica and tubercular meningitis tended to be infantile(79). When not fatal, these diseases could leave the victim deformed or disfigured(80). Tubercular meningitis was almost always acute and fatal(81). Scrofula attacked the lymph nodes, as did tabes mesenterica, which also caused wasting(82). Incidence of scrofula and tabes mesenterica declined along with the decline in the birth rate

from 1876(83). This may have been because they were predominantly diseases of the poor(84), and with a smaller family, the pressure on resources was lessened and, subsequently, the life chances of the individual members increased. In addition, the mother was likely to be healthier, if bearing fewer offspring

The accuracy of statistics for non-pulmonary tuberculosis are in doubt because of the difficulty of diagnosis in the very young. An increase in the accuracy of diagnosis may have distorted the figures available, making the decline in mortality from this category of disease appear less than it was(85). With this in mind, the figures for extra-pulmonary tuberculosis are as follows;

TABLE 1.8

Annual death-rate from extra-pulmonary tuberculosis per 1 000 living in England and Wales: 1851-1910(86)

	male	female
1851-60	0.8	0.6
1861-70	0.7	0.5
1871-80	0.7	0.5
1881-90	0.7	0.5
1891-1900	0.6	0.5
1900-10	0.5	0.4

Whatever the influences on the infant death rate, infant mortality in 1900 was a 'sobering reminder ..... that, whatever vast strides the Victorians had made in sanitary reform, there was still a long way to

go'(87), since the infant mortality rate is a 'sensitive barometer of environmental conditions'(88).

## CHAPTER TWO

### THE MCKEOWN HYPOTHESIS

Thomas Mckeown and his associates formulated a theory to explain the fall in mortality in the nineteenth century(1). As Robert Woods and P R A Hinde claimed 'much of our knowledge and understanding of the secular decline in mortality . . . in the nineteenth century is based on the work of McKeown'(2). The McKeown model thus forms a basic platform upon which much work has depended; and this makes it an ideal and respectable tool with which to assess the data available in the localities.

#### A Summary

McKeown determined that the fall in mortality could be isolated to a decline in the death rate in a certain number of diseases of the infectious class(3). This is shown by figure 1(4). Non-infectious, or diseases not caused by micro-organisms, contributed only 8% to the overall fall, in McKeown's opinion(5). Within the infectious class, McKeown then isolated the principal components of the decline by examining the trends in the behaviour of the specific diseases. Figure 2 shows the mean annual mortality from certain diseases of the infectious class(6). McKeown attributed the decline in mortality to the first five, namely, tuberculosis; the typhus, typhoid, continued fever group; scarlet fever; cholera, diarrhoea and dysentery; and smallpox(7). Measles did not begin to decline until 1915, diphtheria death rates rose at the end of the century(8); and the decline in whooping cough was

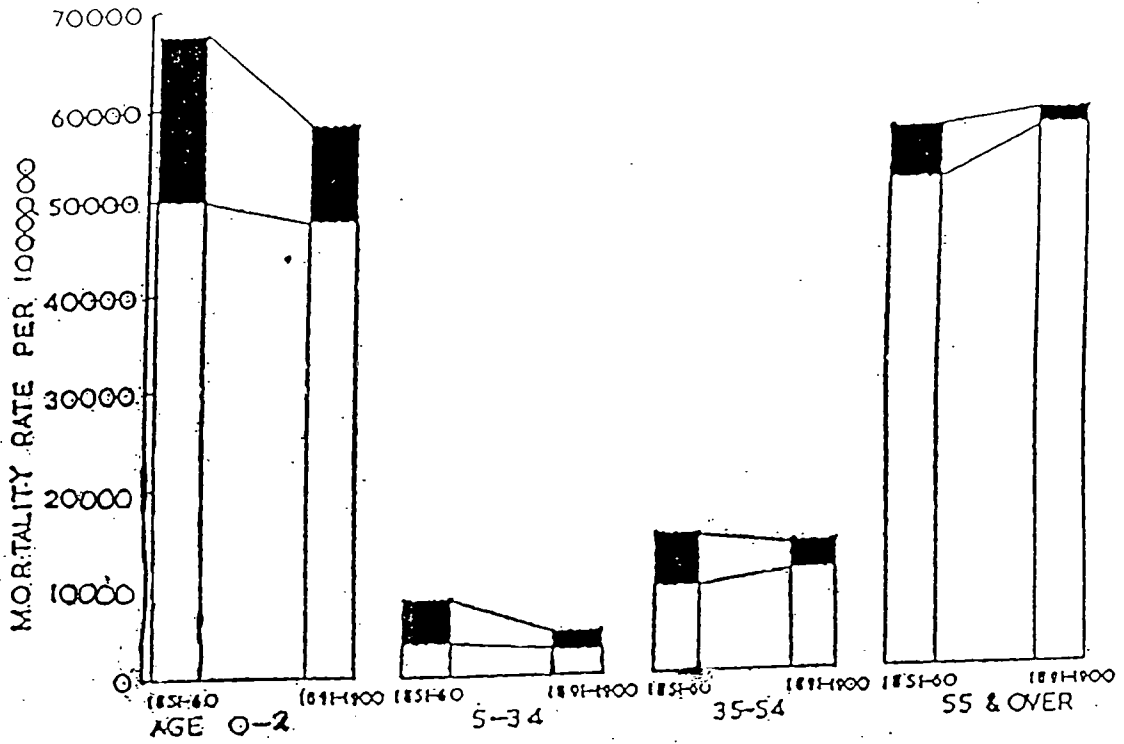


FIGURE 1: MORTALITY RATES IN DIFFERENT AGE GROUPS: COMMUNICABLE\* DISEASES (SHADED) AND OTHER CAUSES (UNSHADED). 1851-60 and 1891-1900

\*tuberculosis, typhus, enteric fever, scarlet fever, diarrhoea dysentery, cholera and smallpox.

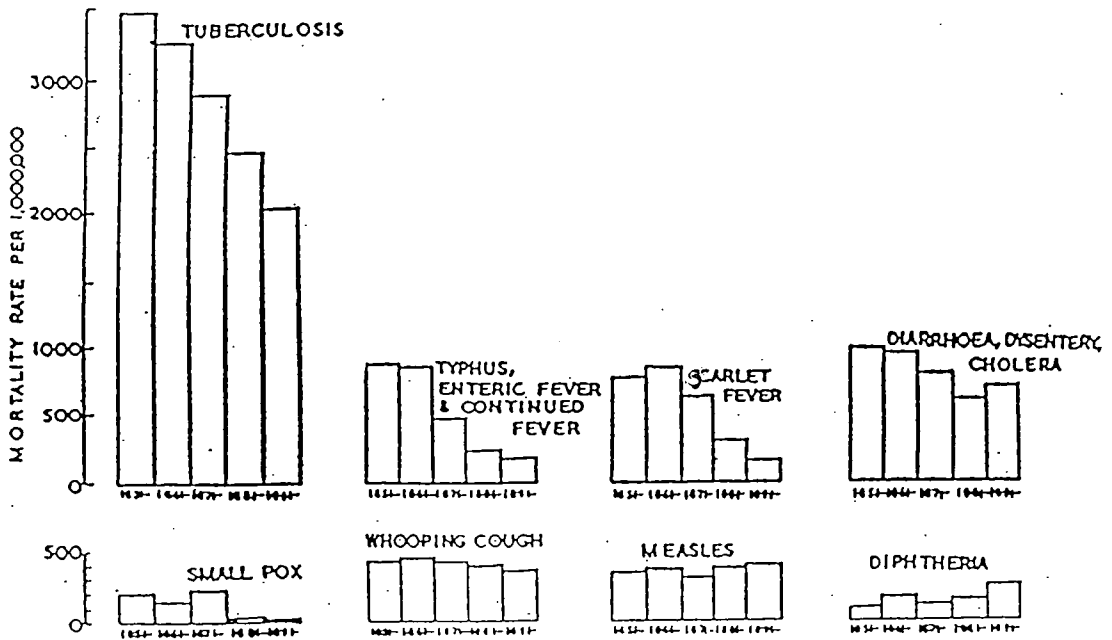


FIGURE 2: ANNUAL AVERAGE MORTALITY RATES FROM CERTAIN COMMUNICABLE DISEASES.

'trivial'; registering only 2.3% of the total mortality fall(9). The five groups were assigned a percentage score relating to the proportion of the decline for which each was held to be responsible. Thus, tuberculosis 47.2%; the Typhus group 22.9%; scarlet fever 20.3%; cholera, diarrhoea and dysentery 8.9%; and smallpox 6.1%(10). Figure 3(11) shows the degree of decline at different ages in each case.

McKeown further examined the falling trends to isolate the causal factors. Scarlet fever he found to have declined as a result of a change in the haemolytic streptococci (the micro-organism responsible for scarlet fever), which rendered it less lethal(12); the typhus group and cholera group were affected by sanitary reform measures associated with public health and personal hygiene(13); and tuberculosis was thought to have declined because of environmental change and improvements in the standard of living, particularly diet(14). Smallpox was largely eradicated by vaccination, although another factor, probably a change in the character of the disease itself, is considered by some to have made a significant contribution(15). McKeown regards smallpox as playing a very minor part in the overall mortality decline(16). He also believes that the vaccination debate has led to an exaggeration of the position of smallpox as a nineteenth century killer(17). It was, and is, incurable and was one of the most lethal infections known to man, but it was already on the wane in 1780, thanks to the work of Jenner(18); and the Government took a progressively more invasive role and legislated for compulsory vaccination of infants in 1853, with fine penalties introduced in 1871(19). The fact that the conquest of smallpox represented the sole medical advance of the

TUBERCULOSIS  
 TYPHUS, ENTERIC FEVER, CONTINUED FEVER  
 SCARLET FEVER  
 DIARRHOEA, DYSENTERY, CHOLERA  
 SMALL POX

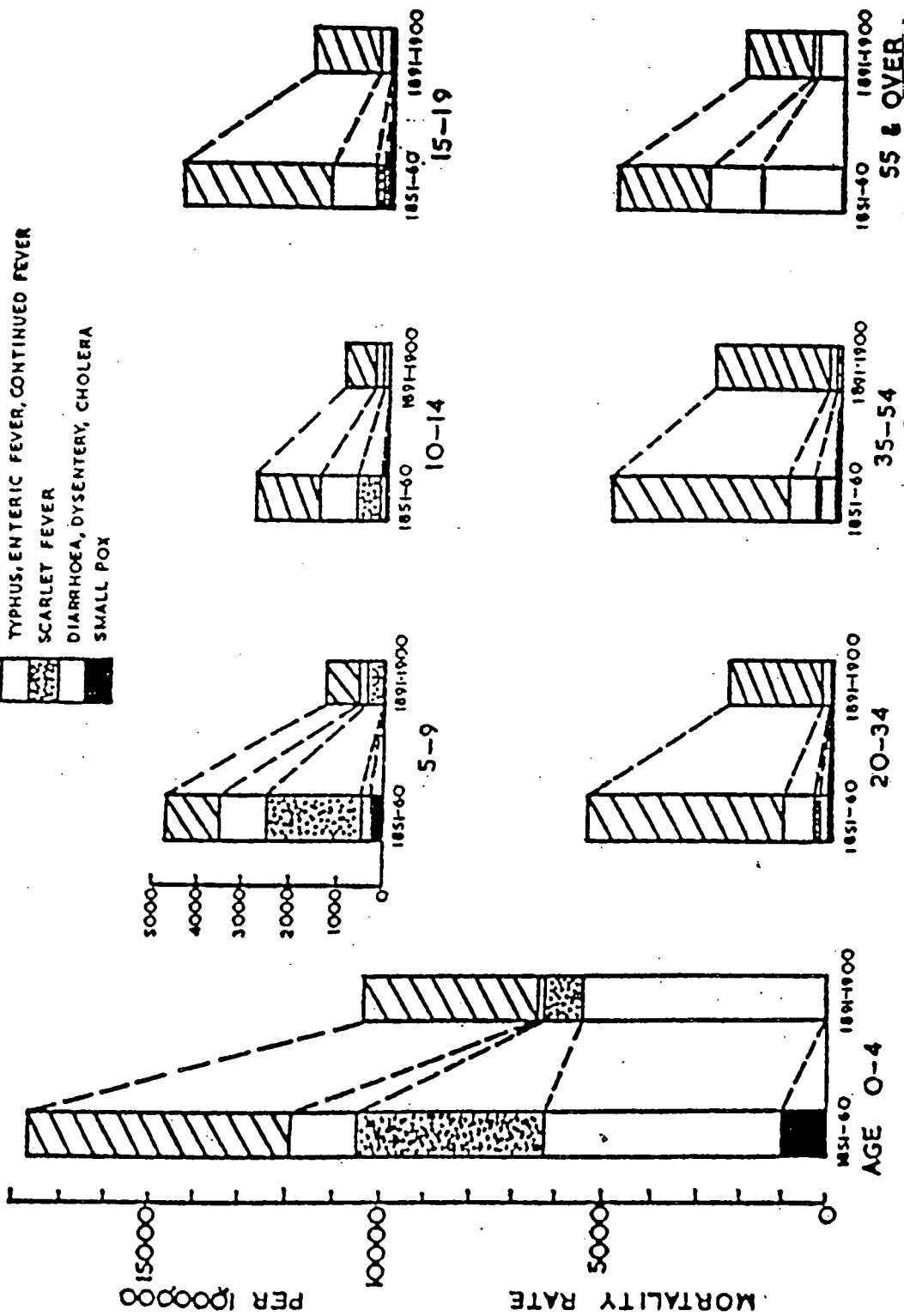


FIGURE 3: CONTRIBUTION OF SPECIFIED COMMUNICABLE DISEASES TO MORTALITY RATES AT DIFFERENT AGES.

century(20) may have encouraged historians to give it more prominence than it's statistical impact merits. In addition, the fact that the state became involved in public health through the introduction of vaccination acts makes it an important subject for legislative historians. Nevertheless, as a factor in the decline of the death rates of the nineteenth century, smallpox ranks very low in terms of importance.

The causal factors are also assigned scores denoting the degree of contribution to the decline made by each one. A simplified explanation of this is given in figures 4 & 5(21). Environmental change associated with rising living standards and dietary improvement is reckoned to contribute 50% of the decline, because it was related, by McKeown, to the fall in the tuberculosis death count. Sanitary reform contributes a quarter of the decline, influencing the cholera and typhus groups; and change in the character of disease accounts for a further quarter, principally from association with scarlet fever(22).

It is useful to have some information on the cause and progress of these diseases, and this will be provided, briefly, before giving a more detailed account of McKeown's theory.

### **Tuberculosis**

Tuberculosis is caused by mycobacterium tuberculosis(23). This organism is an airborne infective, although it can also be carried in contaminated milk(24). The bacteria expelled in the sputum of a victim

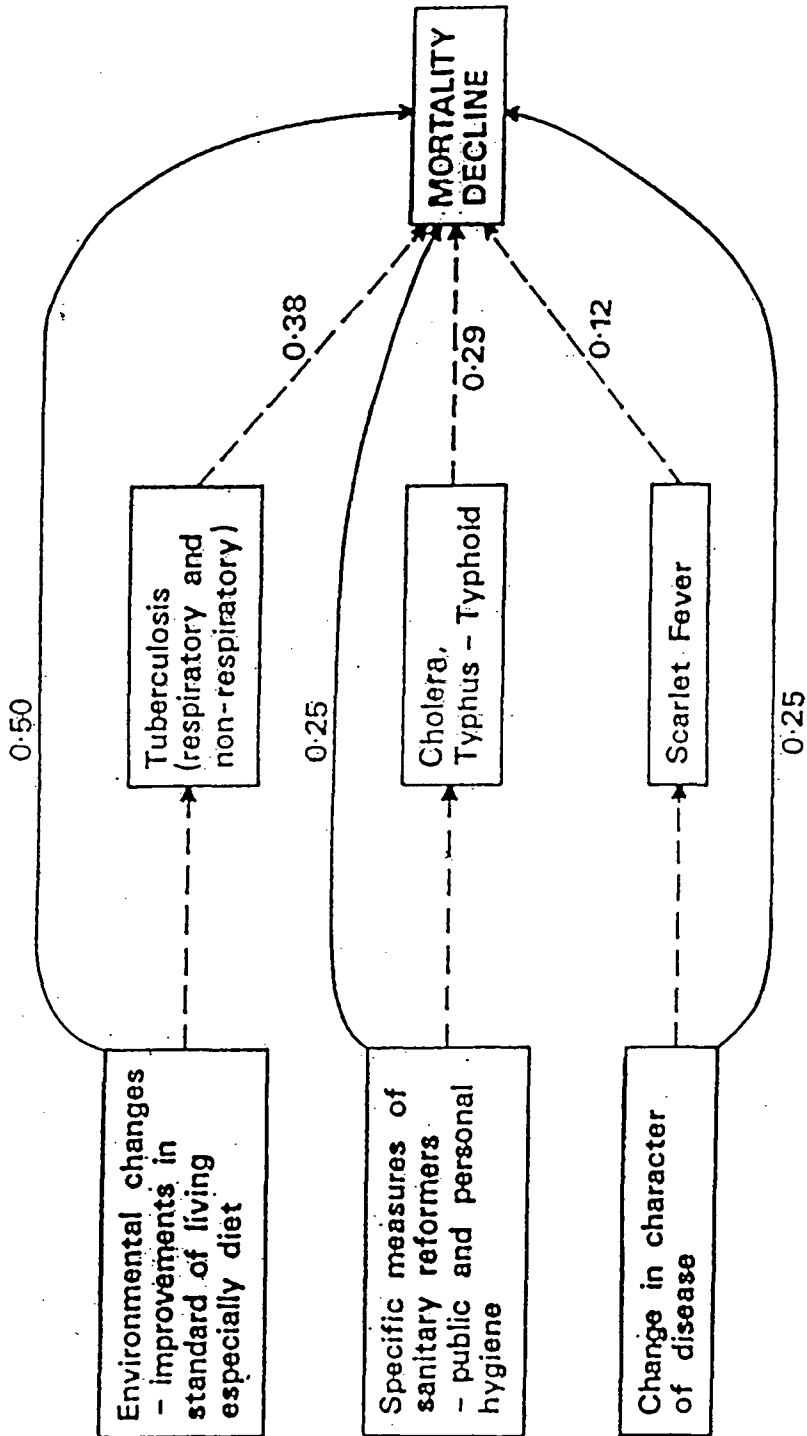


FIGURE 4: GENERALIZED MODEL OF THOMAS MCKEOWN'S INTERPRETATION OF THE REASONS FOR MORTALITY DECLINE IN THE NINETEENTH CENTURY

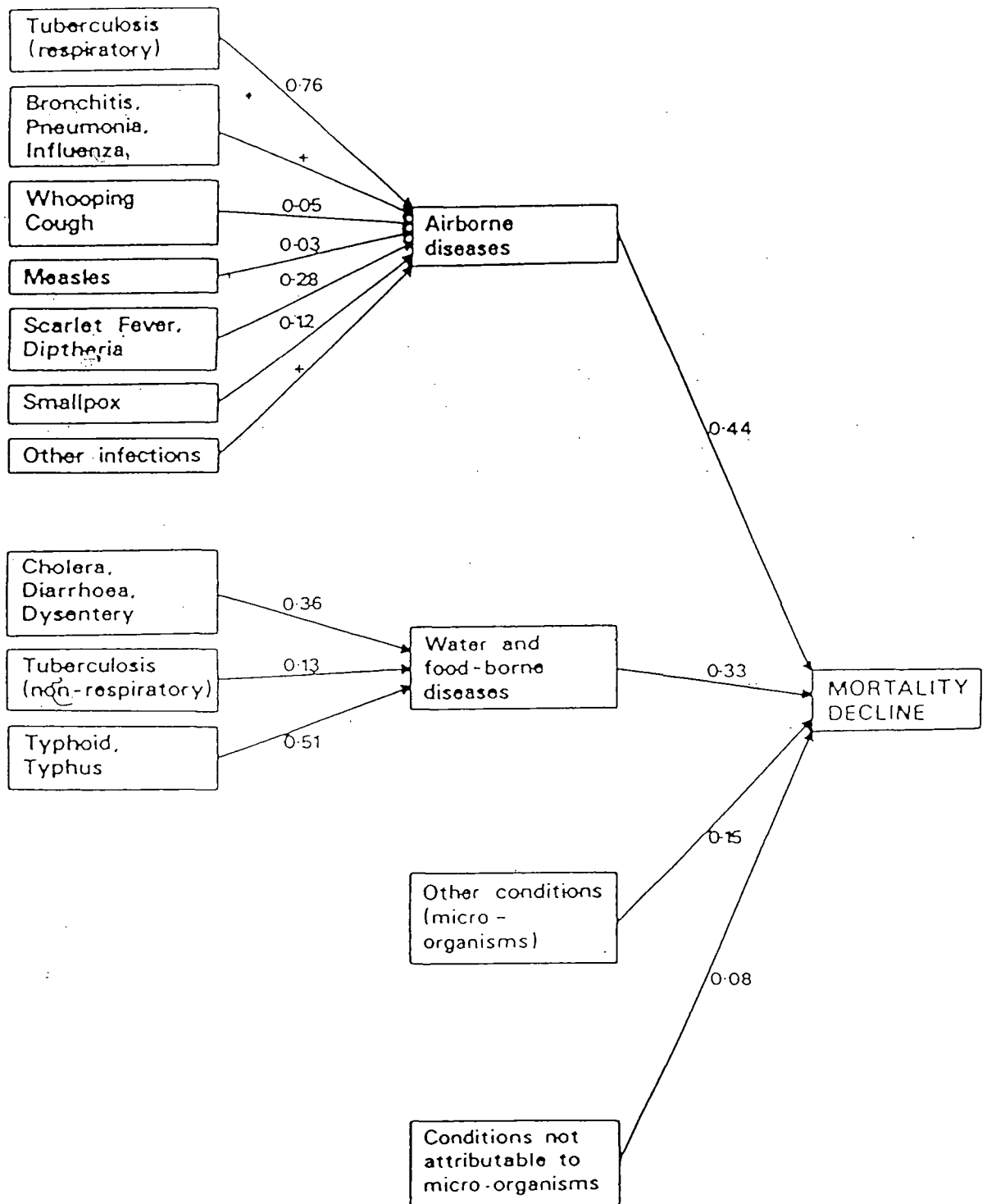


FIGURE 5: CAUSE SPECIFIC INFLUENCES ON MORTALITY DECLINE: ENGLAND AND WALES 1848-1854 to 1901. (INCLUDING McKEOWN'S NUMERICAL WEIGHTS).

can live for months(25), thus offering a chance of infection from the immediate environment, even after the sufferer is dead. Once ingested, the bacilli lodges somewhere and multiplies forming a foci of infection. This leads to necrosis of the tissue and the formation of lesions and cavities(26). The next stage depends upon the natural or acquired resistance of the victim. The infection can be contained by calcification of the affected areas and eventual destruction of the bacilli, although a contained infection can be reactivated; or the disease can progress, as the bacilli continues to destroy tissue, leading to death(27).

Exposure to the tuberculosis bacilli is thought to have been widespread. A study conducted in London in 1930-1, using the tuberculin test, showed that 58.3% of the sample (children aged 10-15) reacted positively, with a positive reaction in 82.2% of the 14-15 year olds(28). Since this was in an era when tuberculosis had declined severely, the degree of exposure experienced in the nineteenth century towns can only be speculated upon. Mass exposure argues for a large proportion of the population being sufficiently strong to resist the ravages of phthisis(29). The prognosis for tuberculosis increased throughout the century, from 12 months at mid century to 3-5 years by 1910(30). It seems likely that this phenomenon was related to improvements in diagnosis of symptoms, rather than any increase in host resistance.

Tuberculosis was primarily, although not exclusively, a disease of the poor(31). This is because susceptibility increases when the victim is in a malnourished and/or debilitated state. Bad housing, inadequate

clothing, poor nutrition, insanitary conditions and general disability from ill health are all closely identified with the disease(32). Tuberculosis was found to be 50% higher in back-to-back housing than in other houses located in the same area(33). During the nineteenth century, up until the time of the Koch's discovery of the organism in 1882(34), there was a belief in 'tubercular diathesis' or innate, inherited susceptibility(35). However, with the realisation that it was infectious, the disease lost this stigma(36). Nevertheless, members of the same family were likely to succumb to the infection, not only through the obvious factor of close proximity with the sufferer, but also because family health was likely to be similarly debased.

Extra-pulmonary tuberculosis was a serious malady, but its fatalities were largely infantile. It has already been explored under infant mortality. The death rates of pulmonary tuberculosis were extremely high. Between 1851 and 1910, an estimated four million died from this cause in England and Wales(37). This represents nearly 13% of the whole mortality for this period, making phthisis the biggest single killer of the nineteenth century(38). Over a third of these deaths were in the 15-34 age group, and, indeed, more than 40% of the young adult mortality was attributable to tuberculosis(39). The rate was even higher among the 20-24 age range, where it accounted for almost half the mortality(40).

TABLE 2.1

Mean annual death rate of respiratory tuberculosis, per 100 000 living(41)

1851	277
1861	258
1871	232
1881	189
1891	156
1901	128
1911	104

Although tuberculosis undoubtedly did decline(42), it was still a grievous killer, and in 1894, was annually claiming three times as many lives as were lost through action and disease in the whole Crimean war(43). Moreover, these deaths were principally located among the young adult population, who represented the most economically productive age group.

#### Cholera, dysentery and diarrhoea

These three diseases do not really fit comfortably into the same category. Cholera was an exceptional disease. It was of indian origin and spread in pandemic waves, at intervals throughout the nineteenth century(44). It's impact was not statistically high in comparison with many of the endemic diseases. The 1849 epidemic claimed only a few more lives than typhus in the same year; and, over a five year period around 1849, deaths from common diarrhoea exceeded those from cholera(45). In 1854, the two totals were virtually equal(46). However, cholera was of exotic origin and appeared suddenly, so it attracted a great deal of

contemporary comment and government activity(47). Subsequently, historians have accorded it a disproportionate degree of attention, because of it's social impact rather than it's statistical importance(48). Diarrhoea was a mainly infantile killer(49) and the information specifically regarding dysentery is very scarce.

### **Typhus and typhoid**

Typhus and typhoid, although classified together until 1869, are totally dissimilar organisms(50). Typhoid is caused by salmonella typhi bacillus, which is contracted from contaminated food and water(51). Approximately 5% of those infected will become carriers, thus providing a reservoir of disease(52). Bill Luckin(53) estimates that a closed community obtaining water from a polluted source could expect to lose a fixed proportion of the population every year. Typhoid was most common in the summer and autumn and carried a case fatality rate of 15-20%(54). Typhoid was declining from 1870; and during the last thirty years of the nineteenth century, it's mortality fell by half.(55)

Typhus is a rickettsial disease, the causal organism is rickettsia prowazeki(56). It is transmitted via the louse vector, which is why the disease is synonymous with famine and disasters, and was associated with jails and ships(57). All these environments offered massed humanity, in a poor sanitary state, with little means of achieving personal hygiene. Typhus is principally associated with the cold months, although not exclusively(58). The case fatality of the nineteenth century was, probably, around 20-45%, although in intense epidemics, the rate can be

over 50%(59). Edwin Chadwick, writing in 1842, declared that "the annual slaughter in England and Wales from preventable ... typhus .... appear[ed] to be double the amount .... suffered by the allied armies at Waterloo"(60). However, by 1870, when the two fevers were separated, typhus was in steep decline(61). The swift and sudden plunge in the typhus death rate(62) suggests that the rickettsiae might have undergone a change. However, case fatality rates remained unchanged, which tends to mitigate against this theory(63). Typhus killed 4 281 in 1869, but by 1878, the number fell to below 1 000, and by 1886, it was under 250(64). Typhus never became a major killer again. Typhoid killed more people over a longer period(65). Between 1871-1880, the average death rate from typhoid was 0.32 per 1 000(66). However, the death rate 1891-1900, was almost half that figure.

TABLE 2.2

Typhoid, typhus and pyrexia death rate per 1 000 living: 1847-1910(67)

1847-50	1.24
1851-5	0.98
1856-60	0.84
1861-5	0.92
1866-70	0.85
1881-5	0.21
1886-90	0.17
1891-5	0.17
1896-1900	0.11
1900-10	0.07

TABLE 2.3

## Fever deaths: England and Wales 1869-91(68)

	typhus	simple/ill-defined	enteric
1869	4281	5310	8659
1870	3297	5254	8731
1871	2754	4248	8461
1872	1864	3352	8741
1873	1638	3081	8793
1874	1762	3089	8861
1875	1499	2599	8913
1880	513	1490	6710
1885	318	662	1765
1890	160	361	6146

**Childhood zymotics: smallpox, scarlet fever, measles, whooping cough and diphtheria**

Smallpox has already been discussed above, so it is not necessary to go into any more detail here, other than to give some statistical evidence. The last major smallpox outbreak in Great Britain occurred in 1900-1905(69). Previous to that, there had been a short epidemic in 1870-1, which had highlighted the need for vaccination(70); and from 1870-1900, there was a definitive fading in major smallpox mortality(71).

TABLE 2.4

Smallpox mortality rate: 1771-1880, per 1 000 living(72)

1771-80	5	
1801-10	2	
1801-35	0.83	
1837-40	2.3	major epidemic 1837-40
1841-50	0.40	
1851-60	0.28	
1861-70	0.28	
1871-80	0.45	major epidemic 1871-2

The actual figures for the two epidemics, 1837-40 and 1871-2, are remarkably similar. In the first, there were 41 600 deaths; in the second, 42 000. The 1871-2 outbreak also took place amid a larger population, and achieved it's death toll in two years rather than four(73). However, the latter epidemic was preceded by a scarlatina outbreak which probably killed many of the potential smallpox victims(74). However, smallpox was a diminishing threat, by 1830 there were already four diseases ahead of it in the mortality tables(75).

### Measles

Measles did not decline in the nineteenth century, killing at least 7 000 per annum throughout the period(76). Measles is most lethal in the six months to two years age group(77); and is especially virulent when accompanied by malnutrition, so that case fatality was probably higher in poor families(78). In poor nations, in the 1960s and 70s, case fatality was measured at 25%(79)

TABLE 2.5

Measles mortality rate per 100 000 living: 1838-1910(80).

1838-42	53.9
1847-52	40.3
1856-60	42.5
1866-70	42.8
1876-80	38.5
1886-90	46.8
1896-1900	42.1
1901-5	32.7
1906-10	29.1

TABLE 2.6

Deaths from measles: 1838-1911(81)

1838-40	26 777
1847-50	28 097
1851-60	78 211
1861-70	94 149
1871-80	93 928
1881-90	121 068
1891-1900	126 841
1901-10	105 481

In the years 1861, 1863 and 1874, measles mortality exceeded that of scarlet fever(82). The growing elementary schools contributed a reservoir of infection which enabled the disease to be constantly maintained(83). When scarlet fever declined, measles and whooping cough took over as leading childhood killers(84).

## Diphtheria

Diphtheria was only recognised by the Registrar-General in 1855, and may only have appeared then(85). It was classified with scarlet fever until 1861; and because of the uncertainty of diagnosis, any conclusions about it must be speculative(86). It is caused by coryne bacterium diphtheriae, an airborne infective, which can also be transmitted in milk and on clothes and objects(87). It affects the throat, but also produces a toxin which can spread through the body and cause permanent damage to the heart and nervous system(88). It was a predominantly childhood disease, with half the mortality being in the under fives, with four-fifths of the remainder located in the five to thirteen year olds(89). Between 1855 and 1869, it killed over 61 000 people at an average rate of 35 per 100 000.(90)

TABLE 2.7

Death rate per million from Diphtheria: 1855-1893(91)

1855	20
1860	261
1865	126
1870	120
1875	142
1880	109
1885	163
1890	179
1893	302

An antitoxin was discovered in 1894, but, as with all early antitoxins, it had to be administered within the first four days of infection, which is when symptoms are the least evident(92). However, the mortality did decline from 9 445 deaths in 1894 to 7 661 in 1898(93). This fall probably relates to the wealthier classes, since the antitoxin was expensive, and not distributed free(94). It was 1913 before there was an effective diphtheria prophylactic and 1923, before the first safe vaccine was produced(95).

#### Whooping cough

Whooping cough has been touched upon in the section dealing with infant mortality. It became a leading killer of the child population, when scarlet fever declined(96).

TABLE 2.8

Whooping cough death rate: 1841-1905(97)

1841	8 099
1851	10 275
1861	12 309
1871	10 360
1881	10 830
1891	13 612
1901	10 205
1905	8 709

TABLE 2.9

Whooping cough death rate per 1 000(98)

1881-5	0.46
1886-90	0.44
1891-5	0.40
1896-1900	0.36

As McKeown stated, the decline is small(99). Whooping cough had a higher death count in the towns, and it's real impact may be understated in the figures, since it often accompanied outbreaks of cholera or measles; and was, frequently, a precursor of pneumonia and severe respiratory ailments(100).

Scarlet fever

Scarlet fever was an acute infection of the throat, skin and middle ear(101). It's causal agent was the haemolytic streptococci, which, as has been stated, underwent a mutation which resulted in the decline of scarlet fever as a killer disease(102). Scarlet fever affected mainly the under-ten age bracket, with only 5% of deaths being over that age(103). Although most prevalent amongst the four to eight year olds, it had a higher case fatality (50%) in infants, which dropped to around 27% in the two to four group(104). After 1859, scarlet fever represented between 4 and 6% of the total mortality, killing nearly 34 000 in 1863 and over 26 000 in 1874(105). There then followed a dramatic fall to 17 per 100 000 in 1886, representing an overall decline in scarlet fever mortality of 81%.(106)

TABLE 2.10

Annual death rate per million from scarlet fever; 1851-90(107)

1851-60	832
1861-70	972
1871-80	716
1881-90	338

**Diseases and decline: McKeown**

Having outlined the nature of the diseases which had the greatest impact in the nineteenth century, it is now possible to discuss McKeown's theories on the decline of the five he selected. While it might seem sensible to begin with tuberculosis, since this made the biggest difference to mortality, McKeown's theory on this is more complicated and controversial than any of the others, so it is best to leave it until last.

The decline in the mortality from scarlet fever was associated with a change in the character of the disease itself. This can be determined by the fact that the changes in scarlet fever mortality levels were rapid, happened over a short period, were independent of any specific measures, and were inconsistent with any environmental progress(108). These facts lead to the conclusion that it must have been a mutation of the haemolytic streptococci that altered the course of scarlet fever mortality(109). A change in the nature of host resistance does not fit into the pattern either, so it can safely be discounted as a factor(110). The autonomous shift in the virus cannot be clinically

proven, obviously, but this explanation is generally accepted as correct(111).

Smallpox, as has been explained, was largely controlled by medical science. The vaccination advance was the only contribution of the medical community to the mortality decline in the nineteenth century, and it was underway before mid century(112). Even those who seek to advocate a wider role for the medical establishment, for example, Woodward(113), are unable to offer anything other than increased hospital provision, which is a dubious proposal, and lobbying for public health reform by doctors. Curative therapy for most diseases was not available until well into the twentieth century, when the chemotherapeutic revolution offered major advances in combatting disease(114). Tuberculosis had to wait for streptomycin in 1947, and the BCG vaccination was not introduced until 1954(115); bronchitis and pneumonia were untreatable until the sulphonamide drugs were developed in the 1930s, and they only really affected lobar pneumonia(116). Whooping cough, too, was not treatable until the sulphonamides, with immunisation only available from 1952(117). Measles and scarlet fever also awaited the introduction of sulphonamide drugs(118). The medical establishment, then, achieved little in the curative field in the nineteenth century, although the origins of spectacular advances in applied medical science can be traced to the work of Jenner, Koch, Lister and Pasteur(119). The conquest of smallpox, therefore, was unique.

The typhus/typhoid group and cholera/dysentery/diarrhoea class are denoted as having been affected by sanitary reform(120). McKeown attributes the fall in mortality from the water-food borne diseases to this factor, and clearly this is sensible, since, when the water supply was cleaned, the principal mode of transmission for these infections was destroyed(121). Additionally, when the water supply ceased to be a source of suspicion to public health inspectors, their attention was drawn to other carriers of the disease, that is, unsafe food and milk(122).

Typhus, however, is not a water-food borne infection, and is only classed with this type through a desire to retain statistical consistency with pre-separation figures(123). McKeown concludes that the elimination of typhus was due to a rise in living standards, specifically, improved hygiene; for example, better water supply and improved personal cleanliness (which could be expected to show results in the eighth decade), and better diet(124). The issue of nutrition will be covered in the section dealing with tuberculosis. However, environmental reforms could have affected typhus, since the destruction of lice would have prevented the transmission of the infection. Improved conditions in the home would certainly limit the number of lice, and the measures introduced to inspect lodging-houses, would have reduced or eliminated these potential reservoirs of louse infestation and typhus(125).

The timing of the decline of tuberculosis mortality will never be fully proven, since the decline was evident in the fifth decade, and many

believe it to have been falling since the early part of the century(126). By 1850, however, it was definitely exhibiting a downward trend - at least three decades before other causes of death began to do so(127). McKeown eliminates the possibility of any medical contribution to the decline, which seems reasonable(128). Susceptibility to tuberculosis is genetically determined to a certain extent, according to McKeown(129). However, this does not prove that the trend of mortality was due to genetic selection since the selection effect would have been operating at a maximum upon first exposure; it could hardly account for the dramatic change amid a population continually exposed to the bacteria for centuries(130). McKeown also offers the suggestion that, if tuberculosis is judged to have declined in response to an autonomous change in the causal organism, then the contribution of this factor alone would represent two thirds of the overall reduction of mortality (scarlet fever and tuberculosis combined)(131). McKeown regards this as so mathematically improbable, as to provide grounds for doubt about this factor in the fall of tuberculosis mortality(132). This argument does not seem valid. Just because something is not mathematically likely does not prohibit it from being historically true.

As far as the influence of environmental improvement on tuberculosis is concerned, there are few factors which would have had an effect. The difficulty with assessing the role of these factors (overcrowding and diet) is dating the decline in tuberculosis. Sanitary reform is not reckoned to have been of any significance to tuberculosis(133), since it would not inhibit the spread of the bacteria. A gap exists in the death records 1843-6, which makes interpretation problematic, but the figures

show that the decline was not evident 1837-40, and was very small up to 1842(134). From 1842 to 1847, there is a drop in the death rate from 3 678 per million to 3 114, but bronchitis deaths rose by an even greater margin(135). McKeown suspects that respiratory diseases may have been misclassified, and tuberculosis deaths assigned to bronchitis, thus, the evidence is judged of insufficient quality to prove that tuberculosis was declining before 1847(136). After this date, the facts are not in doubt. Mortality was down to 2 629 per million in 1850, and the rates for bronchitis had shown a declining trend(137). The reduction from 1847 is believed by McKeown to be genuine, and he, therefore, concludes that mortality from tuberculosis was declining certainly from 1847, possibly from 1840, but not between 1837 and 1840, and this fact should instil caution in accepting any decline for the early nineteenth century(138). Farr, however, believed that tuberculosis was declining from 1810; Brownlee dates it from the early part of the century; a suggestion that Greenwood, also, supports(139).

The two environmental factors likely to affect tuberculosis, according to McKeown, are crowding and diet(140). Levels of crowding will determine the frequency and extent of exposure to the bacillus. House building in the nineteenth century, did little more than keep pace with the population explosion in the towns; and the number of persons per house only declined from 5.6 in 1801 to 5.3 in 1871(141). Exposure at work must have increased in the first half of the century, as the numbers poured in, and McKeown does not regard it as likely that it would have decreased much before the end of the century(142). Isolation in sanatoria was not a factor, since by 1900, only a few such

institutions existed, and most patients continued to be admitted to the general wards of the Poor Law infirmaries(143). The infectious nature of tuberculosis was not established until Koch's work in 1882, and it took another decade for this fact to be accepted in Great Britain(144).

Thus, McKeown discounts a reduction in overcrowding, and, hence, exposure, as a factor in the fall of tuberculosis mortality. He is left with diet(145). There is, by no means, unanimous support for the diet hypothesis, as will be shown below, but it is recognised that improvements in the standard of living will have an effect on health(146). What is not certain is which particular features of the improvement have the greatest impact(147). McKeown quotes a rise in the levels of tuberculosis during both world wars, and finds it reasonable to associated this phenomenon with diet(148). For example, in Holland, diet was the only factor to change. Mortality in the post-war era fell again(149).

Milk is a dietary component which needs separate consideration, because of it's role in the transmission of tuberculosis.(150) In the nineteenth century, milk was heavily infected. A survey in 1897-99 of some counties, discovered a 50% incidence of tubercular evidence in cows(151). Therefore, increased milk consumption would affect the mortality from tuberculosis in two ways: first, by increasing early mortality from the bovine forms; and second, by affording a degree of immunity, in later life, to the more dangerous pulmonary forms(152). Data for milk consumption is not conclusive, but the statistics show a rise in the death rate from scrofula and tabes mesenterica, the former

doubling, and the latter increasing fivefold, up to 1850(153). Cronje shows, however, a decline in the rates for non-pulmonary tuberculosis in the period 1851-1910(154), and it is in the latter half of the century that McKeown claims the fall in the tubercular mortality was significant. That is, he wishes to date the decline from no earlier than 1840, and prefers to regard it as existing only from 1847(155). Hence, the information regarding a rise in the scrofula-tabes mesenterica rates up to 1850 would only be significant for one generation, that is, only they would benefit from the supposed increased protection in later life(156). This evidence is, then, debatable, particularly in the absence of any reliable information regarding the levels of milk consumption(157).

McKeown regards the evidence available, as being consistent with the diet theory. There is, and can be, no definite proof about the decline of tuberculosis before the fifth decade, and economic historians are divided about the standard of living before this date(158). However, there is no doubt that conditions had improved by 1850, and continued to do so, through the rest of the century(159). Diet must have been a notable feature of this improvement(160). Mortality fell rapidly from the 1850s, so there is a positive chronological link between dietary improvement and the decline of tuberculosis(161).

McKeown's theory on the decline of tuberculosis, therefore, rests heavily on the dating of the decline, since to fit his theory it must be positively correlated with the rise in the standard of living. However, the standard of living 'debate', is, of itself, a divisive and

controversial issue, especially before mid century; and the dietary factor is a particularly nebulous feature in this controversy(162). In principal, McKeown's hypothesis is based on Holmesian logic(163). Diet is the only factor to fit the evidence, ergo, the nutritional improvement theory, however improbable, must be the truth(164). It is, perhaps, better to regard the retreat of tuberculosis as inexplicable, rather than to hang an argument on such unsatisfactory and unconvincing pegs as these

By assigning the decline in tuberculosis to environmental improvement, McKeown is crediting that factor with half of the mortality decline in the nineteenth century (typhus was thought to be influenced by this too)(165). Sanitary reform claims one quarter; and autonomous change in the character of disease another quarter(166). McKeown reaches this conclusion through a series of eliminations(167). The investigation is limited to the 1-45 age group. He, then, eliminated all, but the infectious diseases from a role in the decline of mortality; reduced the number of those diseases to five; then four, as he discounted smallpox. Finally, the causal factors are slowly eliminated, leaving a conclusion based on the surviving factors.

### **The nutrition debate**

There is a lack of agreement about this factor identified by McKeown. He asserts that, because living standards can be seen to improve from 1850, dietary standards must also have risen from this period(168). F B Smith(169) states that the price and real wage indexes do give an

indication of the ability of the population to achieve better nutrition from this period; and he agrees that the long term rising trend of real wages from 1851 is matched by a falling death rate from tuberculosis. He also includes the evidence of the declining birth rate from 1870s, which would leave more resources available per family member. Smith supports McKeown, in that he recognises that a greater availability of foodstuffs and increased per capita consumption of meat, would tend to support a theory of improving diet in the later nineteenth century.(170)

Woods and Hinde(171) stress the significance of the urban-rural dichotomy, and the need for urban life chances to improve before national mortality can alter radically. Urban living conditions did improve in the late nineteenth century, and began to approach rural standards. From 1900, all areas experienced an acceleration in the increase of life expectancy. If improvements in nutrition are to be credited with a major influence on these changes, Woods and Hinde think it necessary to prove that a substantial difference in the rural-urban diet existed, and that the nutritional levels of the urban population rose especially rapidly from mid century. There is very little evidence to this effect, and they conclude that McKeown has overstated the case for nutritional impact. This is mainly because of his elimination method, which ignores the need for positive evidence to make the conclusions convincing and acceptable. Recent research has illustrated that the relationship between nutrition, mortality, morbidity and fertility is very complicated, and whilst a very poor diet will exacerbate morbidity, increase mortality and debase fertility, improvements only in moderately poor diets will not on their own lead to

dramatic changes in the level of mortality. The evidence regarding Victorian dietary standards is not strong enough to conclude anything.

Woodward is more equivocal(172). He concludes that the industrial revolution brought improvements in real wages to many sections of the population and, particularly in the last twenty years of the century, gains as food prices fell. However, he quotes Oddy(173), who has demonstrated that major shifts in the food consumption of the late Victorian population are hard to find. There is no evidence of a change in the height or weight of schoolchildren; and the working class diet was principally composed of carbohydrates, with any movement towards proteins usually limited to small increases in the consumption of dairy products. Nevertheless, Woodward concedes that any improvement in nutritional standards would give a higher degree of resistance to viral and bacterial infection. Even so, the nutritional status of the body, claims Woodward, can be diminished by disease, and muscle will then be metabolised. Infections of the stomach and gut selectively affect the undernourished and, therefore, disease may act cumulatively on the body to debase it's nutritional status. Woodward also refers to the question of food adulteration, which even though it came under scrutiny in 1872, was still pernicious enough to affect many foods(174). Items like flour and condensed milk, for example, may have been less nutritious or even harmful. The pasteurisation of milk in the 1880s apparently affected bovine tuberculosis levels, but the extent of consumption by children, and general access to pure, unadulterated milk is difficult to establish. The question remains whether small advances in diet coupled

with the reduction in the size of the completed family, could, of themselves, induce a rise in the level of health.

Cronje(175) supports a rise in dietary standards in the second half of the nineteenth century, with real wages rising especially from 1870, just at the period when the decline in tuberculosis mortality was most evident. Towards the end of the century, imported food supplemented and enhanced the diet of many sections of the population, with the influx of tinned and, later, frozen meat being particularly valuable. Meat imports rose threefold 1870-90; and by another 50% 1890-1900, whilst butter and margarine imports more than doubled 1880-1910. Wages are of special importance, since the largest single item in the working class budget was food, and this fact tied nutrition closely to poverty. A survey by the Privy Council in 1863, showed that levels of nutrition varied according to wages, particularly with regard to first class protein. Cronje illustrates the importance of diet on tuberculosis with the evidence from the Lancashire cotton famine, 1861-4. Between 1861-70, the Lancashire phthisis rate fell, but between 1864-7, it rose for both sexes. In Salford, male phthisis rates rose 1861-70 on the 1851-60 level; and the female decline was only very slight. However, in rural Lancashire areas, such as Garstang, the mortality from tuberculosis continued to decline steeply and steadily 1861-70.

The diet factor could explain the sex differential in tuberculosis mortality. Men were customarily given the greater portion of, and the better quality, food to enable them to keep working, leaving the women and children more vulnerable to infection through malnourishment.

Cronje determines that diet was an influential factor, especially when allied to the falling birth rate, but concludes that many other influences must have been at work too.

Anthony Wohl(176) does not believe that the working class diet improved until the last quarter of the century. Between 1877 and 1889, the cost of the national basket of butter, bread, tea, milk and meat fell by about 30%, and it is this period that saw an appreciable rise in nutritional standards. Cheaper food imports on refrigerated and freezer ships, and the development of cheap margarine allied with the falling price of consumer goods, led to a rise in the variety and quantity of the working class diet. By the end of the nineteenth century, weekly bread consumption had dropped to 7lbs per family, and consumption of sugar, meat and milk had risen. This is important, given that the staples of both rural and urban diets were bread, potatoes and tea. Burnett, however, found on the evidence of seventeen surveys comprising 2 500 families, that the improvements did not occur much before the last decade of the century(177). Surveys on London schoolchildren in 1889-1895 revealed that 50 000 were underfed; and the Government of 1904 was shocked at the low standards of nutrition achieved by the masses. Although calorific intake may have been adequate, and this is by no means assured, the working class diet was low in protein and deficient in several vitamins, notably C and D. This may be responsible for the high levels of rickets which prevailed, particularly in towns.

Wohl(178), too, regards adulteration as an important issue. It began to affect more foodstuffs as the century progressed and in 1872, an investigation into bread found that half of it was contaminated with alum. Alum is not poisonous, but does inhibit digestion, thus reducing the nutritional value of other foods. In 1877, the Local Government Board found that a quarter of the milk was watered or contained chalk; and a tenth of the butter, and eighth of the bread and half of the gin contained copper. Milk adulteration continued until the end of the century, even though it was made an offence in 1860. In major cities, between a quarter and a half of the milk was contaminated. In Finsbury in 1903, 32% of the milk had pus in and 40%, dirt. In 1901, 10% of all cows still produced tubercular milk. Meat quality was dubious too. The Privy Council findings of 1862 revealed that one fifth of butchers meat was derived from diseased animals, including those who had died from anthracid and anthracoid diseases. The consumption of measly meat meant that tapeworm was common in the working class. Therefore, it is perhaps fortunate that the population was unable to buy vast quantities of these products, since many were potentially lethal. Nevertheless, Wohl does provide support for McKeown in a quotation from the WHO. They found that where sanitary conditions are rudimentary and disease is endemic, diet may be the crucial factor affecting health.

McKeown may well be correct to associate diet with tuberculosis mortality; but his emphasis on this one factor seems extreme. The proper evidence is not available to give positive proof to his claims; and to argue for it simply because there is no other obvious contributor is unsatisfactory.

### Criticisms of the McKeown hypothesis

On scarlet fever, Mckeown is unchallenged, and his argumants for discounting the influence of medical science seem valid(179). Woodward's attempts to argue a positive role for medicine are not convincing(180).

Woods and Hinde are highly critical(181) of the Holmesian logic of McKeown's work, which they regard as unsuitable for historical analysis. They are also in disagreement over the factoral weighting, especially that assigned to environmental improvement, including diet. Instead, they give emphasis to the sanitary reforms, notably those involving the water supply. Sanitary improvements, they argue, are the greatest contributor to narrowing the distribution of mortality experience between environments, and causing an increase in life expectancy. They also stress the effect of local administration. Thus, "the worst effects of urbanisation were counteracted [by the sanitary revolution] in the last decades of the Victorian era, and this .... alone was sufficient to begin the .... decline in national mortality rates".(180)

Woods and Woodward(183) disagree with McKeown over the need to find direct evidence of the rise in the standard of living in the nineteenth century, and to show that there is a causal link between this and the fall in mortality. They also question the quantative contribution of each factor. The standard of living, though controversial, is generally agreed to have risen in the late nineteenth century, and possibly from 1840. There are still arguments regarding advances during the early

stages of the industrial revolution. Therefore, the second half of the century shows clear evidence of rising wages and falling cost of living, and these improvements seemed peculiarly favourable to the working class. In addition, mortality was falling, but were these two occurrences related?

Woods and Woodward argue that recent studies of underdeveloped countries show that it is possible for life expectancy to rise without economic stimulus or rising per capita income. Substantial change, instead, is likely to be linked to improvements in public and private health and to the provision of basic medical care. Britain may have differed from this model, owing to the high incidence of tuberculosis and the huge impact that that one disease had on mortality levels. However, in theory, increased living standards should create an opportunity for life expectancy to climb, and it is reasonable to associate the two for nineteenth century England.

Woods and Woodward also quibble over McKeown's weighting of contributing factors. Their criticism is that McKeown is making a connection between mere observation of which diseases became less statistically significant and the likely major influence on these diseases, and then quantifying them numerically on the basis of this data alone. For example, to state that bowel infections represented one quarter of the decline of mortality, and that, because sanitary improvements were coincident with this decline, sanitary reform caused the overall mortality to fall by a quarter is too big an assumption.

McKeown's hypothesis is not, therefore, accepted without reservations. However, it is reasonable to state that environmental factors, like sanitary reform and improvements in living standards and, possibly, diet, did affect mortality, especially through their interaction with specific diseases. In order to apply this model to local data, it is necessary to isolate the areas to which it refers, namely a fall in scarlet fever, smallpox, tuberculosis, the typhus and cholera groups, and a slight fall in whooping cough. There should be no major fall in any other group. In addition, it will be hoped to correlate falls in the water-food borne infections to environmental improvements, especially as regards the water supply; and to associate the decline in tuberculosis and typhus, with improvements in living standards. Scarlet fever need not be correlated with any specific measures.

## CHAPTER THREE

### LOCAL STATISTICAL EVIDENCE

The examination of local statistical patterns is intended to provide a comparison with the national figures; and to isolate the individual trends in the general death rates and the cause specific mortality rates for certain diseases. This is necessary to determine whether the same diseases implicated in the fall of the national death rates were responsible for the local declines. The diseases are scarlet fever; tuberculosis; the typhus group; cholera, diarrhoea and dysentery; and smallpox. It is also interesting to observe whether the other principal diseases followed the national pattern, thus, a rise in the mortality from measles and diphtheria, and a minor downward fluctuation for whooping cough.

The local figures are, necessarily, less complete and detailed than the national statistics. Institutions for the collation of such information were established at different times in different places, and for different purposes(1); and the data they collected is of variable quantity and quality. The diversity in the material available means that full comparison is impossible. Nevertheless, it is hoped to develop a sense of the trends in mortality and certain disease categories, and these will be sufficient to demonstrate the degree of comparability between McKeown's national figures and the local statistical evidence; and to identify the beginning of the declines, thus providing the chronological point at which correlation with any

environmental changes should be sought. In the following chapter, the juxtaposition of the mortality pattern with features of the sanitary and environmental reform programme in each town will enable causal factors in any decline to be identified. For ease of comparison, the national figures will be repeated here, alongside the local data.

### Population

The speed of population growth and urbanisation has been explored earlier. Here, the figures for the individual towns studied, along with the national averages are given. These illustrate the different sizes and industrial capabilities of the chosen cities; and their demographic fortunes throughout the nineteenth century:

TABLE 3.1

Population rates per 1 000, selected towns: 1801-1911(2)

Year	Bath	Birmingham	Nottingham	England & Wales
1801	33	71	29	9 061
1811	38	83	34	10 332
1821	47	102	40	12 106
1831	51	144	50	13 994
1841	53	183	52	15 929
1851	54	233	57	17 938
1861	53	296	75	20 119
1871	53	344	87	22 789
1881	52	401	187	26 046
1891	52	478	214	29 086
1901	50	522	240	32 612
1911	51	526	260	36 136

The population statistics show that Bath was expanding until the 1850s, although not at a rate beyond that of the nation, whereafter it's growth stopped and the population remained steady. Birmingham grew faster than the national average rate, increasing it's population by 73% between 1801 and 1831; and redoubling it between 1831 and 1871. It continued to expand down to the end of the century. Nottingham's growth did not greatly exceed that of the nation in general in the first thirty years, and in the 1831-1841 decade, dropped beneath it. There was a major increase in population by 1881.

#### **Mortality rates**

The general mortality rates are of a less consistent quality than the population figures. The national data is fully documented and reliable, but the local statistics are not. For Bath, the annual figures are available only from 1880. Previous to that, the Medical Officer of Health reports were weekly, with quarterly figures being given in each case. For the purposes of this research, therefore, a single week was selected, and the reports studied for that week, 1866-1875(3). This arrangement means that there is insufficient detail available for annual comparisons, and, although mortality rates per quarter are an adequate guide of the general trend, the weekly evidence of cause specific mortality cannot be extrapolated to provide adequate data for analysis. For that reason, cause specific mortality rates will be taken from 1880 onwards, with the addition of the statistics for zymotic mortality from 1875, which were provided in the 1880 report.

## Mortality in Bath

The general death rates for Bath are taken from the Spring Quarter figures, 1866-1875, and from the annual reports of 1880, 1890 and 1900. They are as follows:

TABLE 3.2

Mortality rate per 1 000: Bath and England & Wales(4).

	Bath	England & Wales
1865		23.2
1866	25.7	
1870	31.9	22.9
1875	24.9	22.7
1880	20.4	20.5
1885		19.2
1886	21.1	
1890	19.4	19.5
1900	17.7	18.2

The trend in the death rate at Bath shows a fall from 1870. The figures up until 1870 are Spring Quarter rates, and, so, cannot be taken as wholly representative. However, the spring quarterly rate in 1875 was 24.5, the annual figure, provided by the 1880 report, was 24.9.

Although this cannot be taken as a guarantee of the accuracy of the quarterly rate in relation to the annual rate, it suggests that the quarterly figures are not likely to be totally out of line with those for the year. Therefore, if the statistics are accepted, the Bath rate of mortality did not slip below the average national rate until 1880.

Following a slight aberration in 1836, the mortality plunged to 0.5 per thousand below the national figure by 1900. The 1870 figure for Bath is significantly higher than the rate in 1869, 25.9, and that in 1871 and 1872, 28.1 and 24.1. The scarlatina and smallpox epidemics which were abroad in 1870-1(5), may have inflated the figure, particularly if a large proportion of deaths occurred in the spring. Even if the 1870 figure is discounted, however, the beginning of a downturn in mortality is not evident before the 1870s. Thus, changes likely to affect death from certain causes must be sought from this period.

#### **Cause specific mortality**

It is unfortunate that the annual figures for zymotic disease are not available previous to 1875. The zymotic classification in Bath was taken to include smallpox, scarlet fever, measles, diphtheria, whooping-cough, diarrhoea, and typhoid fever(6). Phthisis was entered under constitutional disease, even after the 1880s when the infectious nature of the disease became known(7).

TABLE 3.3

Mortality from zymotic diseases per 1 000 of the population:  
1875-1900(8).

1875	0.9
1876	3.3
1877	2.1
1878	1.4
1879	1.5
1880	1.9
1886	0.4
1887	0.3
1888	1.3
1889	0.3
1890	0.2
1900	0.7

Infectious disease, omitting phthisis, declined from the mid 1870s, with the 1875 figure representing an anomaly. In 1890, the Medical Officer of Health referred to the unusual prevalence of scarlet fever, and 41 cases were notified that year(9). However, there were no deaths(10). This evidence supports the theory of a change in the character of the haemolytic streptococci, which rendered scarlet fever less lethal. Smallpox registered 39 deaths in 1880(11), but after that it disappeared from the mortality tables, and no cases were notified after 1890 when notification of infectious diseases was introduced(12). Smallpox did not appear in any of the weekly reports 1866-75, and although this in no way proves that it did not cause any deaths, it is perhaps significant in that all the other zymotic diseases appeared regularly in those reports(13). Bath had suffered 361 deaths from smallpox in the 1837-40 epidemic(14).

In terms of respiratory diseases, bronchitis, pneumonia and phthisis all appeared regularly in the weekly statistics, and accounted for significant numbers in the annual death count;

TABLE 3.4

Deaths from respiratory diseases(15)

	Phthisis	Bronchitis	Pneumonia
1880	95	123	54
1890	80	123	62
1900	54	92	56

In 1900, tubercular meningitis and other forms of tuberculosis accounted for a further 23 deaths(16). The Medical Officer in 1900, recorded that tuberculosis had the third highest total in the mortality table, preceded only by heart disease, 93, and bronchitis, 92(17). Two thirds of the tubercular deaths were among those aged under 45 years(18). The high number of heart disease cases was not surprising given the age structure of the population of Bath(19). Although it is not possible to pinpoint the moment of decline in tuberculosis, it certainly appeared to be falling from 1880; and Frederick Field's evidence in 1841, gave a total of 185 deaths from consumption and decline, representing one sixth of the whole mortality for the year(20). In 1880 the proportion was one eleventh, and the actual number of deaths had almost halved by that year. In 1890, phthisis accounted for less than one twelfth; and by 1900, it accounted for one fifteenth of the death rate in Bath.

Infant mortality in Bath was high, but it did begin to decline well before the national levels:

TABLE 3.5

Infant mortality per 1 000 live births: 1880-1900(21)

	Bath	England & Wales
1880	154	153
1885		138
1886	138	
1890	134	151
1900	126	154

The fall in infant mortality in Bath is surprising, since the national infant mortality rate remained steady until the twentieth century. Infant mortality is an extremely sensitive barometer of environmental conditions(22) and these figures suggest that Bath's environment may have become significantly improved in the last twenty years of the nineteenth century.

The available statistics for Bath accord reasonably well with the McKeown hypothesis. Smallpox seems to have been little represented, and scarlet fever evidenced a decline both in deaths and virulence. Tuberculosis mortality fell definitely from 1880, and was probably declining before that. Typhus disappeared from Bath in the last quarter of the century. There were certainly no deaths from it in 1880, 1890 and 1900, and no cases either in the last two named years. The zymotic

class declined as a whole, and in it were contained diarrhoea, typhoid, and cholera. The individual deaths from these three, and measles, whooping cough and diphtheria/croup are given below:

TABLE 3.6

Deaths from certain diseases: 1880-1900(23).

	1880	1890	1900
Typhoid	14	2	--
Diarrhoea	7	8	7
Cholera	--	--	--
Measles	7	2	4
Whooping Cough	14	20	9
Diphtheria	9	2	9

Diarrhoea was the only disease not to register a decline, although the figures could be deceptive since they only show the mortality in three years. It is possible that major fluctuations occurred, and that one or more of the figures represents a temporary respite in virulence. Bath, then, shows statistical correlation with McKeown. In the following chapters, the extent to which the local evidence supports his findings on the causal factors of decline will be explored.

#### Mortality in Birmingham

The death rates for Birmingham are taken from various sources(24), since the Medical Officer of Health reports available only begin in 1880.

TABLE 3.7

Death rate per 1 000: Birmingham and England &amp; Wales.(25)

	Birmingham	England & Wales
1811-20	25.1	23.0
1848	30.1	20.0
1851-60	26.5	22.2
1871-5	25.2	21.9
1880	20.5	20.5
1890	20.0	19.5
1900	20.8	18.2

Although these figures are not evenly distributed chronologically, they do give an indication of the trend in the mortality of Birmingham. Hence, the death rate does not show evidence of decline until the 1870s, but that decline is very steep. By 1880, the death rate is equal to that of the nation, although it never falls beneath it, and, in fact, loses parity in the next decade. The rise in 1900 is noticeable.

#### Cause specific mortality

Figures for the mortality from different diseases are only consistently available from 1870. However, comments made by doctors(26) do give an indication of the levels of certain diseases. Thus, in 1866, typhus was prevailing at a level higher than that for the nation at large(27). In addition, it was stated that deaths from cholera, diarrhoea and dysentery had accounted, throughout the century, for one-twentieth of all deaths nationally. In Liverpool, Leeds and Birmingham, however, the proportions were one eleventh to one twelfth(28). The emergence and

prevalence of diphtheria was also noticed in 1866(29); as was the immunity from asiatic cholera which Birmingham had sustained through each successive epidemic(30).

The death rate from zymotic diseases from 1870 onwards is given below:

TABLE 3.8

Mortality from specific zymotic diseases: 1871-1910(31)

	1871-80	1881-90	1891-1900	1900-10
Smallpox	0.44	0.08	0.10	--
Measles	0.44	0.60	0.52	0.53
Scarlet fever	1.12	0.42	0.30	0.24
Diphtheria	0.22	0.15	0.30	0.30
Whooping cough	0.90	0.66	0.59	0.54
Typhus	0.02	---	---	---
Typhoid	0.33	0.18	0.25	0.15
Diarrhoea & dysentery	1.84	1.27	1.27	1.15
Phthisis	2.48	2.15	1.99	1.29

These figures show a steady decline in the zymotic class of disease. In fact, in 1880, the zymotic group (smallpox, measles, scarlet fever, whooping cough, typhoid, diarrhoea, cholera and fever) accounted for 16.3% of all deaths (32). In 1890, the percentage was 13.7% (33); and in 1900, 12.6%(34). Within this group, smallpox and typhus ceased to claim any victims(35). Measles did not show any evidence of decline, nor did diphtheria, which indeed increased. Whooping cough and scarlet fever declined, scarlet fever dramatically, and whooping cough fairly substantially. Typhoid, except for a rise in the 1891-1900 period, also

evidenced a fall in mortality; as did diarrhoea and dysentery. Phthisis showed a steady decrease, too, accounting for 8.8% of the total mortality in 1880, but 7.5% by 1900(36). Nevertheless, the respiratory diseases were still a powerful force. Bronchitis, pneumonia and phthisis caused 28.9% of the whole mortality of Birmingham in 1880, 30.1% in 1890 and, still, 27.9% in 1900(37)

TABLE 3.9

Mortality from respiratory diseases: 1880-1900(38)

	Phthisis	Bronchitis	Pneumonia
1880	718	1175	453
1890	812	1299	791
1900	817	combined with pleurisy 2227	

The infectious diseases in Birmingham follow the McKeown hypothesis in trend. It is unfortunate that the figures for earlier in the century are not available, since it is impossible to state when each disease began to fall, a particularly important question with regard to phthisis. Nevertheless, the statistics given show that there was a very substantial drop-off in mortality from scarlet fever and typhoid after 1870. Typhus was of little impact even in 1871-80. The smallpox statistic for 1871-80 may be inflated because of the epidemic of 1871-2(39). The decline in whooping cough seems rather more substantial than McKeown suggested(40), but it is only because of the fall between 1871-80 and 1881-90. After this point, the decline slows significantly.

Without the figures for earlier in the century, it is impossible to compare the rate for 1861-70, to determine whether 1871-80 represented a sudden peak in mortality. Given that whooping cough was often an accompaniment to other zymotic epidemics, this is possible.(40)

### **Infant mortality**

The statistics for this are, again, scarce. However, in 1841, it was stated that the infant mortality rate in Birmingham was very high, exceeding that of the metropolis and the agricultural districts, although not of the other large towns(42). However, in 1880, the rate was 184.3 per 1 000 live births(43). Unfortunately, because of the lack of data regarding birth rates, the same statistic cannot be provided for the 1890 and 1900 report. Instead, the proportion of all deaths represented by infant deaths is given, hence, 34.1% in 1880; 27.1% in 1890 and 30.9% in 1900(44). These figures are not as illuminating as those per 1 000 live births, but they seem to show that infant mortality was not declining in Birmingham in the nineteenth century.

### **Nottingham Mortality.**

The general death rates for Nottingham are derived from the sanitary committee reports 1855-1870, and from the annual reports of the Medical Officer of Health for 1885, 1890 and 1900.

TABLE 3.10

Mortality rate per 1 000: Nottingham and England &amp; Wales(45).

	Nottingham	England & Wales
1855	25	22.6
1865	24	23.2
1870	24	22.9
1875	26.6	22.7
1880	23.3	20.5
1885	18.2	19.2
1890	19.0	19.5
1895	18.5	18.7
1900	19.2	18.2

The trend, therefore, shows a decline from the mid 1870s, with a swift fall over the decade 1875-1885. Thereafter the decline slows. Not until 1885, does Nottingham's death rate fall below that of the country, nor indeed below the 23 per 1 000 limit designated by the General Board of Health as constituting an unacceptable level of mortality(46). In 1885, Nottingham attained it's lowest mortality rate on record, by far(47), and was also able to claim the third lowest mortality rate out of the twenty-eight great towns of England and Wales(48). This achievement was sustained in 1890, but, by 1900, Nottingham had fallen to nineteenth out of the thirty-three cities termed great towns(49). Significant changes in conditions should be noticeable in Nottingham around the 1870s.

#### Cause specific mortality

Figures for zymotic diseases are available from 1855, and the zymotic mortality rate from 1856. The zymotic death rate included smallpox,

measles, scarlet fever, diphtheria, whooping-cough, 'fever', diarrhoea and phthisis.

TABLE 3.11

Death rates per 1 000 from the principal zymotic diseases:  
1856-1900(50)

	Nottingham	England & Wales
1856-60	5.98	4.03
1861-5	3.83	4.22
1866-70	4.34	4.08
1871-5	4.30	3.76
1876-80	3.0	2.94
1881-5	3.22	2.32
1886-90	2.39	2.25
1895	2.64	2.14
1900	2.35	2.0

Apart from the quinquennium 1861-5, the local zymotic mortality exceeded that of the nation. The specific rates are as follows:

TABLE 3.12

## Specific zymotic death rates per 1 000: 1856-1900(51)

SX = smallpox; M = measles; SF = scarlet fever; DIPH = diphtheria; WC = whooping cough; F = fever; DIA = diarrhoea; P&TB = phthisis and tuberculosis.

	SX	M	SF	DIPH	WC	F	DIA	P&TB
1856-60	0,21	0,8	1,08	0,13	0,76	1,02	2,0	3,22
1861-5	0,09	0,43	0,98	0,12	0,51	0,78	1,09	3,19
1866-70	0,07	0,44	0,73	0,09	0,51	0,92	1,57	2,78
1871-5	0,79	0,31	0,53	0,02	0,26	0,84	1,53	2,42
1876-80	---	0,35	0,62	0,03	0,43	0,34	1,06	1,85
1881-5	0,06	0,41	0,77	0,12	0,46	0,31	1,09	1,99
1886-90	0,01	0,42	0,11	0,06	0,45	0,31	1,04	1,52
1895	---	---	0,23	0,04	0,14	0,24	0,60	2,10
1900	---	0,19	0,23	0,12	0,43	0,32	1,08	2,02

These statistics show a definite fall in the mortality for smallpox, measles, scarlet fever, fever, and phthisis and tuberculosis. The diphtheria rate was variable, but the 1900 total was the same as that for 1881-5, and only 0.01 down on 1856-60. Whooping cough declined from 1856-60 to 1871-80, then rose again, and steadied at that level.

Diarrhoea maintained a fairly high rate, although there was no repetition of the 1856-60 level. Thus, the individual diseases conform to McKeown's pattern, with phthisis and tuberculosis declining steadily from the 1850s, although there was a pick-up in the rate in the last five years; smallpox disappeared, and scarlet fever mortality fell from 1856-60. Diphtheria and diarrhoea failed to decline, and the fall in the whooping cough figures was small. Only measles fails absolutely to fit the McKeown model, and even then, it maintained a high level until 1890. Fever is too nebulous a term to be analysed here.

TABLE 3.13

Deaths from certain diseases: 1855-1900(52).

	Phthisis	Pneumonia	Bronchitis	Typhus	Typhoid
1855	216	65	---	--	--
1865	218	95	185	26 (inc typhoid)	
1869	175			14	
1875				6	61
1880				--	58
1885	392	225	568	--	42
1890	400	314	514	--	58
1900	339	343	504	--	75

The respiratory diseases were not receding in intensity, but typhus vanished from the death rates, and typhoid appeared to be declining until 1885. The figures representing the percentage of the total deaths substantiate this picture, typhoid accounted for 2.8% in 1875; 1.2% in 1880; 1.0% in 1885, and then rose to 1.4% in 1890, and again to 1.6% in 1900. This fails to concur with McKeown's national findings. The Medical Officer of Health, however, identified a very localised picture for enteric fever(53), which may explain the anomaly in a manner compatible with McKeown's theory.

Scarlet fever declined in mortality, and, seemingly, in terms of virulence. In 1885, the Medical Officer of Health described scarlet fever as continuing the remarkable decrease since 1881(54); and in 1890 reported that mortality for the disease had fallen to 66% below the past ten years average(55). Incidence was still high, however, 390 cases in 1885; 984 in 1890; 1 250 in 1895; and 1 394 in 1900(56). This argues that the virus was had become less lethal.

## Infant Mortality

The level of infant mortality in Nottingham was high throughout the century, never falling below the national rates. However, there did appear to be a lull, 1885-1895, although this phenomenon was matched in the national rate, so may represent a country-wide fluctuation. If this were so, then it is unlikely to be a sign of a sudden drastic improvement in the environmental conditions of Nottingham. In terms of the other large towns, Nottingham had the sixteenth lowest mortality out of twenty eight in 1885; the fourth lowest in 1890; but in 1900, it was stated that, for the period 1890-99, Nottingham's average infant mortality had been 179 per thousand, and that of the thirty-three large towns, 172.(57)

TABLE 3.14

Infant mortality per 1 000 live births: 1856-1900(58)

	Nottingham	England & Wales
1856-60	209	153
1861-5	192	151
1866-70	200	159
1871-5	192	153
1875	199	158
1880	201	153
1885	158	138
1890	158	151
1895	189	161
1900	196	154

Nottingham shows a substantial correlation with McKeown's national statistical trends.

## CHAPTER FOUR

### PRE-DECLINE CONDITIONS.

The mortality pictures suggest that the death rate did not begin to decline in any of the towns before 1870. Therefore, the first part of the following chapter will be dedicated to an evaluation of local conditions before the downturn, with major concentration on the evidence up to mid-century.

### BATH

Bath was a flourishing spa town of the eighteenth century, and, at the end of the century, it was bigger than towns such as Liverpool, Manchester, Birmingham, Bristol and Nottingham(1). The influx of visitors continued in the first few decades of the nineteenth century and Bath's population rose by 16%, 1801-11 and by 21%, 1811-21, rates which are roughly equivalent to the national growth(2). Wealth was still being attracted into the city; the Kennet and Avon canal opened in 1810(3); and the Gas Light and Coke company was established in 1817(4). The 1820s saw speculation about a railroad to connect Bath with Bristol(5); and Bath was still a social success in the 1830s, with its baths crowded; its concerts unrivalled; and its theatre judged second only to that of London(6). The occupational index(7) shows a town geared towards providing for the pursuit of leisure. In 1831, the occupation of men over 20 years [one sixth of the population] was as follows:

14.0% capitalists, bankers and other educated men  
 14.6% building trade  
 4.8% furniture and coachmaking  
 7.2% shoemakers  
 4.7% tailoring  
 9.0% domestic service  
 7.7% cloth work  
 20.0% non-agricultural labourers  
 1.5% labourers  
 30.3% other: mainly retail, including some craftsmen.

As Bath declined as a leisure resort, it became a place of residence and retirement for the affluent middle classes(8), and, consequently, the opportunities for domestic service rose accordingly. The employment of women in 1851 in Bath was as follows:(9)

Domestic servant	7 751
Milliner	1 829
Washerwoman	1 436
Seamstress	509
Shoemaker	441
Staymaker	129

There was a preponderance of women in Bath, 32 517 compared to 21 737 men in 1851(10). This was, perhaps, partly because of the vast market for domestic labour.

Bath did not suffer the troughs of depression related to trade cycles and industrial slumps, since it was not dependent on any industry and was comfortably buffered from sudden economic crises by the vast population of affluent middle class residents(11). G S Gibbes declared in 1837 that Bath was 'not a city of trade. No manufacturer worthy of notice is carried on within it's limits, nor is it a resort of commerce .... Bath is best fitted for the retirement of individuals with independent incomes .... trade in Bath consists, principally, in the sale of articles connected with refinements, rather than the necessities of life'(12). By 1841, ninety percent of the working population were involved in providing goods and services for the wealthy consumers living in Upper Town and Bathwick(13). The railway built in the same year had created a premium for labour in the period 1837-1841(14).

Bath did, however, have an industrial zone, and this was located on the south side of the city, where the mills and foundries jostled with the working class houses(15). The incipient industrialisation of the 1830s had been evident in St James Parish, with the growth of metals and engineering connected with Stotherts ironfoundry(16). By 1851, this enterprise employed 540 men(17). Artisans concentrated in St James and the manufacturing and factory industries of the south influenced the growth of population in the Lyncombe and Widcombe parish(18). The south side also formed the crux of the communications network(19). The woollen industry, providing fine woollen cloth, was located in Lyncombe and Widcombe, distinguishing this district occupationally from the rest of Bath(20). All 565 men engaged in the trade in 1831 resided in that parish, along with the railway labourers and coal carriers(21). The

unskilled and semi-skilled labourers lived in the Holloway district and in the Dolmeads, with the remaining poor crowding into tenements and houses in Galloway buildings, Chapel street, Thynne street and various courts and alleys around the city(22).

The slums were notorious. Situated on the low-lying flood plain of the Avon, they were damp, ill drained and unhygienic(23). Avon street was particularly bad, registering more than half of the total mortality from cholera in Bath in 1832(24). Reverend Elwin observed that whatever contagious or epidemic disease prevailed, Avon street formed the foci of it's ravages; and 'everything vile and offensive is congregated there. All the scum of Bath - prostitutes, beggars, thieves, - are piled up in the dens .... of which the street consists(25)'. Bath's social strata: 14% leisured and professional; 26% tradesmen; 33% artisan, from printers and coachmakers to shoemakers; and 19% unskilled labourers, translated into residential division, with the poorest of the population living in the least salubrious neighbourhoods(26). The ravages of disease were likely to follow the paths of such social segregation, as illustrated in the evidence of cholera in Avon street(27).

Sir Henry T de la Beche(28) found Bath, in 1845, to be well built on the whole, the streets being generally airy and good, with the exception of the lower and older parts of the town. Courts and narrow alleys was not very frequently found. His investigation revealed that there was no plan of the sewerage of Bath. The constituted authority for about one fifth of the city area, the commissioners for the out-parish of Walcot, had powers to construct sewers, but the remaining four-fifths were

without such provisions. Scavenger duties kept the town fairly well cleansed, but there was a want of co-operation between the many commissions for this purpose, leading to unnecessary expense. This expense was borne by the landlords or tenants and not by any public body(29). Courts and alleys were cleansed twice a week, using carts to carry the refuse away. Dust boxes were emptied twice a week, this refuse being sold in the country for manure.

Mr George, the town clerk of Bath(30), argued that the streets, courts and alleys were paved and had generally sufficient inclination for surface drainage to occur. He knew of no instances where stagnant pools existed. Main sewers were located in the streets or in the vicinity of the houses. Sir Henry, for example, found that a main sewer ran between Avon street and Milk street, which was swept clean by water from the hot baths every day(31). There were no public necessaries, but the houses were well-supplied with privies, emptying into drains which rarely required cleansing, although some of the main sewers were occasionally obstructed. Traps to prevent the escape of noxious vapours were in frequent use; and all drainage led into the River, except for a few old houses and some new ones in the suburbs, which had cesspits(32).

Sir Henry discovered(33) that, although Milk street and Avon street were fairly wide and well paved, the houses therein were usually dirty and ill ventilated. The courts located behind these streets were also ill cleansed. Selden street in Southgate was only 4ft wide and dark, but it was well drained and clean, despite being represented as not healthy. There were no regulations regarding building, and a court had recently

been constructed, being narrow and ill-ventilated, although clean. The chief centre of evil was the Dolmeads, sited on the alluvial flats and subjected to frequent flooding. The houses in New street, Dolmead, were so sunk as to be beneath the high water mark of the Avon. The drainage was imperfect, despite the presence of a sewer provided at the houseowners expense. The highways in that district were in a bad state, the poorer houses were ill-ventilated, and there was much personal dirt. However, there were no cellar dwellings.

Sir Henry(34) learned from the Police commission that twenty-seven lodging houses existed in the lower part of town, the character of the keepers being generally bad. The number of vagrants sleeping each night in each house was estimated by the police to be between four and six. Therefore, if five were reckoned to stay in each house, that represented 135 persons; and if each vagrant remained for two days, that amounted to about 20 000 vagrants passing through Bath each year. The lodging-house bed cost about 3d per night, and each bed usually accomodated two or more, without regard for sex or age. The influx was greatest during the season, and it was feared that the indiscriminate charity that prevailed was in part responsible. The deserving poor travellers could apply at the Refuge for the Destitute, where they might be given food and a bed for the night.

The provision of public baths was plentiful(35). The Kings and Queens Bath cost one shilling; but the Crossbath was cheaper at 6d, and was meant to cater for the less affluent. Sir Henry reported that this

seemed to be well patronised, with approximately 3 500 persons using it each year, and that number was rising.

Investigating the domestic water supply, Sir Henry found that water was obtained from springs or wells, sunk in different places(36). Although there were eight water companies, several of them supplied a very limited number of houses, being confined to the boundaries on which the spring had its rise. No company had the protection of an Act of Parliament, and were, therefore, unable to provide competition by extending pipes into the neighbouring districts(37). Mr George(38) regarded the water supplied as very pure, although no filters were used. A supply was also provided by the corporation, and, of the 8 200 houses in the town, 3 000 were served, each having a separate cistern, except for small courts which had a common cistern. The corporation operated six conduits, gratuitously, for the poor, which were open for five hours a day. In Edinburgh, in 1818, the poor lost their free water supply when piped water was introduced into the city. The well-to-do installed water-closets and ceased to use their pump wells, the excess supply from which had been allowed to flow down the High street(39). The city and suburbs of Bath were generally well supplied, although no standpipes existed for cleansing the streets, and the annual charge, depending on the cistern capacity, was 27 shillings for 40 gallons per day(40). The average cost in the small towns of southern England, in 1861, was 2s 6d per 1 000 gallons(41).

Mr Frederick Field, a surgeon in Bath, gave evidence to the Commission of 1845, dealing with mortality and disease(42). He found that the

proportion of mortality over sixty years was large, but probably only because the proportion of the elderly population exceeded that of any other place of like size and population. Deaths from consumption and decline represented one sixth of all deaths. Infant mortality was higher in the poor districts than the rich. Field regarded Bath as a healthy place, where the mortality from consumption was no higher than the national rate, and fever deaths were very few. He attributed the large mortality under five years of age in poor districts to the great carelessness exhibited by the poor in regard of the health of their children. There were many physical causes of disease in Bath, such as the floods in the lower part of the city; the imperfect drainage; and the crowded and dirty nature of the dwellings inhabited by the poor. Disease would, he thought, prevail to a great and fatal extent were it not for the very active benevolence exercised toward the poor. General mortality per district was given as below:

**General mortality per district**

Parish	population	deaths	deaths under 5	deaths under 1	deaths from TB	deaths from fever
Lansdown	14 111	297	88	47	47	8
Walcot	12 102	250	80	45	49	7
Abbey	12 103	216	75	48	35	8
Lyncombe & Widcombe	9 920	163	72	50	33	11
Bathwick	4 973	81	20	10	9	4

Lansdown had a 2.1% mortality, and contained Milk street and Avon street; Walcot registered 2.0% mortality; Abbey, 1.7%; Lyncombe and Widcombe, 1.6%; and Bathwick, 1.6%. The Dolmeads, according to Field, did not return a high mortality.

The picture of Bath is one of relative health at the mid century point. A population which is fairly well housed and served by sanitation provision of reasonable quality. Nevertheless, Bath contained some appalling slums, and epidemics hit hard there. The smallpox outbreak of 1837-40 killed 361; and cholera killed 49 in 1832 and 90 in 1849. (43)

## BIRMINGHAM

Birmingham had a great diversity of occupation, with over 500 classes of trade, which gave elasticity to the economy of the town(44). The work was generally located in small workshops, not in large factories, and this did not change during the nineteenth century(45). Much of the labour force of the town was skilled, and relatively well off in economic terms(46). The industrial base of Birmingham remained the metal finishing trades, but the town adapted to changing conditions throughout the century, and developed into an important centre for services and trading facilities(47). By the end of the nineteenth century, it had become the commercial capital of the midlands(48). Birmingham's ability to change with the tides of industrial fortune enabled it to evade the devastating effects which sectoral industrial declines and depressions had on less economically diverse towns, since even in periods of slump in one industry, others were flourishing(49). The variety of trade and occupation of the population led, in the opinion of the Sir Robert Rawlinson, to a more equal and general diffusion of wealth(50). There were few, if any, millionaires connected with trade, but the workpeople were comparatively independent and self-reliant(51).

Birmingham's physical growth began in the eighteenth century, and maps of the period reveal courts and alleys already in existence by 1730, and infilling of gardens and orchards taking place(52). By 1775, more than a third of the housing stock was built back-to-back(53). By the late nineteenth century, salubrious city centre accomodation had disappeared,

as the pressure of population led to the utilisation of all available space and the multiple tenancy of houses for both domestic and industrial purposes(54). In 1840, the Health of Towns commission(55) found Birmingham to be in favourable contrast to other large towns. It's employments did not seem injurious to health; the custom of each family residing in their own separate dwelling was conducive to good health; and the good site, with it's dry, absorbant soil was of positive advantage to health.

The health of Birmingham was well scrutinised in the first half of the nineteenth century, with four surveys being undertaken in the 1840s(56), and, another in 1852(57). Josphe Hodgson was convinced, in 1840(58), that Birmingham was superior to many of the industrial towns in Lancashire, but by 1849, Rawlinson found Birmingham to be less healthy than it could be(59). The inhabitants of the town in 1840, lived neither in flats above one another, nor in cellar dwellings, and, although close courts did exist in the old town and many houses were back to back, Hodgson believed that the injury attendant on such accomodation was dependent on the size of the rooms, number and size of windows and the spaciousness of the court itself(60). Birmingham courts were built either back-to-back, or in the expectation of being so, and, therefore, there were no outward facing windows to facilitate through ventilation(61). In 1841, a committee of physicians and surgeons surveying the town, concurred with Hodgson that the system of back-to-back housing did not necessarily cause injury to health and afforded economical accomodation to the working classes(62). This type of building, in their opinion, was much preferable to the crowding of many

families into single large houses(63). However, the building of the railway that occurred in the 1840s, destroyed much working class housing, which was not replaced. Thus, overcrowding had occurred in some places(64).

The committee of 1841(65) found that although the principal streets were well paved and drained, the lesser ones and the courts were dirty and neglected, with pools of stagnant water in them. The courts were very numerous and widespread, containing a large proportion of the poor. By 1849 there were 2 000 courts containing 50 000 persons(66). The old courts were narrow, filthy, ill-ventilated and badly drained, although the newer ones were generally not so(67). Perhaps Hodgson had not visited the old courts in 1840. Such courts contained between four and twenty houses, and were three storeys high and built back-to-back. The court entrance was narrow, 3-4ft wide, and covered in, thus providing no means of ventilation and preventing the entrance of carts to remove refuse. The refuse had to be emptied into the street from where it was carried away in the morning. A proportion of the matter was always left behind(68).

A washhouse, ashpit and privy stood at one end of the court and, not infrequently, there were pigsties and heaps of manure too(69). Hodgson believed the practice of keeping pigs in the houses and courts ought to be prohibited and prevented(70). The committee of 1841 had noted the practice too(71), stating that they believed that even if passages were opened between the back-to-backs, they would be filled with livestock and their filth. Hodgson was also in favour of a system of supervision

for privies, ashholes and receptacles of filth, since these were in a very bad state in some places(72). The committee of 1841 found the old court privies filthy, many being in a condition where they regarded it as inconceivable how they could be used, being without doors and overflowing with filth. By 1849, the privies, where they existed at all, were a nuisance(73). They were often built against dwelling houses, so that filth penetrated through into cellars and pantries(74). The privies of many manufactories were similarly inadequate, and those for men and women were often adjacent(75), whence it was impossible to visit without being observed and taunted by the people in the workshops. Many families had recourse to utensils in the home, which were emptied at night, and the offensive nature of many houses was traceable to this practice(76).

Birmingham had great natural advantages in respect of drainage, since the hillside siting facilitated natural drainage, as did the sandy, gravelly soil which absorbed any moisture that did not drain off(77). Some noisome drains existed in 1840, but fewer than had done so in the cholera year, when the epidemic had created the impetus to clean and cover the drains at the expense of the Cholera Board(78). Commissioners now bore this expense, and the precautions had been beneficial to some degree(79). However, the committee of 1841 found that the old court drains were often above ground, uncovered and discharged into the gutters of the street(80). By 1849, Rawlinson(81) found that ditches full of refuse and nightsoil were commonplace in the town. Hodgson had pointed out that, although the commissioners did have powers to levy fines for the non-removal of refuse from ashholes, if the offender

could not afford to have the ashhole emptied, then financial penalties would be of no use. He believed that the commission should be empowered to have it done at the expense of the landlord or ratepayers(82).

By 1840, a surveyor had been appointed who was improving the roads and drainage conditions(83). Difficulties arose from the lack of any plan of sewerage, and the fact that not all of the sewers laid had been put in at sufficient depth. Therefore, in many places they were too high to drain the cellars(84). Few of the sewers of the town were large enough to admit a man to cleanse them, so that blockages only came to light when a stagnant pool of corruption had accumulated underground and become a nuisance(85). Birmingham was fairly well provided with sewerage, but Hodgson thought that additional powers were necessary, since, although the connection of domestic drainage to the main sewers was permitted, it could not be enforced(86).

The lodging houses represented a source of disease in Birmingham, as well as being a sink of immorality, crime and misery. The police could not conceive of there being any worse such places in England(87). The 1841 committee found three types: mendicants, irish and low(88). They were all neglected, squalid and crowded. The mendicants houses being frequented by beggars, trampers and thieves, who slept in rooms and beds together, regardless of sex(89). The 252 Irish lodging houses catered for resident labourers employed in the building trade, and they were principally located in the old town(90). There were a further 187 lodging houses, which housed the prostitutes(91). These houses were not conducive to health. Hodgson recommended that a system of inspection be

introduced, as well as regulations for the cleansing of such premises and for the containment of any contagion arising therein, a conclusion which Rawlinson also reached in 1849(92).

The water supply was judged ample in 1841, with pumps existing in almost every court(93), but by 1849, the supply was limited and poor in quality(94). In 1849, domestic water was made available from private wells and pumps, and some public pumps and private water carts, whose provision was expensive at 10s 5d per 1 000 gallons. Rawlinson reckoned that a proper scheme of supply could furnish the same quantity for 3d(95). A public company also existed, which supplied some portions of the town three times a week, with about one million gallons(96).

However, offensive matter had infiltrated into the substrata and was polluting the water at it's source, so that many wells which had once yielded pure water, were, by 1849, providing a supply unfit for anything other than washing. The public pumps yielded the same source, but from a lower depth, the bright appearance being no guarantee of purity(97). The waterworks company established in 1825 provided water from it's reservoirs served by the River Tame. There was no filterbed, but the water passed through canvas strainers. One third of the town was supplied from this source(98). In 1841, this waterworks had supplied water to all parts of town and their supply was seldom insufficient in quantity(99). Evidently demand had outstripped the capacity of the waterworks to supply.

The Rea was in an appalling state, and could be regarded as the main sewer of the town. The flow was sluggish and the quantity of water

insufficient to wash away the refuse it received on it's route through the town. In hot weather it was very offensive, sometimes being covered in a scum of decomposing matter.(100)

Rawlinson found Birmingham mortality high, but less so than in other large towns(101). However, there was evidence that the health of the town was deteriorating, since, in 1840, Hodgson had been of the opinion that fever was rare in Birmingham(102). The 1841 committee of physicians and surgeons had been unable to designate any particular district or type of abode as being more susceptible to fever, although it had been noticed that the poor were more often the victims of epidemics(103). However, Rawlinson was able to detail a long list of localities which could be fixed as the focus of epidemic, endemic or contagious disease, in particular crowded lodging houses and courts(104).

John Postgate's 'Sanitary Aspect of Birmingham' printed in 1852(105) found the rate of mortality in Birmingham, despite it's natural advantages of site, soil and generally prosperous community, to have risen steadily over the preceding few years. By 1852, it had almost equalled the rates for other large towns. The last report of the Registrar General had found diarrhoea rife and several cholera cases; and had considered that the high rate of mortality proved a neglect of sanitary measures. Postgate instanced streets containing heaps of dirt, which remained for a fortnight or more; gutters so full of puddles that the street was virtually impassable; and even some of the better streets were dirty and rarely cleansed. Open sewers were inviting epidemic

disease and were 'a disgrace to a large town abounding with wealth and intelligence'(106).

The evidence for Birmingham shows a city which had been lauded as healthy in the early years of the nineteenth century, but which had quickly deteriorated to the same levels of squalor and degradation as the other large towns. Hence, by 1849, the previous immunity to fever, quoted by Edwin Chadwick in 1842, had dissipated, and nests of contagion could be identified. By 1852, disease transmitted by polluted water was common, suggesting a sustained contamination of supply.

## NOTTINGHAM

In the early nineteenth century, Nottingham suffered from two major disadvantages: The narrow economic base, which depended on only two industries; and the physical restriction on the size of the town. Nottingham was dependent on lace making and hosiery, for the majority of it's economic welfare(107). By 1830, lace making was the more profitable and progressive, whilst hosiery was declining(108). These two industries were virtually the sole employers of the growing population and there were simply more labourers than the market could support(109). The hosiery industry remained unmechanised, with the stocking-frame almost unchanged since Parson Lee invented it in the sixteenth century(110). The industry was located in the home or in small workshops rather than in factories(111). There was no incentive to change production methods because the hosiery markets were glutted and labour cheap and plentiful. The workers over-produced, causing the industry to become stagnant, uncompetitive and depressed(112). A reaction to this state of affairs occurred in 1811 with Luddism. Machines were smashed in an organised protest by the Nottingham stockingers, but, although many of the employers were sympathetic to their plight, the government cracked down and hanged seven men for machine breaking in Loughborough(113). Luddism died out through fear and the distress continued.

Although lace making boomed between 1821 and 1831, this industry too was somewhat insecure(114). It was more progressive, factory based and technologically advanced than hosiery, but at least 40-50% of the lace

production was destined for overseas sale(115). Therefore, Nottingham was peculiarly vulnerable to the fluctuations of British overseas trade(116). The textile industries also suffered from seasonal unemployment because of the export mechanism. European sales were made mainly at the spring fairs, American sales in autumn(117). This narrow economic base and the problems faced by the textile industries meant that Nottingham always had a high proportion of people on poor relief(118). In boom years, the staple industries attracted workers, increasing pressure on living space(119). In years of depression, the congestion was further aggravated by poverty and pauperism on a large scale. The narrow economy, based as it was, primarily in the export market, naturally resulted in employment insecurity, and the political machinations which delayed the advent of enclosure, exacerbated the situation with regard to environmental conditions.

The restriction on the size of the town was the result of the disinclination of the corporation to enclose the common land and the city was surrounded on three sides by open fields(120). Admittedly, some of this was in the hands of private owners, but the municipal corporation owned one third or 654 acres, and also controlled the Forest and Mapperley common which were open all year, and Hunger Hills which were used as gardens. The Coppice farm, 120 acres, was let, on an annual basis(121). Thus, the corporation was responsible for more than 1 400 acres of land surrounding the cramped and overcrowded city(122). It certainly appears iniquitous that, at a time of crisis, with the population rising faster than ever before, the corporation should refuse to enclose the land available outside the boundaries. Among the

reasons for this intransigence was the pressure of opinion from the burgesses or freemen of the city, who were against enclosing the common fields, despite the fact that use of the grazing rights had declined steadily over the years(123). Many of the burgesses of Nottingham were artisans and tradespeople and they were determined to retain their traditional privileges, despite the growing misery within the city(124). This was not because they were unaware of the squalor and filth of the overcrowded courts. In fact, the framework knitters, who represented the largest group of burgesses(125), were amongst the poorest in the city and many actually lived in the slums, but they saw their common rights as a mark of status(126).

A second factor impeding enclosure was the influence of the slum owners on the council(127). Several of them were actually members of the corporation and, therefore, had some leverage. These property owners had a vested interest in preventing the enclosure of the common grounds around the city, since the demand for housing was outstripping the supply, thus putting accommodation at a premium(128). Property owners feared that the enclosure of the common lands would end their monopoly on housing and bring down rents(129). This attitude ensured that the squalor and overcrowding increased, with three thousand dwellings being erected between 1821 and 1831, many of which were sub-standard(130). Lean-to's and cellars were rented out to families desperate for accommodation(131), and, by 1832, there were about 8,000 back to back dwellings in Nottingham, arranged in courts with a single entrance(132).

The municipal corporations act of 1835(133), ended the reign of the oligarchical and closed corporations, and replaced them with councils elected by the resident householders. In Nottingham, the corporation comprising seven aldermen and twenty four councillors, elected by freemen, was replaced by a council of forty two, elected by ratepayers(134). The first election returned the mayor, Thomas Wakefield, to office, and, also, William Enfield, as town clerk. Five of the six aldermen survived election, but only three of the twenty four councillors. Nevertheless, the council retained it's domination by whig dissenters, and the same select occupations were represented as before, namely hosiers [one third], shopkeepers, professionals and 'gentlemen'(135). However, the new corporation proved less hostile to enclosure, and only the freemen and slum property owners continued to adamantly oppose it(136). Their tenacity and remaining influence delayed general enclosure until 1845, and when government permission was finally solicited, and gained, this was done by independent citizens, and not the council(137). It was, even so, the lessening of the influence of the burgesses in the corporation, coupled with the pressure of public opinion, that finally enabled enclosure to proceed. Whilst most of the enclosed land was destined for building, provision was made for land to be left free for the use of the townfolk. An arboretum of twelve acres; a walk named Elm Avenue, of ten acres; and six acres for recreation grounds were provided. In addition, four acres were left for conversion into a cemetery(138). The enclosure act(139) laid down specifications for construction of new buildings in an attempt to abort any repetition of the squalid conditions prevailing in the old town. It cannot be presumed, however, that these regulations were adhered to,

since the Act was merely a statement of intent. In fact, back-to-back housing, without drainage, was erected on the Meadows, although the notorious court construction was avoided(140).

The commissioner, J R Martin, visited Nottingham in 1844(141), and stated that 'nowhere else shall we find so large a mass of inhabitants crowded into courts, alleys and lanes as in Nottingham, and those too of the worst possible description'. A report by the sanitary committee of 1847-8(142), stated that 'nuisances greater than could have been believed' prevailed in the poorer areas of Nottingham, and fever and other such diseases were endemic in those places. Two hundred cellar dwellings were discovered in 1844, located beneath back-to-back houses, and consisting of rooms eleven or twelve feet square and six feet and eight inches high. In these hovels dwelt eight to twelve people. None of the houses had damp courses and the walls were thin and often porous(143). Dr Williams, an assistant with the Commission, was vociferous in his disgust over the state of the poor in Nottingham. The rich, he believed, were 'rightly served' when disease invaded their homes, since they had done nothing to prevent the establishment of such appalling housing conditions as existed(144). One nine acre area contained 883 houses, into which were crammed 4 283 inhabitants(145). Another particularly bad locality was Broad Marsh, whose population Sir Richard Phillips likened to maggots in carrion flesh(146).

In the early nineteenth century, the water supply, impure and unfit for consumption, had been hawked around the streets by water sellers or pumped to standpipes erected by the corporation(147). In Narrow Marsh,

for example, water cost a farthing per ten gallon bucket, but it was often putrid(148). The River Leen, despite being the town's main sewer, was often used as a source of water by people reluctant to rely on the intermittent and polluted supply at the standpipes(149). This changed in 1832, when the waterworks constructed by Thomas Hawksley opened. The works supplied 8 000 houses, containing 35 000 people, as well as several breweries and dye houses, with pure, filtered water at high pressure, day and night(150). Each unit provided eighty or ninety gallons a day at a cost of one penny per house, representing a 75% cut in the price of water(151). The supply of fresh water did much to improve the sanitary conditions and health in those lucky households connected, since water ceased to be a limited and, therefore, precious commodity, and could be used to improve hygiene standards. By 1849, nine tenths of the dwelling houses were supplied with eighteen to twenty gallons of water per head per day at a cost of 5s a week, which was usually paid by the landlord and recouped in the rent(152). There was normally one common tap in a court, although several of the larger ones had two or more(153). The unpolluted water prevented the propagation of certain diseases, and the permanent availability of the supply, at a standard charge, enabled the courts to be sluiced out and kept much cleaner than had been possible before.

The 1847-8 sanitary report(154) described houses erected over privies and open dustholes; others adjacent to pigstyes and yards where bones were boiled. Many inhabitants had to share a few privies, some of which had no door and were 'so noisome as scarcely to be approachable and so exposed as to offend all sense of decency'. Some were so badly built

that the drainage flowed into houses nearby. Conditions had seemingly improved little since 1832, when the sanitary committee(155) reported there had been defective drainage in the courts; unpaved streets, without sewers, which contained distended heaps of putrifying dung along their course; groups of forty to eighty houses which had not had the facility of a privy since they were built; and hundreds of privies unfit for human use. Many dwellings were built over cesspits, and the Board of Guardians, being aware of this, had refused to advance outdoor relief to the inhabitants because of the unhealthiness of their living conditions.

Conditions like these prompted the corporation to include building specifications in the Enclosure Act for Nottingham of 1845(156). These provisions give an indication of the type of dwelling which existed in the old part of Nottingham. Hence, in future, no house should adjoin another on more than two sides; no apartment below ground level or without a window was to be lived in, worked in or slept in; house walls were to be no less than nine inches thick, and the ground floor had to be twelve inches above street level and be hollow, or of a substance able to resist damp; drains and pipes for the removal of sewage were to be covered; no cesspools were to be built under or nearer than ten feet from the house; and any such cesspit was to be air and water tight; each house have a privy or dustpit, with a trapped soilpan and water for cleansing; all public privies were to be screened for privacy. No ashpits, piggeries, stables or other buildings likely to exude noxious stench could be built within fifteen feet of human habitation.



It was in the same appalling conditions that many of the labouring population worked. In 1845, the cotton and silk stocking makers and lace and bobbin net manufacturers employed 40 000 inhabitants of the town on a domestic system. Rooms in many houses were, therefore, converted into workshops(157). Lace dressing required a hot, humid atmosphere, so the rooms were heated by stoves. This encouraged vermin, which swarmed through the house, spreading disease(158). Women and children were employed in the textile trade, and the Commission found many instances of fifteen to twenty children working in a twelve foot square garret for fifteen hours a day(159). This practice was still going on in 1862, when the Children's Employment Commission discovered workshops which allowed, for example, only 67, 90 or 92 cubic feet per child(160).

A sanitary committee had been set up by the council because it was felt that 'all was not done which was needful to secure the health of such a densely crowded population'(161). This may have been true, but a justification in the following paragraph has a more authentic ring. The authorities, by acting swiftly, had pre-empted the intervention of the Central Board of Health. The sanitary committee was established to improve the health of the town, and was granted many powers for this purpose. However, it took care to avoid antagonising local property owners, and persuasion was used rather than legal force. Negotiation and consultation took place, sometimes over twelve to eighteen months, to reconcile the needs of the residents with the private interests of the property owners.

By employing such means, the sanitary committee sought to achieve improvement amicably and to preserve social harmony, even though such methods meant that removal of nuisances took more time than simply ordering demolitions and abatements would have done. The committee gradually gained public approval, and, by 1849, had made some headway with improvements. Eighty four dwellings, sited over cesspools and privies, had been removed, as had twenty four collections of 'dangerous manure'. Several sets of privies were relocated; and twenty one piggeries were removed. A selection of the worst courts in the town had been drained and paved and channels or sewers constructed. Despite all this activity, the Beck and Leen remained full of sewage; many courts were still without drainage in 1849; and putrifying refuse was still allowed to accumulate in the central channels, although it was permitted to remain for a much shorter period. In addition, houses were being built on the land sold by the enclosure commissioners, without access to sewerage and without regard to the specifications of the Enclosure Act, 1845(162). Back-to-back houses appeared and, in 1852, the General Board of Health issued a letter indicating that Nottingham's death rate was in excess of the 23 per thousand limit.(163)

Marriot Ogle Tarbotton was appointed surveyor in 1858, under the provisions of the Local Government Board Act(164), and produced his first report in 1860(165). The report dealt with the Rivers Leen and Tinker's Leen, and the Meadow's, and showed that Nottingham had failed to build on the earlier attempts to improve the city environment. The Leen consisted of 'foul and excrementitious water', which collected into stagnant reservoirs wherever it was held up by water wheels or

industrial diversions. Tarbotton recommended the Leen be recognised as the main sewer that it was, and treated accordingly, by removing all impediments to its flow and, thus, preventing the feculant build up of matter. Drain connections and public sewer junctions were bad, and resulted from 'everybody doing as he likes'. The Leen sewer was 'self damnatory'. The construction of low level housing on the frequently flooding land of the Meadows was 'short sighted' and wilfully inviting destruction; and the houses themselves were inadequately drained and without sewers. Water lay stagnant on the street surfaces, and cesspools overflowed into yards 'covering them with pestiferous matter'. Other cesspits oozed their contents into the boundary walls and then on into drinking wells. Walls were found 'saturated with faecal moisture', and Tarbotton asserted that, even though ventilation was important to health, 'the best ventilation .... will not compensate for deficient sewerage, stagnant water and damp houses'. Tarbotton urged the Local Board of Health to take steps to rectify the conditions before it was too late, and gave details of plans he had made, which have since disappeared.

Although Nottingham had made early attempts to redress the appalling conditions in the town, those attempts had seemingly come to little by the middle of the century. Certainly, the provision of the water supply was of incalculable worth, but the prevailing conditions of the town meant that the death rate was still above that of the nation in general.

## CHAPTER FIVE

### CAUSAL FACTORS IN THE DECLINE OF MORTALITY: THE LOCAL EVIDENCE

The previous chapters have described the mortality trends of the three towns, and the sanitary conditions prevailing in the years before the death rates declined. This chapter will attempt to explore the extent of correlation between the causal factors in mortality decline identified by McKeown, and those influencing the mortality rates in the towns. It is unnecessary to discuss scarlet fever in this context, since the local statistical data substantiates McKeown's national findings, suggesting that a mutation of the haemolytic streptococci was the cause of its retreat as a leading killer(1). Smallpox, too, can be discounted here, since it was fading out early in the century, and contributed little to the declines. The advent of vaccination had quelled both incidence and fatality from this disease, and it is not necessary to seek further for a cause of decline.(2)

This chapter, therefore, will concentrate on the decline of mortality in Bath, Birmingham and Nottingham, with respect to typhoid; cholera, dysentery and diarrhoea; typhus; and tuberculosis. It is not intended to give an exhaustive chronological narrative of the reform programmes of each town, but merely to include those measures which, when introduced, may have had an effect on cause specific mortality. For this reason, it is more analytically cohesive to examine individual aspects of reform, referring to each town, rather than to give a critique of the separate local experiences. The examination will

concentrate on, first, water supply and sewage disposal; and then on overcrowding. The former is identified as the principal factor in the decline of typhoid, cholera, diarrhoea and dysentery; the latter might have influenced tuberculosis and typhus rates. It will not be possible to debate the contribution of the rise in living standards, particularly affecting diet, since the evidence is only partially available at a local level for this purpose.

#### **Water/food borne diseases and sanitary reform**

At mid-century, ownership of the water supply of the city of Bath was shared by the Corporation and twenty other companies(3). In 1851, the corporation had obtained an Act, permitting the building of a reservoir at Batheaston, but this had not eliminated the difficulties experienced with supplying the higher areas of the town(4). Street watering was done with water from the Avon, which was polluted and unhealthy(5). In 1870, an Act was passed enabling the corporation to extend the supply. New reservoirs were built to the North of the city and an 18in main pipe was constructed leading into the town(6). By the 1880s, the mains had been extended further, supplying the suburbs, and ample water was available for the increasing number of water closets in use(7). In 1864, plans had been accepted for the reform of the sewerage system(8). Streets were to be pitched with non-absorbant materials, sewers were to be laid down and a deodorising plant was to be established(9). Even so, in 1905, sewage was still discharging into the Avon.(10)

The Medical Officer of Health reported in 1880 that the sanitary efforts which had had the most beneficial effect had been the provision of pure water in place of the old polluted supply from contaminated wells(11). He expressed the hope that all wells would be closed, since no water therein could remain pure, given the nature of the surface drainage(12). The Medical Officer in 1900 was proud of the exceptionally good water supply of Bath, especially with regard to its abundance and admirable purity.(13)

The mortality of Bath had begun to drop from the 1870s, typhoid being reduced from 1880-90 from 14 cases to none(14). There was no cholera reported after 1870, and, although the diarrhoeal figures remained steady, it must be remembered that it was a principally infantile ailment, in which poor nutritional practices were implicated(15). There does, therefore, seem to be a correlation between the cleaning of the water supply and the decline from the water/food-borne zymotics in Bath.

Birmingham mortality declined abruptly after 1875, with the death rate from the typhoid group falling from 1871-80 to 1880-90(16). This, too, can be closely associated with reforms in the sanitation of the town. Thus, in 1876, the corporation of the town, under the leadership of Joseph Chamberlain, purchased the waterworks company, and the town began to benefit from cheaper, more widely available, piped water from the 1880s(17). Within eight years of the purchase, over 3 000 wells, supplying 60 000 users had been condemned as dangerously contaminated(18); and this elimination of polluted provision was as important a factor as the improvement of the clean supply in retarding

mortality. In addition, the sewage works of Saltley were extended and modernised from 1877(19), and a sewage farm and treatment works had been established as an alternative to using the River Tame(20).

Typhoid fever mortality dropped significantly in the first decade of the reforms, but then achieved a plateau until the end of the century(21). This can be explained by the concentration of the deaths in specific locations, supplied by private wells(22). The practice of using wells did gradually decline with the introduction of piped water(23), but the recourse to such sources can explain the seeming anomaly between a town well supplied with clean water, and a steady typhoid death rate. The earlier fall would be accounted for by the elimination of many contaminated outlets. The diarrhoeal death rate did not decline, but this can be explained in terms of infant mortality, especially as it falls in the 1900-10 period(24), precisely the time when the infant mortality rates began their precipitous decline(25). The sewage and water improvements in Birmingham, therefore, do coincide with the declining mortality from the water borne infections.

The water supply of Nottingham had been upgraded in the 1830s, with the construction of the Trent Waterworks(26). However, in 1879, the corporation purchased this company, and built new reservoirs and works on Mapperley Plains and at Papplewick(27). In addition, a sewage farm opened in 1880, before which the Trent had been heavily polluted with sewage(28). The frequent flooding of the lower town by the Trent(29) was, therefore, doubly harmful, because of the feculant nature of the water. In 1885, the Medical Officer of Health reported that Nottingham

[now] had an abundant supply of pure water, derived from deep wells. The average daily consumption was 18.8 gallons a head, and scarcely any private wells then existed for drinking purposes(30). In 1900, however, typhoid was prevailing in the Meadows, Leenside, and Lower Sneinton districts described by the Medical Officer of Health as the usual haunts of such fever(31). He was unable to ascribe it to any contamination of food or milk, nor to the water supply, but instead found it to be caused by the sewerage system which continued there, which, being a conservancy system, greatly increased the risks of infection. (32)

The evidence of Nottingham is more difficult to interpret given the early pioneering work of Hawkesley. However, the purchase, and subsequent extension, of the waterworks by the corporation and the reforms of the sewerage system, and the consecutive decline in mortality seem suggestive. The evidence of typhoid cases being locally discrete also substantiates the connection, since it was greatest in those areas with faulty sewage systems and poor drainage. Thus, the Nottingham evidence does accord with McKeown's theory, and the mortality decline of the waterborne infectives can be correlated with reforms in sanitation systems.

### **Typhus and tuberculosis**

The lack of data on the rise of living standards and improvements in nutrition at a local level mean that it is impossible to assess the impact of these factors on the decline of typhus and tuberculosis. However, although McKeown correlated nutrition with tuberculosis

decline, he also suggested that overcrowding is a potential influence on the disease(33). Typhus, too, he connected with diet, although he also assigned a part of it's retreat to improvements in cleanliness and the water supply(34). It is worthwhile to assess the degree of influence which crowding had on these diseases, since exposure and proximity play such a significant part in their propagation(35). The main problem is that tuberculosis is already in decline at the start of registration(36), so that it is impossible to locate the point of initial downturn. Nevertheless, if crowding were a primary factor in tuberculosis mortality, any dramatic change in the density of the population would be expected to elicit a response in the statistics.

Bath, under a Health Act of 1875, began to combat the malconstruction of housing by laying down specifications regarding road width, sanitation and wall thickness(37). Large areas of the city were laid out in new respectable terraces(38). Considerable changes were also made in the core of the town, where the corporation managed to exercise planning control(39). In 1890, a project had been approved for the construction of a new street from Milk street to Westgate Buildings(40). The Medical Officer of Health had high hopes of the scheme, since it would sweep away the slum housing of those streets.(41) Overcrowding existed in the slum quarters(42). However, in 1900, the Medical Officer reported that Bath was not an overcrowded city, and that undesirable buildings were continually destroyed and new improved ones provided(43). Under the Housing of the Working Class act, the first issue was the improvements of the Dolmeads(44). This did not begin until 1898, and the Holloway

district, Circus slums and the notorious district on the north of the river all remained intact until the twentieth century.(45)

There seems to be little scope for correlation between tuberculosis and typhus decline, and housing reform, since there is no evidence of significant change in the housing situation until the late 1890s and early 1900s. However, sanitary reform cannot be satisfactorily implicated in the decline of typhus either, since the disease had disappeared by the last quarter of the century, before the advent of the new systems(46). The inspection of lodging houses which began in the 1880s(47), is also too late to be a factor in the disappearance of typhus. Having, thus, eliminated the other possible factors, the question of living standards is left. The economic fortunes of Bath underwent a revival in the late Victorian era(48), and phthisis mortality declined more swiftly in the last decade than before. However, Bath had been prosperous in the eighteenth century, and had suffered a downturn in the nineteenth(49), but the tuberculosis mortality evidently fell during the century. Therefore, it is impossible to tie tuberculosis to economic forces and the explanation for the decline in the mortality from phthisis and - to a degree - typhus in Bath, must remain unproven.

The same pattern emerges in Birmingham too, where the improvements in housing began too late to be accorded responsibility for the decline of tuberculosis and typhus. Chamberlain's improvement act of 1876 displaced many of the working classes(50) and the worst courts were removed by the commercialisation of the city centre(51). Of the ninety

three acres regarded as slum areas fit for redevelopment by the corporation, forty three were acquired, thus eliminating much sub-standard housing(52). By 1884 there was, according to the Medical Officer of Health, no overcrowding(53). However, the decline of phthisis mortality did not speed up in response to these changes(54). Typhus had been of minor importance from at least the 1870s(55), and cannot, therefore, be tied to any of the reforms mentioned.

Birmingham's overall mortality did respond swiftly and dramatically to the improvements initiated by Chamberlain's corporation(56). However, it is not possible, in this instance, to isolate the degree of influence each measure had on each disease.

Nottingham housing showed no improvement up until the 1870s, when the Nottingham Improvement Act enabled the erection of dwellings by the corporation(57). A year later, the Artisans and Labourers Dwellings Act facilitated the destruction of three principal slums, one of which contained about 1 000 houses(58). By 1885, there were no cellar dwellings and overcrowding was uncommon(59), although the houses in the old quarters were densely constructed in close, narrow, ill-ventilated alleys(60). Typhus had disappeared before the reforms, and tuberculosis mortality showed no extraordinary reaction to the spur of housing improvement(61). There was a economic boom in the town in 1871-5, which increased overcrowding for a while(62) - again, the statistics for tuberculosis do not reflect this. The trend is steady, with a slight upswing in the last decade, when the reforms were well established.

The local evidence, then, shows no direct correlation between housing and tuberculosis, and indeed, the case of Glasgow bears this out. In 1917, the Royal commission on Housing found that Glasgow had housing of unbelievable density, with over four to a room in 11% of the houses, and more than three to a room in 30%(63). Nevertheless, tuberculosis had fallen in the city, and typhus had left it(64). Neil Munro MacFarlane does find evidence to connect tuberculosis and crowding(65). He describes the tuberculosis death rate as stagnating between 1850 and 1870, when overcrowding increased from 202 per 100 rooms to 210; the proportions inhabiting a single room rising from 34% to 41%. The mortality then fell 1870-90, as overcrowding decreased to 187 per 100 rooms, with only 25% living in a single apartment(66). MacFarlane does suggest that the case is not wholly proven, since the extreme overcrowding of Glasgow did not translate to unusually high returns of tuberculosis mortality(67). McKeown's theory is that overcrowding did not decrease significantly enough to affect exposure(68), and this does seem to be true in the case of Glasgow. The levels of overcrowding are such that exposure must have remained at a premium.

All the conclusions reached are tentative, since the evidence needed to substantiate McKeown's theory is not available. The living standard and nutrition question remains unanswered, although it is true that tuberculosis is a disease of deprivation, and that bad housing and poor nutrition would have increased the susceptibility of the population(69).

McKeown's findings have been proven to be statistically compatible with the local data, but no comment can be made with regard to the causal

factors, and particularly their numerical weights, because of the paucity of the necessary evidence. The water supply and housing improvements do seem to have had an overall effect on the zymotic mortality, but the extent of that effect, numerically and in terms of specific diseases, is not discernible. Therefore, although mortality did decline in Bath, Birmingham and Nottingham in the nineteenth century, the causes of this decline are not proven by local evidence. The juxtaposition of specific reforms with the mortality rates of typhus and tuberculosis, fail to correlate with the declines, and the evidence of prosperity and diet is not available in sufficient quantity to merit any reasonable deductions being made.

FOOTNOTES: METHODOLOGY

- 1 Weekly Reports of the Medical Officer of Health for Bath; 1866-1875
- 2 Annual Reports of the Medical Officer of Health for Bath; 1880; 1890 and 1900.
- 3 Postgate, J. *The Sanitary Aspect of Birmingham*. (Birmingham 1852)
- 4 Heslop, T P. 'The Medical Aspects of Birmingham' in Timmins, S (ed.) *The Resources, Products and Industrial History of Birmingham and the Midland Hardware District*. (Birmingham 1866)
- 5 Quarterly Reports of the Medical Officer of Health for Birmingham; 1880 and 1890; Annual Report of the Medical Officer of Health for Birmingham; 1900
- 6 For example, Rawlinson, Sir Robert. *Report to the General Board of Health on the Sanitary Condition of Birmingham, 1849*.
- 7 Woods, R. and Hinde, P R A. 'Mortality in Victorian England: Models and Patterns'. *Journal of Interdisciplinary History* XVIII:I. (1987). p29
- 8 Civil Registration Act; 1837, 6&7 William IV. Cap 86
- 9 McKeown, T. and Record, R G. 'Reasons for the Decline of Mortality in England and Wales during the Nineteenth Century', *Population Studies* 16. (1962-3) p98
- 10 *Ibid*, p99
- 11 Woods, R. and Woodward, J. (eds). *Urban Disease and Mortality in Nineteenth Century England*. (London 1984) p21; McKeown, T. *The Modern Rise of Population*. (London 1976) pp7-8
- 12 McKeown and Record. 'Reasons for the Decline of Mortality'. p99; McKeown. *The Modern Rise of Population*. p7.
- 13 McKeown, T. *The Modern Rise of Population*. p7
- 14 *Ibid*, p6
- 15 McKeown, T., Record, R G. and Turner, R D. 'An Interpretation of the Decline of Mortality in England and Wales in the Twentieth Century' *Population Studies* 29. (1975) p391
- 16 Woods and Woodward. *Urban Disease and Mortality*. p21
- 17 McKeown and Record. 'Reasons for the Decline of Mortality'. p95
- 18 *Ibid*, pp98, 102

- 19 Cronje, G. 'Tuberculosis and Mortality Decline in England and Wales: 1851-1910' in *Urban Disease and Mortality*. (London 1984) p86
- 20 Smith, F B. *The Retreat of Tuberculosis: 1850-1950*. (Beckenham 1988) p2; McKeown and Record. 'Reasons for the Decline of Mortality'. p102
- 21 Smith, F B. *The People's Health: 1830-1910*. (London 1979). p149
- 22 Alexander Dumas claimed in the 1820's that 'it [was] the fashion to suffer from the lungs". McGrew, R E. *Encyclopedia of Medical History*. (London 1985) p355
- 23 Smith, *The People's Health*, p288; Cronje, 'Tuberculosis and Mortality Decline'. pp80, 83
- 24 McKeown and Record. 'Reasons for the Decline of Mortality'. p103
- 25 Woods and Hinde. 'Mortality in Victorian England'. p32
- 26 McKeown and Record. 'Reasons for the Decline of Mortality'. p95
- 27 Wohl, A. *Endangered Lives*. (London 1984). pp16, 35; Smith, *The People's Health*, p87; Cronje, 'Tuberculosis and Mortality Decline', p86
- 28 Wohl. *Endangered Lives*. pp35, 24
- 29 Smith. *The People's Health*. p149
- 30 *Ibid*
- 31 Gale, A H. *Epidemic Diseases* (Harmondsworth 1959) p72
- 32 Luckin, W. 'Evaluating the Sanitary Revolution: Typhus and Typhoid in London 1851-1900' in *Urban Disease and Mortality*. p104
- 33 McKeown and Record. 'Reasons for the Decline of Mortality'. p101. Cholera was separated from diarrhoea and dysentery in the 1870s, but the latter two remained together throughout the century.
- 34 Gale. *Epidemic Diseases*. p83. They were not amalgamated until 1910
- 35 Smith. *The Retreat of Tuberculosis*. p2
- 36 Smith. *The People's Health*. p87., mentions the confusion of atrophy and debility with diarrhoeas. See below, under infant mortality.; Annual Report of the Medical Officer of Health for Nottingham, 1890., mentions that whooping cough was confused with bronchitis
- 37 McKeown and Record. 'Reasons for the Decline of Mortality'. p101
- 38 Smith. *The People's Health*. p67

39 *Ibid*

40 *Ibid*, p68

41 Woods and Woodward, *Urban Disease and Mortality*, p86

42 *Ibid*

43 Woods and Hinde, 'Mortality in Victorian England', p32

FOOTNOTES: CHAPTER ONE

- 1 McKeown, T. *The Modern Rise of Population*. (London 1976) p15
- 2 Mitchell, B R. and Deane, P. *Abstract of British Historical Statistics*. (Cambridge 1962) pp29-30
- 3 McKeown. *The Modern Rise of Population*. p15
- 4 Woods, R. and Woodward, J. (eds). *Urban Disease and Mortality in Nineteenth Century England*. (London 1984) p20; Shelston, D and A. *The Industrial City: 1830-70*. (London 1990) p16; Wohl, A. *Endangered Lives*. (London 1984) p80
- 5 Shelston. *The Industrial City*. p21
- 6 Woods and Woodward. *Urban Disease and Mortality*. p47
- 7 Morris, R J. and Rodger, R. *The Victorian City: 1820-1914*. (Harlow 1993) p4
- 8 Woods and Woodward. *Urban Disease and Mortality*. p66
- 9 Mitchell and Deane. *Abstract*. pp8-10
- 10 Morris and Rodger. *The Victorian City*. p3
- 11 Wohl. *Endangered Lives*. p3
- 12 *Ibid*, pp3-5
- 13 Mitchell and Deane. *Abstract*. pp36-7
- 14 Harrison, J F C. *Early Victorian Britain: 1832-51*. (London 1979) p16; Calculations from Mitchell and Deane. *Abstract*. pp36-7
- 15 Chadwick, E. *Report on the Sanitary Condition of the Labouring Population of Great Britain*. (1842; republished with an introduction by M Flinn (ed.) Edinburgh 1965) p13
- 16 *Ibid*, p14
- 17 *Ibid*
- 18 *Ibid*
- 19 Smith dates the overall mortality decline from the late 1870s. Smith, F B. *The People's Health: 1830-1910*. (London 1979) p196; Woodward from the early 1870s. Woods and Woodward. *Urban Disease and Mortality*. p66; whilst McKeown and Record put it in the eighth decade. McKeown, T. and Record, R G. 'Reasons for the Decline of Mortality in England and Wales during the Nineteenth Century' *Population Studies* 16. (1962-3) p99

- 20 Wohl, *Endangered Lives*, p344
- 21 Mitchell and Deane, *Abstract*, p36. It dropped from 3.4 per 1000, 1875-1885, and by a further 1.0 per 1 000, 1885-1900
- 22 Woods and Woodward, *Urban Disease and Mortality*, p66
- 23 Information in this section is derived from Woods, R. 'Mortality Patterns in the Nineteenth Century' in Woods and Woodward, *Urban Disease and Mortality*, pp37-64
- 24 Information in this section is derived from Woods, 'Mortality Patterns in the Nineteenth Century', pp37-64 & from Woods, R. and Hipde, P R A. 'Mortality in Victorian England: Models and Patterns', *Journal of Interdisciplinary History* XVIII:1 (1987) pp27-54
- 25 McKeown and Record, 'Reasons for the Decline of Mortality', p100
- 26 Woods, 'Mortality Patterns in the Nineteenth Century', p40
- 27 Smith, *The People's Health*, p65
- 28 *Ibid*
- 29 Mitchell and Deane, *Abstract*, pp36-7
- 30 Smith, *The People's Health*, p65
- 31 *Ibid*, p66
- 32 Wohl, *Endangered Lives*, p11
- 33 Civil Registration Act, 37 & 38 Victoria, Cap 88
- 34 Wohl, *Endangered Lives*, p11
- 35 *Ibid*
- 36 Cartwright, F F. *A Social History of Medicine*, (London 1977) p111
- 37 Wohl, *Endangered Lives*, p11
- 38 Woods and Hinde, 'Mortality in Victorian England', pp40-1
- 39 Wohl, *Endangered Lives*, p11
- 40 *Ibid*, p12
- 41 Woods and Hinde, 'Mortality in Victorian England', p44
- 42 Chadwick, *Report on the Sanitary Condition of the Labouring Population*, Introduction by Flinn, pp15-16

- 43 Woods and Hinde, 'Mortality in Victorian England', p44
- 44 Smith, *The People's Health*, p65
- 45 *Ibid*, p85
- 46 Woods and Hinde, 'Mortality in Victorian England', p44
- 47 Smith, *The People's Health*, p85
- 48 *Ibid*, p90
- 49 *Ibid*
- 50 *Ibid*, p87
- 51 *Ibid*
- 52 *Ibid*, p85
- 53 *Ibid*
- 54 *Ibid*, p88
- 55 *Ibid*, p87; Cronje, G. 'Tuberculosis and Mortality Decline in England and Wales; 1851-1910' in Woods and Woodward, *Urban Disease and Mortality*, p81
- 56 Smith, *The People's Health*, p88
- 57 *Ibid*, p89; Wohl, *Endangered Lives*, p20
- 58 Smith, *The People's Health*, p85; Wohl, *Endangered Lives*, p25
- 59 Wohl, *Endangered Lives*, p34
- 60 Smith, *The People's Health*, p97
- 61 *The Times*, 12 April 1844.
- 62 Smith, *The People's Health*, p98
- 63 *Ibid*; Wohl, *Endangered Lives*, p35
- 64 Wohl, *Endangered Lives*, p35. Dr Greenhow investigating for the Privy Council found that children "kept in a state of continued narcotism will be thereby disinclined for food and be but imperfectly nourished".
- 65 Wohl, *Endangered Lives*, p20
- 66 *Ibid*, p36
- 67 *Ibid*, p35

- 68 Smith, *The People's Health*, p104
- 69 *Ibid*, p 105
- 70 Creighton, C. *A History of Epidemics in Britain; 1666-1893*, Volume II, (Cambridge 1894) p765
- 71 Smith, *The People's Health*, p13
- 72 Wohl, *Endangered Lives*, p16
- 73 *Ibid*
- 74 *Ibid*
- 75 *Ibid*, p17
- 76 Woods and Woodward, *Urban Disease and Mortality*, p47
- 77 Leeson, J. and Gray, J. *Women and Medicine*, (London 1978) pp138, 158; Wohl, *Endangered Lives*, pp12, 13
- 78 Cronje, 'Tuberculosis and Mortality Decline', p86
- 79 Smith, F B. *The Retreat of Tuberculosis; 1850-1950*, (Beckenham 1988) p8
- 80 Cartwright, *A Social History of Medicine*, p123. Between 1889 and 1890, tuberculosis surgery, (surgery carried out on account of tubercular disease of glands, bones and joints) accounted for 26% of all operations. By 1901, the proportion was still 20.5%
- 81 Smith, *The Retreat of Tuberculosis*, p3
- 82 *Ibid*
- 83 *Ibid*, p8
- 84 Smith, *The People's Health*, p289. Dr C R Drysdale estimated that the poor had four times the tuberculosis morbidity of the rich.;  
Smith, *The Retreat of Tuberculosis*, p8
- 85 Cronje, 'Tuberculosis and Mortality Decline', p86
- 86 *Ibid*, p87
- 87 Wohl, *Endangered Lives*, p39
- 88 *Ibid*, p38. From Lyon Playfair, 1874.

FOOTNOTES: CHAPTER TWO

- 1 This is explained in McKeown, T. and Record, R G. 'Reasons for the Decline of Mortality in England and Wales during the Nineteenth Century', *Population Studies* 16, (1962-3); and in McKeown, T. *The Modern Rise of Population*, (London 1976).
- 2 Woods, R. and Hinde, P R A. 'Mortality in Victorian England: Models and Patterns', *Journal of Interdisciplinary History* XVIII:I (1987) p27
- 3 McKeown and Record. 'Reasons for the Decline of Mortality'. p96
- 4 This is taken from McKeown and Record. 'Reasons for the Decline of Mortality'. p106
- 5 McKeown. *The Modern Rise of Population*. p66
- 6 This is taken from McKeown and Record. 'Reasons for the Decline of Mortality'. p104
- 7 McKeown and Record. 'Reasons for the Decline of Mortality'. p103
- 8 Woodward, J. 'Medicine and the City: The Nineteenth Century Experience' in Woods, R. and Woodward, J. (eds). *Urban Disease and Mortality*. (London 1984) p67
- 9 McKeown and Record. 'Reasons for the Decline of Mortality'. pp103-4
- 10 *Ibid*, p103
- 11 This is taken from McKeown and Record. 'Reasons for the Decline of Mortality'. p106
- 12 *Ibid*, p97
- 13 *Ibid*, pp116-7
- 14 *Ibid*, pp115-6
- 15 *Ibid*, p97
- 16 Only 1.6%. McKeown. *The Modern Rise of Population*. p99
- 17 McKeown and Record. 'Reasons for the Decline of Mortality'. p97
- 18 Smith, J R. *The Speckled Monster*. (Chelmsford 1987) p118;  
Smith, F B. *The People's Health; 1830-1910*. (London 1979) p156
- 19 Smith. *The People's Health*. p161. Vaccination was not complete, even in 1885, when the dominant trend in the mortality decline became clear. Even in 1898, 33% of London infants were unvaccinated. Notification and isolation from 1885, may have played a part in the decline; and improvements in living standards, too,

may have helped victims withstand the disease. A decrease in the virulence of the smallpox virus would also have had an influence on its mortality.

Wohl states that the percentage take-up of vaccination fell between 1873 and 1897 from 85% to 70%. Wohl, A. *Endangered Lives*. (London 1984) p133. Gale, however, believes that between 1870 and 1900, infant vaccination was more widespread than ever before and associates this with the fall off in the smallpox death rate. Gale, A H. *Epidemic Diseases*. (Harmondsworth 1959) p64. McKeown concurs with Gale and shows the decline from smallpox in graph form, especially from 1870, when enforcement was introduced. McKeown. *The Modern Rise of Population*. p99. There is, then, some debate about the degree of contribution of vaccination to the decline of mortality from smallpox. However, it did play some part in the fall.

- 20 Woods and Woodward. *Urban Disease and Mortality*. p31
- 21 These are taken from Woods and Woodward. *Urban Disease and Mortality*. pp29-30.
- 22 McKeown and Record. 'Reasons for the Decline of Mortality'. pp119-120
- 23 Smith, F B. *The Retreat of Tuberculosis; 1850-1950*. (Beckenham 1988) p3
- 24 Cronje, G. 'Tuberculosis and Mortality Decline in England and Wales; 1851-1910' in Woods and Woodward. *Urban Disease and Mortality*. pp80-1
- 25 Smith. *The People's Health*. p289
- 26 Cronje. 'Tuberculosis and Mortality Decline'. p81. Haemoptysis or spitting of blood occurs when cavities form in the lungs.
- 27 *Ibid*, pp81, 89; Smith. *The Retreat of Tuberculosis*. p2
- 28 Cronje. 'Tuberculosis and Mortality Decline'. p82
- 29 *Ibid*
- 30 *Ibid*
- 31 *Ibid*, p80
- 32 *Ibid*, p83; Wohl. *Endangered Lives*. p130; Information contained in a letter from Dr John Porter, of the London School of Hygiene and Tropical Medicine, May 12, 1993, in which he confirms the link between factors debasing the immune system (for example, poor nutrition), and development of tuberculosis; and also confirms that tuberculosis is a disease of poverty and deprivation.

- 33 Wohl, *Endangered Lives*, p131
- 34 Magnusson, M. *Chambers Biographical Dictionary*, (Edinburgh 1990) p835
- 35 Cronje, 'Tuberculosis and Mortality Decline', p80
- 36 Smith, *The Retreat of Tuberculosis*, pp37-9. It took a long time before the infectious nature of tuberculosis was accepted by the medical establishment in Britain. Smith (*The People's Health*, p290) puts it at a decade.
- 37 Cronje, 'Tuberculosis and Mortality Decline', p83
- 38 *Ibid*
- 39 *Ibid*
- 40 *Ibid*
- 41 Smith, *The Retreat of Tuberculosis*, p7
- 42 McKeown and Record, 'Reasons for the Decline of Mortality', p108; Smith, *The Retreat of Tuberculosis*. Hence the title.  
Porter, J H. 'Tuberculosis in Developing Countries', *Communicable Diseases*, Volume I, Review 12, (1991) p136
- 43 Wohl, *Endangered Lives*, p130
- 44 *Ibid*, p118
- 45 Morris, R J. *Cholera 1832*, (New York 1976) p12
- 46 *Ibid*
- 47 Wohl, *Endangered Lives*, pp118-125
- 48 Wohl, *Endangered Lives*, p118; Luckin, W. 'Death and Survival in the City', *Urban History Yearbook*, (1980) p59
- 49 Smith, *The People's Health*, p90
- 50 Luckin, W. 'Evaluating the Sanitary Revolution: Typhus and Typhoid in London 1851-1900' in Woods and Woodward, *Urban Disease and Mortality*, p104
- 51 Smith, *The People's Health*, p244; Luckin, 'Evaluating the Sanitary Revolution', p104
- 52 McGrew, R. *Encyclopedia of Medical History*, (London 1985) p397
- 53 Luckin, 'Evaluating the Sanitary Revolution', pp102-3, 104
- 54 *Ibid*, p104

- 55 *Ibid*, p107; Wohl. *Endangered Lives*. p128
- 56 Luckin. 'Evaluating the Sanitary Revolution'. p104
- 57 *Ibid*, p102; Smith. *The People's Health*. p238
- 58 Luckin. 'Evaluating the Sanitary Revolution'. p103
- 59 *Ibid*, p104
- 60 Chadwick, E. *Report on the Sanitary Condition of the Labouring Population of Great Britain*. (1842; republished with an introduction by M Flinn. Edinburgh 1965) p78
- 61 Luckin. 'Evaluating the Sanitary Revolution'. p105
- 62 *Ibid*, p113. Luckin estimates it at not less than a 60% fall in the 1870s
- 63 *Ibid*, p115
- 64 Smith. *The People's Health*. p239
- 65 *Ibid*, p244
- 66 *Ibid*
- 67 *Ibid*, p245
- 68 Creighton, C. *A History of Epidemics in Great Britain; 1666-1893*. Volume II. (Cambridge 1894) p212
- 69 Gale. *Epidemic Diseases*. p55
- 70 *Ibid*, p64
- 71 *Ibid*
- 72 Smith. *The People's Health*. p156
- 73 Creighton. *A History of Epidemics*. p615
- 74 *Ibid*, p627
- 75 Smith. *The Speckled Monster*. p118
- 76 Smith. *The People's Health*. p145
- 77 Gale. *Epidemic Diseases*. p102
- 78 Smith. *The People's Health*. p143
- 79 *Ibid*

- 80 *Ibid*
- 81 *Ibid*, p144-5
- 82 *Ibid*, p145
- 83 *Ibid*, p147
- 84 *Ibid*, p106
- 85 *Ibid*, p149
- 86 These issues have been discussed in the methodology.
- 87 Smith, *The People's Health*, p149
- 88 *Ibid*
- 89 *Ibid*
- 90 *Ibid*
- 91 Creighton, *A History of Epidemics*, p742
- 92 Smith, *The People's Health*, p151
- 93 *Ibid*
- 94 *Ibid*
- 95 Gale, *Epidemic Diseases*, p95
- 96 Smith, *The People's Health*, p106
- 97 *Ibid*, p105
- 98 *Ibid*, p107
- 99 McKeown and Record, 'Reasons for the Decline of Mortality', p104
- 100 Smith, *The People's Health*, p107
- 101 *Ibid*, p136
- 102 McKeown and Record, 'Reasons for the Decline of Mortality', p97
- 103 Smith, *The People's Health*, p136
- 104 *Ibid*
- 105 *Ibid*, p137
- 106 *Ibid*

- 107 Creighton. *A History of Epidemics*. p671
- 108 McKeown and Record. 'Reasons for the Decline of Mortality'. p97
- 109 *Ibid*
- 110 *Ibid*, p117
- 111 Woods and Hinde. 'Mortality in Victorian England'. p52.
- 112 Woodward. 'Medicine and the City'. p67
- 113 *Ibid*, p71; 77. Woodward himself recognises the uncertainty and speculative nature of the conclusions about hospital provision and care
- 114 Woods and Woodward. *Urban Disease and Mortality*. p34
- 115 Woodward. 'Medicine and the City'. p67
- 116 *Ibid*
- 117 *Ibid*
- 118 *Ibid*
- 119 *Ibid*
- 120 McKeown and Record. 'Reasons for the Decline of Mortality'. p120. In the following discussion, sanitary reform will be taken to mean those reforms related to drainage, sewerage, sewage disposal, water supply, notification of diseases, removal of nuisances, & etc. Environmental reform will be taken to mean improvements in the standard of living, including diet and crowding.
- 121 McKeown and Record. 'Reasons for the Decline of Mortality'. pp116, 118
- 122 Luckin. 'Evaluating the Sanitary Revolution'. p116
- 123 McKeown and Record. 'Reasons for the Decline of Mortality'. p101
- 124 *Ibid*, p116
- 125 Hardy, A. 'Urban Famine or Urban Crisis: Typhus in the Victorian City' in *The Victorian City: 1820-1914*. (Harlow 1993) pp209, 236-40
- 126 McKeown and Record. 'Reasons for the Decline of Mortality'. p109
- 127 *Ibid*, p115
- 128 *Ibid*, p110

- 129 *Ibid*
- 130 *Ibid*
- 131 *Ibid*, p112
- 132 *Ibid*
- 133 *Ibid*, p113
- 134 *Ibid*, p112
- 135 *Ibid*
- 136 *Ibid*
- 137 *Ibid*
- 138 *Ibid*, p113
- 139 *Ibid*, p109
- 140 *Ibid*, pp112-3
- 141 *Ibid*, p113
- 142 *Ibid*, pp113-4
- 143 *Ibid*, p114
- 144 *Ibid*; Smith. *The People's Health*, p290
- 145 McKeown and Record. 'Reasons for the Decline of Mortality', p116
- 146 *Ibid*, p114
- 147 *Ibid*
- 148 *Ibid*
- 149 *Ibid*
- 150 *Ibid*
- 151 *Ibid*
- 152 *Ibid*
- 153 *Ibid*, p115
- 154 Cronje. 'Tuberculosis and Mortality Decline', p87. 'Between 1851-60 and 1900-10, the extra pulmonary tuberculosis death rate per 1 000 dropped by 31.9% for men and 24.8% for women

- 155 McKeown and Record. 'Reasons for the Decline of Mortality'. pp113 & 115
- 156 Ibid, p114
- 157 Ibid, p115
- 158 Ibid
- 159 Ibid
- 160 Ibid
- 161 Ibid
- 162 Luckin. 'Death and Survival in the City'. p57.  
Woods and Woodward estimate the rise in the standard of living from the late nineteenth century, but possibly from 1840. Woods and Woodward. *Urban Disease and Mortality*. p31; Cronje puts the rise in the standard of living after 1870. Cronje. 'Tuberculosis and Mortality Decline'. p84;
- 163 McKeown and Record. 'Reasons for the Decline of Mortality'. p94
- 164 Ibid, p116
- 165 Ibid, p120
- 166 Ibid, pp119-20
- 167 Ibid, pp100-1, 103, 104, 118, 119-120
- 168 Ibid, p115
- 169 Smith. *The Retreat of Tuberculosis*. p243
- 170 Ibid
- 171 Information in the following paragraph comes from Woods and Hinde. 'Mortality in Victorian England'. pp28, 52-4
- 172 Information in the following paragraph comes from Woodward. 'Medicine and the City'. p68
- 173 Woodward is citing D J Oddy. 'The Health of the People' in Banner, T C. and Drake, M. (eds.), *Population and Society in Britain; 1850-1980*. (London 1982) pp121-39
- 174 Wohl describes the list of adulterated foods as increasing through the century, and suggests that the administration of the Adulteration of Food and Drugs legislation was haphazard and not wholly effective. Wohl. *Endangered Lives*. pp52-4
- 175 Information in the next two paragraphs is derived from Cronje.

- 'Tuberculosis and Mortality Decline'. pp97-8, 101
- 176 Information in the next two paragraphs is derived from Wohl, *Endangered Lives*, pp50-1, 56, except where stated.
- 177 Burnett, J. *Plenty and Want; A Social History of Diet in England from 1815 to the present day*. (London 1966) p3
- 178 Wohl. *Endangered Lives*. pp52-4, 21-2, 56
- 179 Woods and Hinde. 'Mortality in Victorian England'. p52
- 180 Woodward. 'Medicine and the City'. pp65-78
- 181 Information in the following paragraph is derived from Woods and Hinde. 'Mortality in Victorian England'. pp52-4
- 182 Woods and Hinde. 'Mortality in Victorian England'. p53
- 183 Information in the following three paragraphs is derived from Woods, R. and Woodward, J. 'Mortality, Poverty and the Environment' in Woods and Woodward. *Urban Disease and Mortality*. pp31-3

FOOTNOTES: CHAPTER THREE

- 1 *Weekly Reports of the Medical Officer of Health for Bath; 1866-75*  
*Annual Reports of the Medical Officer of Health for Bath; 1880; 1890*  
and 1900. *Annual Reports of Medical Officer of Health for*  
*Nottingham* were available from 1874, and previous to that, there  
were sanitary committee reports from 1849. Birmingham appointed a  
Medical Officer of Health in 1873
- 2 Mitchell, B R, and Deane, P. *Abstract of British Historical*  
*Statistics*. (Cambridge 1962) pp24-5
- 3 *Weekly Reports of the Medical Officer of Health for Bath*. The  
reports used are those for the week ending on or around 25 April in  
each year; 1866-75
- 4 *Weekly Reports of the Medical Officer for Bath; 1866-75*. containing  
figures for the spring quarter; *Annual Reports of the Medical*  
*Officer of Health for Bath; 1880; 1890 and 1900; Mitchell and*  
*Deane. Abstract*. pp36-7
- 5 Creighton, C. *A History of Epidemics in Britain; 1666-1893*.  
Volume II. (Cambridge 1894) p615, 727
- 6 *Annual Report of the Medical Officer of Health for Bath; 1880*
- 7 *Ibid*, 1880, 1890 and 1900
- 8 *Ibid*
- 9 *Annual Report of the Medical Officer of Health for Bath; 1890*.  
pp12, 10
- 10 *Ibid*, p4
- 11 *Annual Report of the Medical Officer of Health for Bath; 1880*
- 12 *Annual Reports of the Medical Officer of Health for Bath; 1890 and*  
*1900*. The 1890 report states that the Notification of Diseases Act  
had come into force in Bath on March 1st, 1890.
- 13 *Weekly Reports of the Medical Officer of Health for Bath; 1866-75*
- 14 Creighton. *A History of Epidemics*. p604
- 15 *Annual Reports of the Medical Officer of Health for Bath; 1880; 1890*  
and 1900
- 16 *Annual Report of the Medical Officer of Health for Bath; 1900*
- 17 *Ibid*, pp21-2
- 18 *Ibid*, p22

- 19 Ibid, pp21-2
- 20 Sir Henry De La Beche. 'Report on the City of Bath and it's Sanitary Condition', contained in the Appendix to the *Second Report of the Royal Commission appointed to Inquire into the State of Large Towns and Populous Districts, 1845*. Volume XVIII. pp176-269.
- 21 Figures are from the *Annual Reports of the Medical Officer of Health for Bath; 1880; 1890 and 1900; and Mitchell and Deane. Abstract*. pp36-7
- 22 Wohl, A. *Endangered Lives*. (London 1984) p38
- 23 *Annual Reports of the Medical Officer of Health for Bath; 1880, 1890 and 1900*
- 24 The 1811-21 figure is from the estimates of the friendly societies, quoted in Chadwick, E. *Report on the Sanitary Condition of the Labouring Population of Great Britain*. (1842, republished, with an introduction by M Flinn, Edinburgh 1965) p13; The 1848 figure comes from Sir Robert Rawlinson, *Report to the General Board of Health on the Sanitary Condition of Birmingham 1849*. p19; The 1851-60 figure from Heslop, T P. 'The Medical Aspect of Birmingham' in Timmins, S. (ed.) *The Resources, Products and Industrial History of Birmingham and the Midland Hardware District*. (London 1866) p693; The 1871-5 figure from Briggs, A. *A History of Birmingham*. (London 1952) p77, citing J T Bunce. *History of the Corporation of Birmingham*. Volume II (1885) chapter iv; *The remaining figures from the Annual Reports of the Medical Officer of Health for Birmingham; 1880, 1890 and 1900.*
- 25 *Ibid; Mitchell and Deane. Abstract*. pp36-7
- 26 For example, Heslop. 'The Medical Aspects of Birmingham'
- 27 *Ibid*, pp695-6
- 28 *Ibid*, p689
- 29 *Ibid*, p691
- 30 *Ibid*, p689
- 31 Woods, R. 'Mortality and Sanitary Conditions in Late Nineteenth Century Birmingham' in Woods, R. and Woodward, J. (eds.) *Urban Disease and Mortality in the Nineteenth Century*. (London 1984) p180
- 32 *Annual Report of the Medical Officer of Health for Birmingham; 1880*
- 33 *Ibid*, 1890
- 34 *Ibid*, 1900

- 35 Smallpox claimed two lives in 1880; typhus, 3. Thereafter, neither disease appeared on the register of mortality. *Annual Reports of the Medical Officer of Health for Birmingham*; 1880; 1890 and 1900
- 36 *Annual Reports of the Medical Officer of Health for Birmingham*; 1880 and 1900
- 37 *Ibid*, 1880, 1890 and 1900
- 38 *Ibid*
- 39 Creighton. *A History of Epidemics*. p615
- 40 That is, 'trivial'. McKeown, T. and Record, R G. 'Reasons for the Decline of Mortality in England and Wales during the Nineteenth Century'. *Population Studies* 16. (1962-3) p104
- 41 Creighton. *A History of Epidemics*. p674-5; Smith, F B. *The People's Health; 1830-1910*. (London 1979) p107. However, whooping cough most often accompanied measles and cholera epidemics, and, in the 1871-80 period, cholera was of no importance, and measles was not unusually virulent.
- 42 *Report on the State of the Public Health of Birmingham by Committee of Physicians and Surgeons*, 1841
- 43 *Annual Report of the Medical Officer of Health for Birmingham*; 1880
- 44 *Annual Reports of the Medical Officer of Health for Birmingham*; 1880, 1890 and 1900
- 45 *Nottingham Corporation Sanitary Committee Reports 1855-70; Annual Reports of the Medical Officer of Health for Nottingham*; 1885, 1890 and 1900; Mitchell and Deane. *Abstract*. pp36-7
- 46 Wohl, A. *Endangered Lives*. (London 1984) p149-50
- 47 *Annual Report of the Medical Officer of Health for Nottingham*; 1885. p17
- 48 *Ibid*,
- 49 *Ibid*, 1890, p19 and 1900, p21
- 50 *Annual Reports of the Medical Officer of Health for Nottingham*; 1890, p15, and 1900, pp13, 15
- 51 *Annual Report of the Medical Officer of Health for Nottingham*; 1890, p15 and 1900, pp13, 27
- 52 *Nottingham Corporation Sanitary Committee Reports 1855-70; Annual Reports of the Medical Officer of Health for Nottingham*; 1885, 1890 and 1900

- 53 *Annual Report of the Medical Officer of Health for Nottingham; 1900,*  
p40
- 54 *Ibid, 1885, p24*
- 55 *Ibid, 1890, p21*
- 56 *Annual Reports of the Medical Officer of Health for Nottingham;*  
1890 and 1900
- 57 *Annual Reports of the Medical Officer of Health for Nottingham;*  
1885, p19, 1890, p19, and 1900, p22
- 58 *Annual Reports of the Medical Officer of Health for Nottingham;*  
1890 and 1900; Mitchell and Deane. *Abstract.* pp36-7

FOOTNOTES: CHAPTER FOUR

- 1 Neale, R S. *Bath 1680-1850; A Social History*. (London 1981) p47
- 2 *Ibid*, p265
- 3 Cunliffe, B. *The City of Bath*. (Gloucester 1986) p154
- 4 Neale. *Bath 1680-1850*. p266
- 5 *Ibid*. The railway was eventually built 1839-41. Cunliffe. *The City of Bath*. p154
- 6 Neale. , *Bath 1680-1850*. p266
- 7 *Ibid*, p268. Have used Neale's occupational index here, which is derived from the Census of 1831.
- 8 *Ibid*, pp275-6
- 9 *Ibid*, p276
- 10 *Ibid*, p277
- 11 Cunliffe. *The City of Bath*. p153
- 12 *Ibid*
- 13 *Ibid*, p154
- 14 *Ibid*
- 15 Neale. *Bath 1680-1850*. p271
- 16 *Ibid*, pp270-1
- 17 *Ibid*, p271
- 18 *Ibid*, p272
- 19 Cunliffe. *The City of Bath*. p156
- 20 Neale. *Bath 1680-1850*. p274
- 21 *Ibid*
- 22 *Ibid*
- 23 Cunliffe. *The City of Bath*. p151
- 24 Neale. *Bath 1680-1850*. p289
- 25 Chadwick, E. *Report on the Sanitary Condition of the Labouring Population of Great Britain*. (1842; republished, with an

introduction by M Flinn (ed.), Edinburgh 1965) p235

- 26 Neale, *Bath 1680-1850*, pp274-5
- 27 Chadwick, *Report on the Sanitary Condition of the Labouring Population*, p235
- 28 Sir Henry T De La Beche, 'Report on the City of Bath and It's Sanitary Condition' contained in the Appendix of the *Second Report of the Royal Commission appointed to Inquire into the State of Large Towns and Populous Districts, 1845*, Vol XVIII, pp176-269
- 29 *Ibid*, Evidence of Mr George, Town Clerk of Bath, p248
- 30 *Ibid*, Mr George, p245
- 31 *Ibid*, *passim*
- 32 *Ibid*, Mr George, p245
- 33 *Ibid*, *passim*
- 34 *Ibid*, *passim*
- 35 *Ibid*, *passim*
- 36 *Ibid*, *passim*
- 37 *Ibid*, Evidence of Mr Little, agent for the Circus Water Company of Bath, p252
- 38 *Ibid*, Mr George, p250
- 39 Smith, F B, *The People's Health; 1830-1910*, (London 1979) pp215-6
- 40 Sir Henry T De La Beche, 'Report on the City of Bath', Mr George, p250
- 41 Smith, *The People's Health*, p216
- 42 Sir Henry T De La Beche, 'Report on the City of Bath', Evidence of Mr Frederick Field, pp254-5
- 43 Creighton, C, *A History of Epidemics in Britain; 1666-1893*, Vol II (Cambridge 1894) pp822, 604-5, 844
- 44 Briggs, A, *Victorian Cities*, (London 1983) p188
- 45 *Ibid*
- 46 *Ibid*
- 47 Briggs, *A History of Birmingham*, Vol II, (London 1952) p28

- 48 *Ibid*
- 49 *Ibid*
- 50 Sir Robert Rawlinson, *Report to the General Board of Health on the Sanitary Condition of Birmingham*, (1849) p41
- 51 *Ibid*
- 52 Chapman, S D, and Bartlett, J N, 'The Contribution of Building Clubs and Freehold Land Society to Working Class Housing in Birmingham' in Chapman, S D, (ed.) *A History of Working Class Housing: A Symposium*, (Newton Abbott 1971) p223
- 53 *Ibid*, p224
- 54 *Ibid*, pp224-5
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- 66 Rawlinson, *Report to the General Board of Health*, p23
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- 73 *Report on Birmingham by a Committee of Physicians and Surgeons*, p3
- 74 Rawlinson, *Report to the General Board of Health*, Evidence of Mr Bliss, Inspector of Nuisances, p34
- 75 *Report on Birmingham by a Committee of Physicians and Surgeons*, p3
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- 81 Rawlinson, *Report to the General Board of Health*, Evidence of Mr Bliss, p34
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- 88 *Report on Birmingham by a Committee of Physicians and Surgeons*, p5
- 89 *Ibid*, p5
- 90 *Ibid*
- 91 *Ibid*
- 92 Hodgson, *Evidence to the Health of Towns Committee*; Rawlinson, *Report to the General Board of Health*, p80
- 93 *Report on Birmingham by a Committee of Physicians and Surgeons*, p2
- 94 Rawlinson, *Report to the General Board of Health*, p48
- 95 *Ibid*

- 96 *Ibid*
- 97 *Ibid*
- 98 *Ibid*, p49
- 99 Report on Birmingham by a Committee of Physicians and Surgeons, p2
- 100 *Ibid*, p2. Chadwick commented in 1842 that the passenger from London "who enters Birmingham .... may perceive a black ... stream, which is the River Rea, made the receptacle of sewers". Chadwick, *Report on the Sanitary Condition of the Labouring Population*, p363
- 101 Rawlinson, *Report to the General Board of Health*, pp19-20
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- 103 *Report on Birmingham by a Committee of Physicians and Surgeons*, p9; Chadwick referred, in 1842, to the fact that Birmingham was "distinguished apparently by an immunity from fever". Chadwick, *Report on the Sanitary Condition of the Labouring Population*, p89
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- 108 *Ibid*, p13
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- 110 *Ibid*, p2
- 111 Gray, D. *Nottingham through Five Hundred Years*, (Nottingham 1960) p170
- 112 *Ibid*
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- 114 *Ibid*, p14; Chapman, S D. 'Working Class Housing in Nottingham in the Industrial Revolution', *Transactions of the Thoroton Society*, Vol 67 (1963) p81
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- 119 Chapman, 'Working Class Housing in Nottingham', p81
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- 122 *Ibid*
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- 126 *Ibid*, p32
- 127 *Ibid*, p38
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- 131 Chapman, 'Working Class Housing in Nottingham', p82
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- 133 Municipal Corporations Act, 5&6 William IV, Cap 76
- 134 Church, *Victorian Nottingham*, p176
- 135 *Ibid*
- 136 *Ibid*, p169; 185
- 137 *Ibid*, p183
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- 141 *Report of the Royal Commission appointed to Inquire into the State of Large Towns and Populous Districts*, 1845, Vol XVII, pp284-381
- 142 *Extract from the Nottingham Town Council Book; 1847-8*, Nottingham

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- 143 *Report of the Royal Commission appointed to Inquire into the State of Large Towns and Populous Districts*, 1845, XVII pp284-381
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- 145 Chapman. 'Working Class Housing in Nottingham', p82
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- 152 *Report of the Sanitary Committee of Nottingham; 1849*,
- 153 *Ibid*
- 154 The following information is taken from the Sanitary Committee report 1847-8 contained in the *Extract from the Nottingham Town Council Book*, pii
- 155 The following information is taken from the *Report of the Sanitary Committee of Nottingham; 1849*,
- 156 The information in the following paragraph is taken from the Nottingham Enclosure Act; 1845, pp226-234
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- 158 *Ibid*, p47
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pp5, 6, 12, 17, 20, 22-4, 30

FOOTNOTES: CHAPTER FIVE

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- 2 See Chapter Two; page 30
- 3 Haddon, J. *Bath*. (London 1973) p167
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- 6 *Ibid*
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- 11 *Annual Report of the Medical Officer of Health for Bath; 1880*. p4
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- 15 See Chapter One, page 23-4
- 16 See Table 3,7 and Table 3,8
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- 20 Morris, R J. and Rodger, R. *The Victorian City; 1820-1914*. (Harlow 1993) p262
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- 38 *Ibid*
- 39 *Ibid*, p171
- 40 *Annual Report of the Medical Officer of Health for Bath; 1890*, p13
- 41 *Ibid*
- 42 *Ibid*
- 43 *Annual Report of the Medical Officer of Health for Bath; 1900*, p28
- 44 *Ibid*
- 45 Haddon, *Bath*, p154
- 46 See Chapter Three, page 66
- 47 *Annual Report of the Medical Officer of Health for Bath; 1880*,

- 48 Haddon, *Bath*, p176
- 49 *Ibid*, p139
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- 51 *Ibid*
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- 53 Briggs. *History of Birmingham*. p82
- 54 See Table 3.8
- 55 See Table 3.8
- 56 See Table 3.7
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- 59 *Annual Report of the Medical Officer of Health for Nottingham; 1885*. p53
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- 61 See Table 3.12
- 62 Church. *Victorian Nottingham*. p346
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- 64 *Ibid*, p49; Cage, R A. (ed.) *The Working Class in Glasgow; 1750-1914*. (Beckenham 1987) p73
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