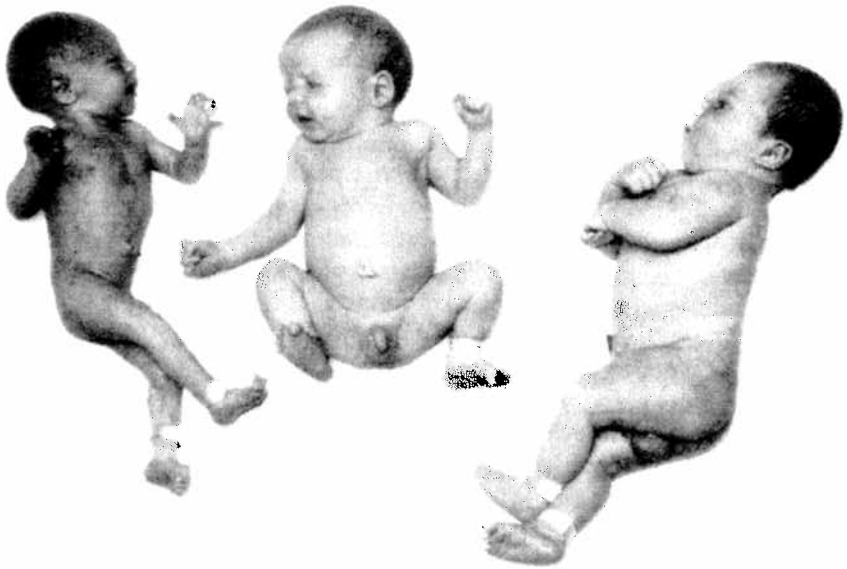
D.J.P. Barker (1998)
Mothers, Babies and Health in Later Life
Edinburgh: Churchill Livingstone

# 1

# Clues from geography

The three babies in Figure 1.1 were born in the same hospital in England. Each was born after an uncomplicated pregnancy and delivery, and their birthweights were within the normal range. Yet the findings which will be described in this book suggest that the baby on the left, the smallest one, will be more susceptible to coronary heart disease, stroke, diabetes and chronic bronchitis as an adult, and is destined to have a shorter, less healthy life.

The thesis of this book is that a baby's nourishment before birth, and during infancy, and its exposure to infection during early childhood, influence the diseases it will develop in later life. Chapters 3 to 7 examine the long-term effects of nutrition in utero; Chapter 8 reviews the control of nutrition and growth in utero; Chapter 9 examines the long-term effects of infection, and



Chapters 10 and 11 discuss the implications of these observations for the prevention of disease.

## CORONARY HEART DISEASE

At the start of this century the incidence of coronary heart disease rose steeply; it rapidly became the most common cause of death in Western countries. Its incidence is now rising in other parts of the world to which Western influences are extending, such as India, China, Eastern Europe and Russia. As such rapid increases in incidence over a short time cannot be the result of changes in gene frequency, attention has been directed at the environment, in particular the lifestyles of men and women in industrialised countries.

Given that the other major heart disorder in adult life, chronic rheumatic heart disease, was already known to be caused by events in childhood, it may seem surprising that adults rather than children were the early focus of research into coronary heart disease. Perhaps discovery of the powerful effects of cigarette smoking on lung cancer directed attention in this way. Whatever the reason, 40 years of research into adult lifestyle have met with limited success in explaining the origins of coronary heart disease: obesity and cigarette smoking have been implicated, and evidence on dietary fat has accumulated to the point where a public health policy of reduced intake is prudent, though unproven: preliminary evidence points to a role for psycho-social stress.[1] Much, however, remains unexplained.

A recent review of trials of a wide range of lifestyle interventions, including exercise, weight loss, smoking cessation and dietary changes, shows that their effects in reducing the incidence of coronary heart disease are small, less than 8% reductions at best, and statistically insignificant.[2] The limited insights provided by research into the links between lifestyle and coronary heart disease has led the British Heart Foundation to conclude that:

> we shall probably never have proof that a particular lifestyle factor or item of diet is important and those who demand proof before any action are condemning us to wait forever.

Physicians are familiar with the need to advise patients in circumstances where there is only limited knowledge. If knowledge subsequently advances the advice can be changed. Unfortunately formulation of public health policies to prevent coronary heart disease, policies based on the best available advice, has simultaneously created a scientific orthodoxy. This states that the disease results from the 'unhealthy' lifestyles of westernised adults together with a contribution from genetic inheritance. Such a view of coronary heart disease, however, leaves its changing incidence and geography largely unexplained, and offers little insight into why, within westernised communities, one person develops the disease while another does not. The effectiveness of preventative measures based on this view of the disease is being questioned.[3]

In many Western countries the steep rise of coronary heart disease has been followed by a fall; in the USA,[4] this has been of the order of one-quarter over 20 years. No parallel changes in adult lifestyle seem to explain it. In Britain there Government food policy led to major and widespread changes in diet, so that fat and sugar consumption fell sharply and fibre consumption rose.[5,6] Death rates from coronary heart disease in middle-aged men and women, however, continued to rise throughout the war and the period of post-war rationing.[7]

The geography of coronary heart disease in Britain is paradoxical. Rates are twice as high in the poorer areas of the country, and in lower income groups.[8] The steep rise of the disease in Britain and other Western countries was associated with rising prosperity,[9,10] so why should its rates be lowest in the most prosperous places, such as London and the home counties, and in the highest income groups?[11,12] Biochemical and physiological measurements in adult life, including serum cholesterol and blood pressure, have been shown to be linked to coronary heart disease.[13] Yet, even when combined with these biological risk factors, adult lifestyle has limited ability to predict coronary heart disease.[14] Rose[15] has pointed out that, for a man falling into the lowest risk groups for cigarette smoking, serum cholesterol concentrations, blood pressure and pre-existing symptoms of coronary heart disease, the most common cause of death is coronary heart disease.

It is, perhaps, surprising that it was geographical studies that gave the early clue that answers to these paradoxes may come from events in utero. Nevertheless, the first indication that coronary heart disease might be linked to impaired fetal growth came from the demonstration that differences in rates of death from coronary heart disease in different parts of England and Wales paralleled previous differences in death rates among newborn babies.[16] In the past most deaths among newborn babies were associated with low birthweight. In these early studies the death certificates for all people who had died in England and Wales during 1968–78 were used to calculate coronary heart disease rates for men and women in each of the 1366 local authority areas.[17] Death rates were expressed as standardised mortality ratios which take into account differences in the age and sex distribution of populations in different areas, and are calculated so that the national average is 100. Figure 1.2 shows how the concentration of low mortality from coronary heart disease in the south and east contrasts with the high mortality in the northern industrial towns, and the poorer rural areas in the north and west. This contrast is seen in men and women, with the exception of north Wales where mortality among women is low.[17]

Figure 1.3 shows infant mortality (deaths under 1 year of age) in England and Wales in the early years of the century. The distribution is surprisingly similar to that of coronary heart disease today. To compare the distribution more formally, the country was divided into the 212 areas used by the Registrar General, comprising each large town (county borough), the London boroughs, the smaller towns in each county combined together, and the rural areas in each county combined. The scattergrams in Figure 1.4 confirm the similarity of the current distribution of coronary heart disease and the distribution of infant mortality in the years 1921–25. The correlation coefficient for this relationship is 0.69 in men and 0.73 in women. In separate analyses for men and women living in large towns, small towns or rural areas, the correlation coefficients

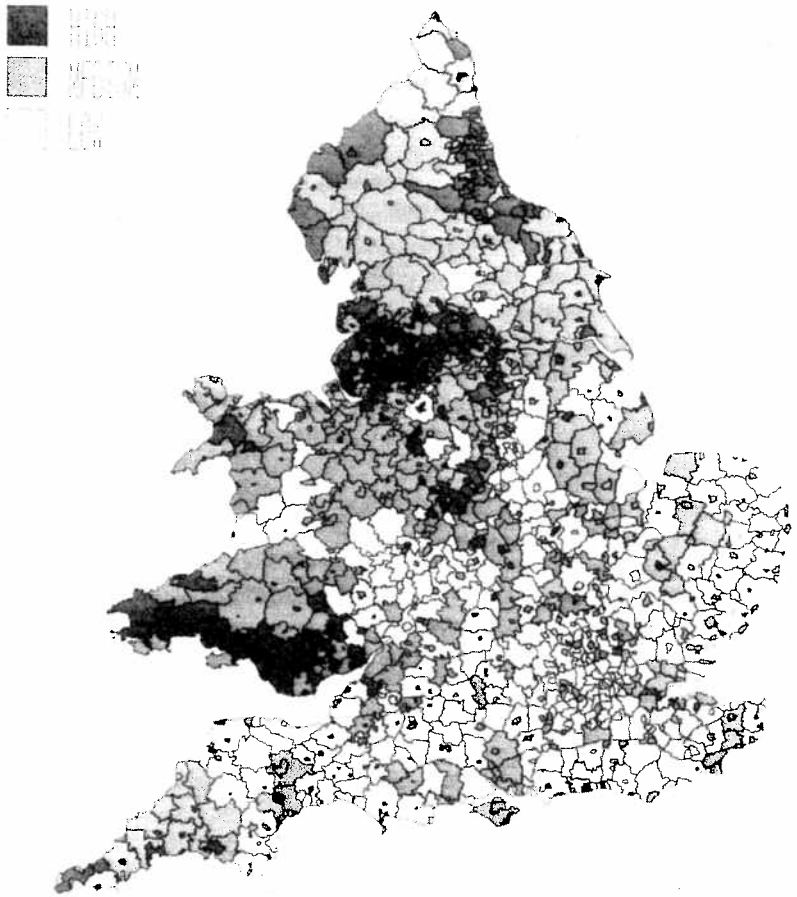


**Fig. 1.2** Standardised mortality ratios (SMR) for coronary heart disease in England and Wales among men aged 35–74 years during 1968–78.

Of the 23 common causes of adult death other than coronary heart disease, only chronic bronchitis, stomach cancer and chronic rheumatic heart disease had a similarly close geographical relationship with past infant mortality. Such a relationship with infant mortality would be expected for these diseases, because they are linked to poor social conditions, and their rates, like those of infant mortality, have declined during this century. It is, however, paradoxical that coronary heart disease is related to infant mortality because the rates have increased during this century.

One possible explanation of Figure 1.4 is that the poor social conditions which caused infant deaths in the past are in some way linked to adult lifestyles which cause death from coronary heart disease. The nature of such a link is not, however, apparent. Differences in cigarette smoking do not appear to follow those of past infant mortality because the distribution of deaths from lung cancer is strikingly different from that of past infant mortality. Therefore it cannot be argued that the social conditions giving rise to infant death led to higher cig-

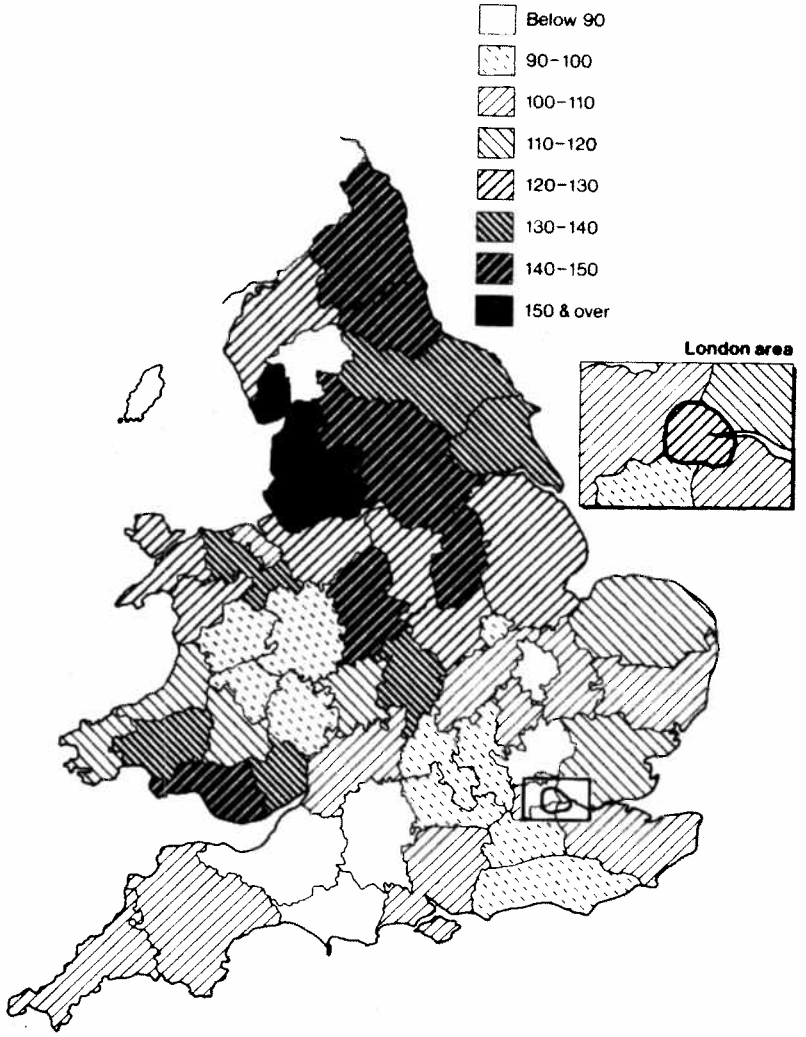



**Fig. 1.3** Infant mortality rates per 1000 births in England and Wales during 1901–10.

tribution as past infant mortality.[18] The close geographical similarity between past infant mortality and current mortality from coronary heart disease is most readily reconciled with their opposing time trends through the hypothesis that adverse environmental influences in utero and during infancy, associated with poor living standards, directly increase susceptibility to the disease.

## THE ENVIRONMENT DURING CHILDHOOD

Findings from other studies support the general hypothesis that coronary heart disease is linked to adverse influences in early life. Forsdahl[19] reported that arteriosclerotic heart disease correlated with past infant mortality in the 20

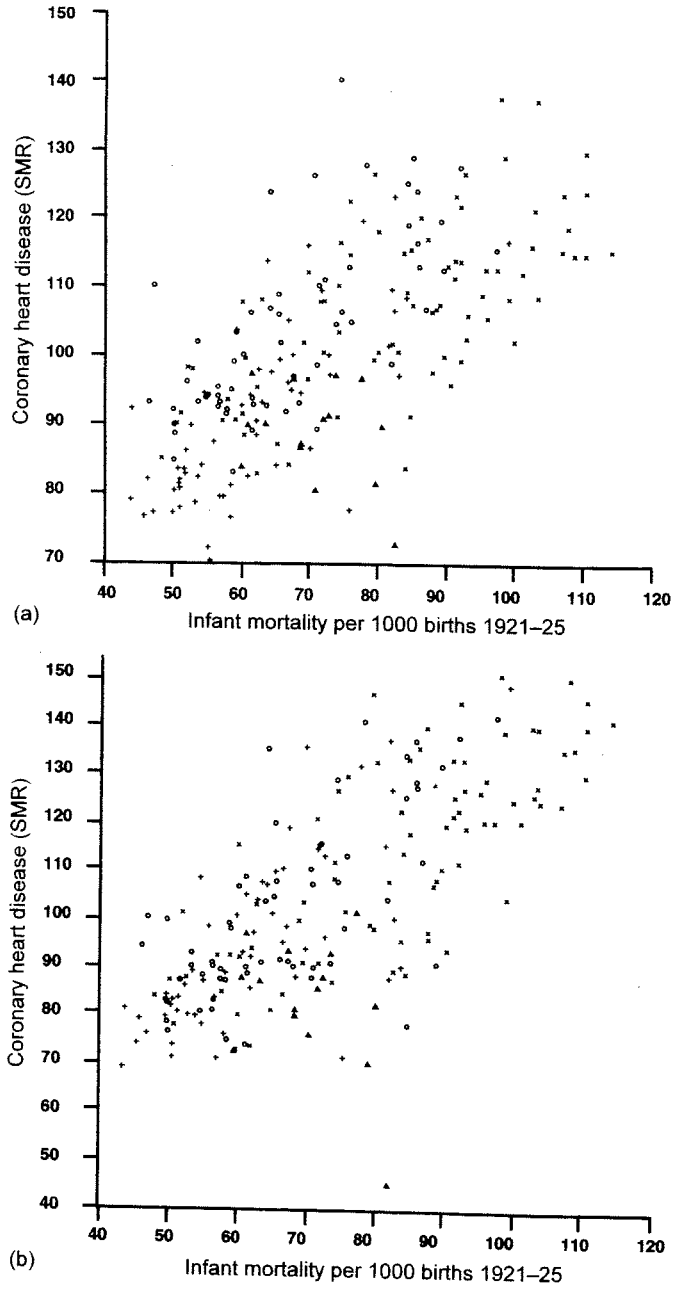



**Fig. 1.4** Standardised mortality ratios (SMR) for coronary heart disease in (a) men and (b) women during 1968–78 and infant mortality during 1921–25 in England and Wales. Δ, London boroughs; X, county boroughs; O, urban districts; +, rural districts.

study compared east and west Finland and came to similar conclusions: that poor living conditions in childhood, with bad housing and recurrent exposure to infection, increased the later risk of coronary heart disease.[20] In 17 states of the USA, mortality from coronary heart disease was shown to be related to infant mortality resulting from diarrhoeal disease, which again suggested that



Other observations which suggest that influences in childhood are linked to coronary heart disease include those made by Rose.[22] He reported that siblings of patients with coronary heart disease had stillbirth and infant mortality rates that were twice as high as those of controls. He concluded that 'ischaemic heart disease tends to occur in individuals who come from a constitutionally weaker stock', a conclusion foreshadowing what is known today. The study of London civil servants by Marmot et al[23] showed that death rates were higher in those who were shorter in stature, and who may therefore have had a worse environment in early life. Among long-term employees of the Bell System Company in the USA, men whose parents had been in 'white collar' occupations had a lower incidence of coronary heart disease than those from 'blue collar' families.[24]

## THE ENVIRONMENT IN UTERO

The size of the geographical study in England and Wales, based on almost one million deaths from coronary heart disease, together with the remarkably complete and detailed infant mortality records, made it possible to examine whether coronary heart disease was associated with specific causes of infant death and hence with particular aspects of the early environment. Infant deaths were divided into neonatal (deaths in the first month after birth) and postneonatal (deaths from 1 month to 1 year). They were also divided into five causes, using a classification devised 50 years ago for an extensive analysis of the social causes of infant mortality:[25] congenital, bronchitis and pneumonia, infectious diseases, diarrhoea and 'other'.

The distributions of neonatal and postneonatal mortality throughout England and Wales were broadly similar. Nevertheless there were areas where the rate of one was high while the other was low. The 15 boroughs of London were important in this respect. London had low neonatal but high postneonatal mortality. Possible reasons for this are examined in Chapter 10. The 212 local authority groups in the country were ordered according to the neonatal mortality rates during 1911–25 and divided into five groups according to the level of mortality.[26] Neonatal mortality rose from 30 per 1000 births in group 1 to 44 in group 5. Five groups with increasing postneonatal mortality were derived in the same way, mortality rising from 32 per 1000 in group 1 to 73 in group 5. In this way the relationship of neonatal and postneonatal mortality to adult mortality could be examined within a grid of 25 cells (Table 1.1). Areas with low neonatal but high postneonatal mortality were mainly in London, although they included the towns of Chester and Great Yarmouth. Areas with high neonatal but low postneonatal mortalities were scattered through the north and west, including the rural areas of Anglesey, Northumberland and Staffordshire.

Table 1.1 compares death rates from stroke, coronary heart disease, and chronic bronchitis. Within any of the five bands of postneonatal mortality, standardised mortality ratios for stroke increase sharply with increasing neonatal mortality. There is no independent trend in stroke mortality with post-

**Table 1.1** Standardised mortality ratios (SMR) from stroke, coronary heart disease, and chronic bronchitis (ages 35–74, both sexes, 1968–78) in the 212 areas of England and Wales grouped by neonatal and postneonatal mortality (1911–25)

| | | Postneonatal mortality | | | | |
|---|---|---|---|---|---|---|
| Neonatal mortality | | 1 (lowest) | 2 | 3 | 4 | 5 (highest) |
| *Stroke* | 1 (lowest) | 85 | 81 | 79 | 78 | 79 |
| | 2 | 86 | 90 | 98 | 74 | 76 |
| | 3 | 102 | 100 | 104 | 104 | 104 |
| | 4 | — | 108 | 110 | 115 | 117 |
| | 5 (highest) | 124 | — | 121 | 123 | 117 |
| *Coronary heart disease* | 1 (lowest) | 84 | 89 | 91 | 88 | 98 |
| | 2 | 85 | 93 | 95 | 88 | 91 |
| | 3 | 86 | 94 | 99 | 106 | 113 |
| | 4 | — | 98 | 109 | 111 | 115 |
| | 5 (highest) | 83 | — | 114 | 119 | 116 |
| *Chronic bronchitis* | 1 (lowest) | 67 | 78 | 106 | 115 | 161 |
| | 2 | 64 | 84 | 85 | 104 | 126 |
| | 3 | 69 | 65 | 89 | 88 | 151 |
| | 4 | — | 91 | 99 | 120 | 142 |
| | 5 (highest) | 41 | — | 108 | 123 | 144 |

chronic bronchitis shows a steep increase with increasing postneonatal mortality, but no independent trend with neonatal mortality.

Seventy years ago most neonatal deaths occurred within a week of birth, and depended on adverse intrauterine rather than postnatal influences.[27] Of such deaths, 80% were certified to be the result of 'congenital' causes, which also correlate geographically with stroke and coronary heart disease. The link between neonatal mortality and coronary heart disease and stroke therefore suggests that early influences predisposing to cardiovascular disease act during prenatal life. Postneonatal deaths were the result of respiratory infection, diarrhoea and other infections, reflecting inadequate housing, overcrowding, and other adverse influences in the environment after birth. The association between chronic bronchitis and respiratory infection in infancy is discussed in Chapter 7.

## MATERNAL NUTRITION AND HEALTH

The relationship between cardiovascular disease and the intrauterine environment can be explored further by examining maternal mortality which, geographically, was closely related to neonatal mortality. In Britain maternal mortality remained at a disturbingly high level from the late 19th century until the mid-1930s:[28] 'A deep, dark and continuous stream of mortality'. In the early part of this century the geographical distribution of maternal mortality in Britain was similar to that of neonatal mortality.[29]

Maternal mortality tends to be highest in rural, sparsely populated counties, and in industrial districts, notably those associated with the textile industries in Lancashire districts in and around London, and in certain large towns, such as Birmingham, Manchester and Liverpool.[29]

Two early reports analysed the causes of maternal mortality,[29, 30] grouping deaths into those caused by puerperal fever (around 40%) and those caused by 'other complications of pregnancy and parturition'. Most of these 'other' deaths resulted from toxaemia, haemorrhage or accidents of childbirth.

Figure 1.5 shows that the geographical distribution of stroke correlates closely with past maternal deaths from these 'other causes',[31] the correlation coefficient being 0.65. The relationship occurs in both sexes and is specific. Among other causes of death, only coronary heart disease correlates as closely with past maternal mortality. As expected from Table 1.1, maternal mortality is unrelated to chronic bronchitis.

In his analysis of infant mortality in Britain, Woolf[25] stated that much of the variation in neonatal mortality depended on variations in poverty, as measured by the percentage of unemployed men in the lower socioeconomic groups. He attributed this to the adverse effects of poverty on maternal nutrition and lactation. Campbell's[29, 32] earlier analyses had also identified poor health and physique of mothers as a major cause of maternal mortality. She attributed them to poor nutrition, rickets in infancy and industrial employment of girls. Baird[33, 34] also related the large geographical differences in perinatal mortality in Britain to differences in the physique and health of women. He concluded that the poor living standards which accompanied industrialisation or economic depression adversely affected the development of young girls, and impaired their subsequent reproductive efficiency.

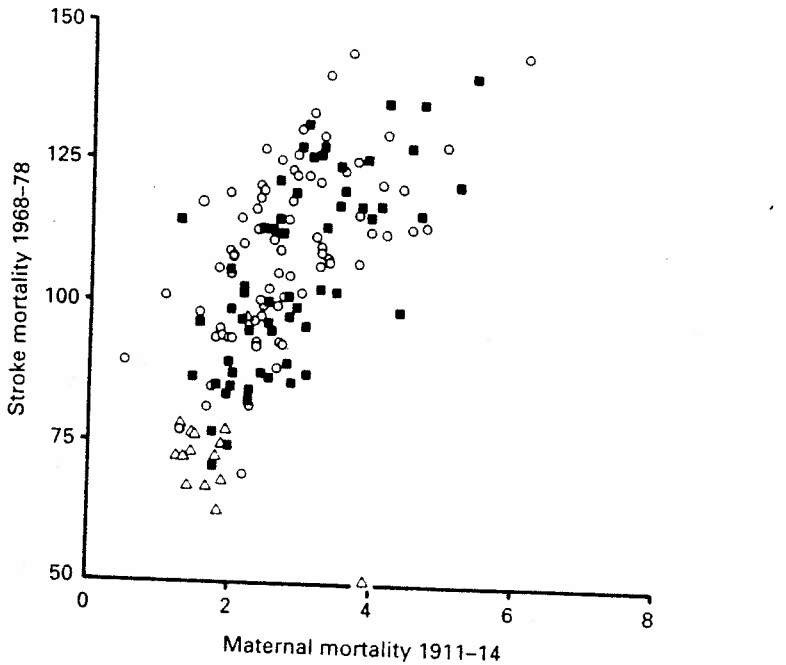



**Fig. 1.5** Standardised mortality ratios (SMR) for stroke in men and women aged 55–74 years during 1968–78, and maternal mortality (

**Hypothesis**

An interpretation of the analyses described here is that poor nutrition, health and development among girls and young women are the origin of high death rates from cardiovascular disease in the next generation. They prejudice the ability of mothers to nourish their babies in utero and during infancy. The fetus responds to undernutrition with permanent changes in its physiology, metabolism and structure, and these lead to coronary heart disease and stroke in adult life.

## MIGRANTS

If the environment in utero and during infancy influences the development of cardiovascular disease, a person's risk of that disease is likely to be related to his or her place of birth. This can be explored by examining disease rates in people who migrate from their place of birth, because the effects of the environment in early life can be distinguished from those encountered later on. In England and Wales, where variations in maternal and neonatal mortality suggest differences in maternal nutrition from place to place, disease in migrants can be analysed using data from death certificates. Place of birth is recorded on death certificates, although it is not routinely coded. For a trial period during 1969–72, however, the Office of Population Censuses and Surveys (OPCS) did code the place of birth. During this time there were almost two million deaths in England and Wales among people who had been born there. Of these people half had migrated to another part of the country during their lives.

Using these data, Osmond and colleagues[35,36] related numbers of deaths from coronary heart disease and stroke, expressed as a proportion of all deaths, to place of birth and place of death. The results showed that a person's risk of dying from coronary heart disease or stroke was predicted by place of birth, independently of place of death. Part of the increased risk among people born in many northern counties and industrial towns, and in Wales, persisted whether or not they had moved to other areas of the country. The low risk of cardiovascular disease, especially stroke, among people born in and around London went with them when they moved.

Other evidence from migrants comes from studies of the 'stroke belt' in the USA. For the past 50 years, despite the continuing decline in stroke rates, there has been a consistent geographic variation in death rates from stroke in the USA.[37] The highest rates are in the south-east, in an area known as the 'stroke belt', that includes the coastal plain region of North Carolina, South Carolina and southern Georgia.[38] Interestingly these areas have among the highest perinatal mortality rates in the USA today. The analysis of Lackland et al of all recent deaths in South Carolina showed that the proportional mortality for stroke was some 25% lower among people born outside the south east – a difference that was considerably larger in blacks than whites.[39] Conversely an analysis of deaths in New York City found that the higher rates of coronary heart disease and stroke in blacks were largely explained by high rates among blacks who were born in the southern states but migrated to

The findings from migrant studies are necessarily inconclusive. People who migrate from the place where they were born differ from those who remain, in physique, mental attributes and health. Lack of information on age at migration makes it impossible to pinpoint the critical stage in early life when susceptibility to disease is acquired. Nevertheless these observations from the USA and Britain are consistent with the hypothesis that the intrauterine environment influences the development of cardiovascular disease.

**Summary**

The suggestion that events in childhood influence the pathogenesis of cardiovascular disease is not new. The implication of the geographical studies described in this chapter is, however, that the search for environmental causes of cardiovascular disease should not focus on the environment of children, their diets, homes, and illnesses, but rather should focus on babies, for whom mothers are the dominant environmental influence. This is a new point of departure for cardiovascular research.

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# 2

# Programming the baby

The findings described in Chapter 1 led to the hypothesis that undernutrition in utero permanently changes the body's structure, physiology, and metabolism, and leads to coronary heart disease and stroke in adult life. The principle that the nutritional, hormonal, and metabolic environment afforded by the mother may permanently 'program' the structure and physiology of her offspring was established long ago. 'Programming' describes the process whereby a stimulus or insult, at a sensitive or 'critical' period of development, has lasting or lifelong significance.[1,2] The development of the sweat glands provides an interesting example of programming.[3] In the early years of this century Japanese military expansion took their soldiers and settlers into unfamiliar climates. They found that there were wide differences in people's abilities to adapt to hot climates. Physiological studies showed that this was related to the number of functioning sweat glands. People with more functioning sweat glands cooled down faster. Rather than attributing the differences in sweat gland numbers to 'genetic effects', Japanese physiologists explored the early development of the glands. They found that at birth all humans have similar numbers of sweat glands; but none of them function. In the first 3 years after birth a proportion of the glands become functional depending on the temperature to which the child is exposed. The hotter the conditions the greater the number of sweat glands that are programmed to function. After 3 years the programming is complete and the number of sweat glands is fixed. The development of sweat glands encapsulates the essence of programming: a critical period when the system is plastic and sensitive to the environment, followed by loss of plasticity and a fixed functional capacity. The development of the eye in early childhood provides another example of programming. The young eye is usually far-sighted and uses visual information to determine whether to grow longer, in the direction of near-sightedness. Reading at an early age may cause a child's eye to grow into focus at the distance of a page, leading to short-sightedness, whereas the eyes of a child living largely outdoors may grow into focus at infinity.

There are many reasons why it may be advantageous, in evolutionary terms,

# 10

# Preventing chronic disease: lessons from the past

We have a winding sheet in our mother's womb that grows with us from our conception and we come into the world wound up in that winding sheet for we come to seek a grave. We celebrate our own funeral with cries even at our birth as though our three score and ten years of life were spent in our mother's labour and our circle made up in the first point thereof. (John Donne 1572–1631).

Chapter 8 outlined the scientific agenda which now has to be explored if we are to understand how the fetus is nourished, and how its nourishment may be improved. It may be a few years before understanding progresses to the point where effective advice can be given to women before and during pregnancy. Meanwhile, history gives an insight into the social conditions which have affected the well-being of mothers and their babies and, in consequence, may have changed the life expectancy of their children.

## THREE LANCASHIRE TOWNS

In Lancashire, England, the cotton industry was harsh to mothers and their babies.[1] Figure 10.1 shows women employed in a mill in Preston, who worked during pregnancy and returned soon after delivery.[2]

Many women returned to full time work just a few days after having had a baby and there was growing concern that this was contributing towards the high infant death rate in Preston. In 1886 Dr James Rigby produced a critical enquiry on the subject. He described a day in the life of a young mother. She would, he said, have to get up at 5.30 am, wrap the baby in a shawl and take it to a nurse who would care for it during the day. These baby-minders had little concern for the children in their care and often drugged them heavily. At 8.30 the mother would rush back from the mill to breast feed her baby and would probably snatch a piece of bread for herself on the way back. At midday she would rush back to feed the baby and then work on until 5.30 pm. This went on day after day until mother and baby were completely debilitated.[2]

Burnley is one of three Lancashire towns, Burnley, Nelson and Colne, situ-

**Fig. 10.1** Women weavers at Tulketh Mill about 1917.

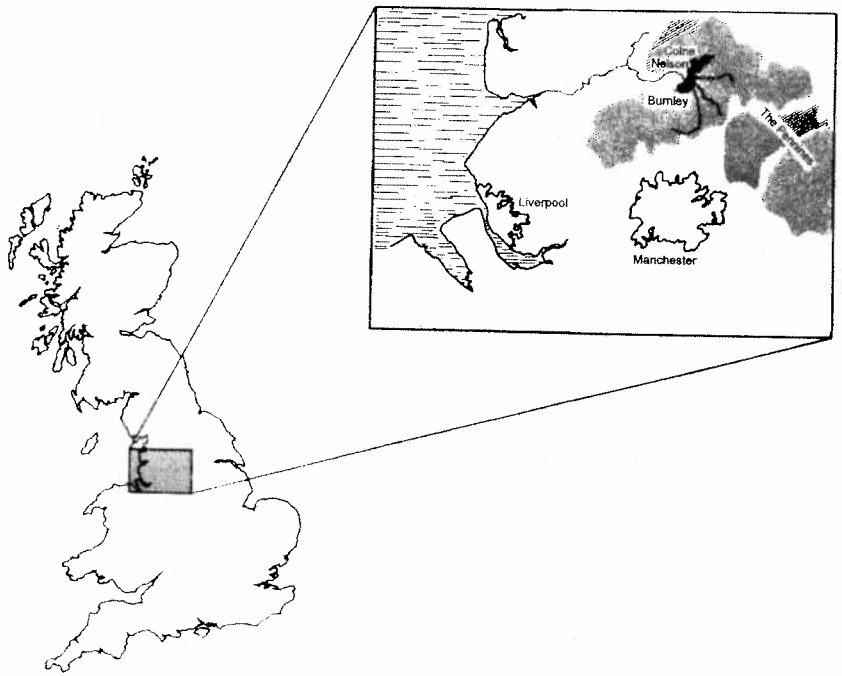


**Fig. 10.2** Map of Great Britain showing the location of Burnley, Nelson and Colne.

Burnley is in the valley where the rivers Brun and Calder meet. Nelson and

**Table 10.1** Three Lancashire towns: standardised mortality ratios for causes of death at ages 55–74 years in 1968–78 (both sexes)

| Causes of death | Nelson | Colne | Burnley |
|---|---|---|---|
| *All causes* | *100* | *109* | *121* |
| Coronary heart disease | 106 | 119 | 120 |
| Chronic bronchitis | 134 | 132 | 188 |
| Pneumonia | 108 | 125 | 174 |
| Stroke | 101 | 121 | 120 |
| Lung cancer | 81 | 83 | 100 |
| Other cancers | 90 | 106 | 101 |
| Other causes | 97 | 93 | 117 |

to Colne, there is hardly a break in the line of houses. For many years there have been large differences in the death rates in the towns.[1] In Burnley adult mortality rates are among the highest for any of the large towns in England and Wales, the standardised mortality ratio (SMR) for all causes being 121 (Table 10.1). In Colne mortality is only 9% above the national average (SMR = 109) whereas in Nelson, situated between the others, mortality is average (SMR = 100). Table 10.1 gives figures for a recent 11-year period for which national death rates were analysed in unusual detail. Of the excess mortality in Burnley, 80% is certified as being the result of coronary heart disease, chronic bronchitis, stroke or bronchopneumonia. Mortality from cancer in the towns is around the national average, and mortality from lung cancer, an index of cigarette smoking, is average or below average.

The close proximity of the towns precludes explaining the large differences in mortality in terms of environmental influences such as climate. It is also unlikely that there are important differences in medical care, since the hospital services for all the towns are centred on Burnley. Could socioeconomic differences between the towns play a role? Recent census data show that all three towns are among the poorer towns in England and Wales, as indicated by the high percentage of manual workers, poor housing, and low income.[3] Socioeconomic differences between Nelson and Burnley are, however, small and less than the differences from the national average (Table 10.2). It is of interest that Nelson has the greatest excess of manual workers but nevertheless has a death rate from all causes that is equal to the national average.

The present similarity of the towns belies the large differences that formerly existed and led to large differences in mortality among infants and young children. Table 10.3 shows infant mortality rates in the towns for four periods from 1896 to 1925.[4,5] Throughout this time rates rose from Nelson to Colne to Burnley. Rates in Burnley were considerably above the average for England and Wales, and were consistently among the highest of any town. Data for 1911–13 distinguish neonatal and postneonatal deaths; rates rose between Nelson and Burnley for both neonatal and postneonatal deaths, and for deaths from the three main groups of causes, that is, diarrhoea, bronchitis and pneumonia, and the so-called 'group of five' diseases. The 'group of five' diseases were premature birth, congenital defects, birth injury, lack of breast milk, and marasmus; the most common of these were premature birth and congenital defects. Differences in mortality between the towns persisted

**Table 10.2** Three Lancashire towns: socioeconomic indices in 1971 compared with those in England and Wales

| | Nelson | Colne | Burnley | England and Wales |
|---|---|---|---|---|
| Employed men in social classes, % | | | | |
| I | 2 | 3 | 3 | 5 |
| II | 13 | 14 | 13 | 18 |
| III, non-manual | 8 | 10 | 10 | 12 |
| III, manual | 48 | 47 | 45 | 38 |
| IV | 18 | 16 | 20 | 18 |
| V | 11 | 11 | 10 | 9 |
| Households with exclusive use of all amenities *, % | 67 | 72 | 63 | 82 |
| People living more than one person per room, % | 14 | 10 | 14 | 12 |
| Households in dwellings of less than five rooms, % | 55 | 44 | 51 | 36 |
| Households owning a car, % | 40 | 36 | 34 | 52 |
| Infant mortality per 1000, 1968–72 | 20 | 19 | 22 | 18 |
| Total population | 31 249 | 18 940 | 76 513 | |

* Hot water, fixed bath, inside lavatory.

**Table 10.3** Three Lancashire towns: infant mortality rates per 1000 births from 1896 to 1925: Infant and child mortality and birth rate from 1911 to 1913

| | Nelson | Colne | Burnley | England and Wales |
|---|---|---|---|---|
| *1896 to 1925* | | | | |
| Infant mortality per 1000 births | | | | |
| 1896–98 | 154 | 170 | 197 | 155 |
| 1907–10 | 107 | 130 | 171 | 113 |
| 1911–13 | 87 | 130 | 177 | 111 |
| 1921–25 | 79 | 109 | 114 | 76 |
| *1911 to 1913* | | | | |
| Infant mortality per 1000 births | | | | |
| Neonatal | 38 | 37 | 49 | |
| Postneonatal | 49 | 93 | 128 | |
| Cause | | | | |
| Group of five diseases* | 35 | 33 | 53 | |
| Bronchitis and pneumonia | 17 | 25 | 26 | |
| Diarrhoea | 16 | 30 | 48 | |
| Mortality at age 1–5 years per 1000 survivors at age 1 year | 58 | 85 | 96 | |
| Birth rate per 1000 population | 18 | 21 | 23 | |

* See text.

## Social conditions in the past

We have an unusually detailed knowledge of social conditions in the towns at the beginning of the century because they were described in a report of the Local Government Board in 1914.[5] The generation whose infancy is described in this report belong to those whose recent death rates are shown in Table 10.1. good. The staple industry was cotton weaving, and the industry employed 40% of all the women and girls aged 10 years and over. Many of the women who worked in the weaving mills of Burnley and Colne were from the second or third generation of Lancashire industrial workers. Nelson, however, had developed more recently and had an eightfold increase in population between 1871 and 1911. Most of the people in Nelson were immigrants from adjacent areas, especially from rural parts of Yorkshire:

> This fact has an important bearing on the question of infantile mortality, owing to the general good health and the habits of cleanliness and thrift characteristic of these immigrants from rural districts.[5]

The women were described as 'sturdier and healthier' than those in Burnley.

There were no creches at the mills. Usually the return of the mother to work was soon followed by complete weaning and the infant, together with other children in the family below school age, was placed in the care of an untrained 'minder' who was paid by the mother.

> In view of the fact that so many mothers are anxious, for the sake of the wages, to get back to employment in the mills as soon as possible after childbirth, a large proportion of children born in Burnley are deprived of the advantages of breast feeding after the first few weeks of life . . . In Colne and still more in Nelson breast feeding is usually continued longer than in Burnley.[5]

Most houses in the towns were built of stone. In Nelson, however, houses were newer and had more rooms (Table 10.4), and so were less crowded. The worst houses were the back-to-back houses in the oldest parts of Burnley and Colne, which were small, had no means of ventilation to the outside air, and lacked facilities for the storage of food and milk. Infant mortality was much higher in such houses: 248 per 1000 in the back-to-back houses of Colne during 1912, for example, compared with 80 in the so called 'through' houses. Much of this excess mortality was the result of diarrhoea. Resettlement of families from back-to-back houses to 'through' houses was accompanied by a fall in infant mortality to around the average for 'through' houses, showing that high mortality was a consequence of the structure of the houses rather than of the habits of those who occupied them.

Sanitary conditions in Nelson were better than those in the other two towns. In Nelson the women kept the streets outside their houses clean: 'more water being said to be used for this purpose in Nelson than in any other town in

**Table 10.4** Three Lancashire towns: housing conditions, mean family size, and total population, 1911

| | Nelson | Colne | Burnley | England and Wales |
|---|---|---|---|---|
| Percentage of population in dwellings of less than four rooms | 5.6 | 15.1 | 13.6 | 19.4 |
| Percentage of population living more than two persons to a room | 3.7 | 6.6 | 9.5 | 9.1 |
| Percentage (no.) of dwellings back-to-back or single room | 0.6 (52) | 17.0 (1000) | 9.9 (2371) | — |
| Mean family size | 4.3 | 4.3 | 4.4 | 4.4 |

Lancashire'. In Nelson communal pits, used for disposal of household refuse, were small and covered and were 'in striking contrast' to the large open pits in Burnley and Colne, which favoured the breeding of flies. Refuse collected from the pits and bins in Nelson and Colne was destroyed, whereas in Burnley around half was put on to 'tips', which were also sites for breeding flies. In Nelson, and to a lesser extent Colne, the manure pits around stables and cowsheds were disinfected in summer to prevent flies breeding. Sanitary regulations for the production and sale of milk were more strictly enforced in Nelson.

Summary

The past differences in infant mortality among the three towns can be linked to differences in the health and physique of mothers, infant feeding practices, housing, and sanitation. They were not related to differences in income or occupation. The children born to the 'sturdier and healthier' mothers in Nelson, more of whom were breast fed, now have lower death rates from cardiovascular disease. After birth these children lived in better, less crowded houses and now have lower death rates from chronic bronchitis.

Mothers in Nelson had better health and physique because it was a newer town. The people were recent migrants from nearby rural areas rather than second or third generation industrial workers. The effects of life in towns in reducing the fitness of successive generations was described by Charles Booth in his *Life and labour of the people in London*, based on surveys carried out in London from 1886 onwards.[6]

## LONDON

For more than a hundred years, people living in the cities and large towns of Britain have had higher death rates than people living in small towns and villages.[7, 8] London is an exception. During 1980–85, for example, standardised mortality ratios for all causes in London, expressed in relation to a national average of 100, were 96 among men and 93 among women. These low standardised mortality ratios resulted largely from low rates of cardiovascular disease.[7] SMRs for coronary heart disease were 90 in men and 87 in women; SMRs for stroke were 78 in each sex. Londoners' low cardiovascular death rates have never been explained.

During 1968–78 standardised mortality ratios in London for coronary heart disease and stroke combined were 87 in men and 83 in women. In none of the 33 London boroughs was cardiovascular mortality above the national average in either sex. London's low cardiovascular mortality contrasts with above average mortality from diseases associated with poor socioeconomic conditions, cigarette smoking, and alcohol consumption. Standardised mortality ratios were 105 for chronic bronchitis in men and 111 in women; 114 and 127 for lung cancer; 101 and 103 for cirrhosis of the liver; and 115 and 130 for suicide. In only four of the 33 boroughs were lung cancer death rates below the national average, the lowest standardised mortality ratio being 92. Thus the lifestyle of Londoners does not seem especially healthy and is not consistent with their low death rates from cardiovascular disease.

In the early years of this century London had low rates of maternal and per 1000 births compared with 4.0 in England and Wales. Neonatal mortality was 33 per 1000 births compared with 39. In the past, maternal mortality was low in places where women had good physique, nutrition, and health, and neonatal mortality was low where few babies had low birthweight.[9, 10] The low maternal and neonatal mortality in London therefore implies that, at the beginning of this century, its women had good physique, health, and nutrition, which is surprising. It conflicts with the picture of London presented by novelists, and with detailed descriptions of life in London given by the surveys which Charles Booth carried out from 1886 onwards. Writing of the London poor, he said:

> Their life is the life of savages with vicissitudes of extreme hardship and occasional excess. Their food is of the coarsest description, and their only luxury is drink.[6]

Amid this savagery, pregnancy, childbirth, and early infancy were unusually safe for both the mother and the baby. Why?

### Social conditions in the past

Young women in London had remarkably low death rates at the beginning of the century. These low rates contrasted with the high rates in girls under 15. Figure 10.3 shows age specific death rates among women in London during 1901–10, expressed as a percentage of the rates in England and Wales.[7] Among girls under 5 years of age London rates were above the national average, and 20% above in girls aged 2–3 years. With increasing age the rates for girls and women fell sharply so that from 15 to 34 years they were well below the average, 17% below in girls aged 20–24. Among older women rates rose and were again above the average. Among men the overall pattern was similar to that in women, with lower rates in young adults; however only at ages 15–24 were